# bida JOURNAL

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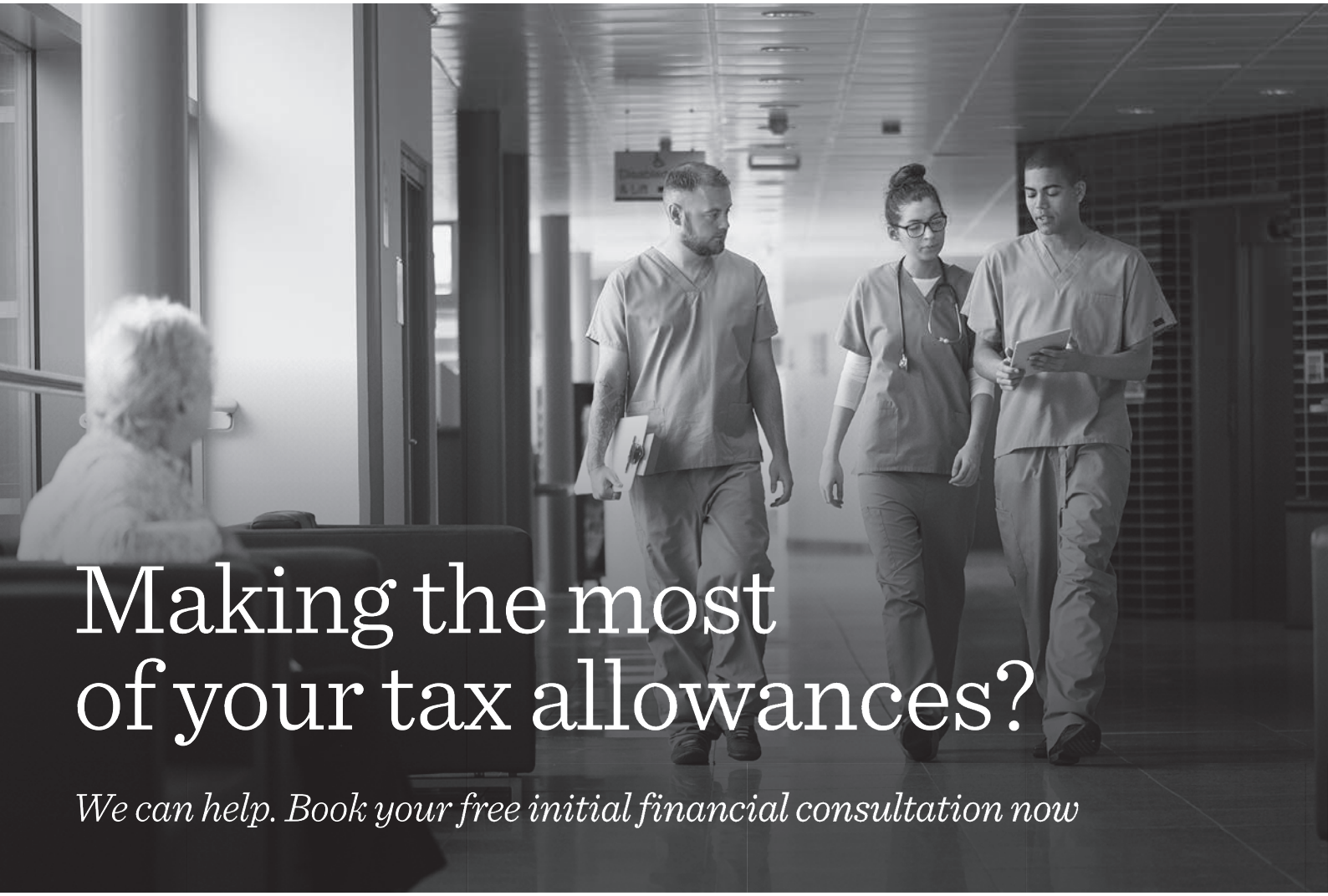

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# Editorial

**Mr Amit Sinha** FRCS (Tr&Orth) Consultant Orthopaedic Surgeon Media & Communication Lead, BIDA Editor, BIDA Journal.

### *It's not over yet?*

The headlines prior to Christmas have troubled us all. The emergence of the new variant Omicron disrupted Christmas plans for several of us. Scientists searching for the source of Omicron suspect it originated in someone in South Africa whose immune system was already weakened by a severe disease such as HIV or cancer. It is very plausible that the emergence of this new variant could be linked, in some cases, to mutations taking place inside immunosuppressed people., those with diabetes, auto-immune diseases, chronic TB, hunger, and obesity. That is because the virus can linger for months in some of these patients – compared to a few weeks for most people – as their immune systems are too weak to combat it. During this prolonged stay the virus can replicate itself over and over again. Occasionally, an error occurs during the virus's copying process, which is how a mutation is created. Most disappear but occasionally they can lead to a new variant. The longer a virus stays inside a person, the greater the risk of generating a "game-changing" mutation. One woman in South Africa continued to test positive for Covid-19 for nearly eight months earlier this year, while the virus underwent more than 30 genetic mutations.

"It's a very rare event. But it is a plausible explanation that individuals that are immuno-suppressed can basically be a source of virus evolution," Professor Tulio de Oliveira, head of the team at South Africa's Centre for Epidemic Response and Innovation, which discovered Omicron informed us.

The number of hospitalisations is small, but the concern remains that even if the symptoms are milder, the sheer number of cases could overwhelm hospitals. We urge people who have chosen to remain unvaccinated to consider changing their minds in light of the fact that it's largely the unvaccinated who are being caught by the virus requiring admission to hospital. Vaccine including the booster dose is perhaps the only route to protecting ourselves. I think it's important that the vaccine hesitant groups are not labelled anti-vaxxers. They should be approached with calm and dignity and with answers to their questions and doubts.

The health service will soon be working on a "war footing". Proposals are being discussed to treat 15% of Covid patients at home, with remote monitoring of their oxygen level. Their homes become virtual wards as the NHS faces the most difficult winter in its history. The other option is "care hotels", in which the NHS would pay for patients to be looked after by live-in carers, with three hotels in southern England already operating. An army of 20,000 NHS reservists – former doctors, nurses and non-clinical staff – may be called on to solidify services.

We are grateful to all the GPs, volunteers and the nursing teams, as the mobilisation of this workforce and the rollout has been outstanding. We're doing something to fix the pandemic, and fixing things is what medicine and nursing is about.

### *Racism*

Ajit Sinha's article on "racism" puts forward his views and what strategies one should adopt for survival and still make progress. It has indeed raised its ugly head with the recent report by Commission on Race and Ethnic Disparities (CRED), which highlights that outright racism still exists in the UK, whether it surfaces as graffiti on someone's business, violence in the street, or prejudice in the labour market. At one time there was a reluctance to acknowledge that racism existed. We now talk about it openly. In my view, I have my doubts that the UK has become a fairer society. It is certainly true that the concept of racism extends from overt hostility to unconscious bias and micro-aggressions. This is partly because ethnic minorities have higher expectations of equal treatment. Quite rightly, they will no longer tolerate behaviour that, only a couple of generations ago, would have likely been quietly endured or shrugged off.

### *Articles*

The article on the "Role of autophagy in diabetes" outlines Amelia Williams' research work. Understanding the process of autophagy provides an opportunity for further research on potential therapeutic effects of autophagy manipulation. Dr Deep Chand, a retired Consultant Radiologist writes about the concept and structure of "Field hospitals", which is relevant during wars as well as major disasters like the current global covid-19 pandemic. Samyak Verma presents the advanced techniques in MISS (minimally invasive spinal surgery) available to spinal surgeons, which will broaden the scope for spinal patients. Catriona Shenton and colleagues have written about "Malignant Otitis Externa", a detailed account, as an early diagnosis and aggressive management can reduce morbidity and mortality.

The New Year is here now. We are optimistic that we enter the next year with the anticipation of almost a fresh beginning. However, we have to accept that the world is seeing a fast changing environment. We need to be adaptable, versatile and be prepared to change our outlook to accept the harsh realities of this environment.

***"Instead of seeing the rug being pulled from under us, we can learn to dance on the shifting carpet. "***. *Thomas Crum.*

**A. Sinha** Editor, BIDA Journal.

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## Instructions for Authors

BIDA Journal is a peer-reviewed journal. We welcome original articles from physicians, surgeons and medical students from any part of the world. These include review articles, scientific articles, case reports, audits and letters to the Editor. Please visit BIDA's website for instructions.

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# bida National President's report

**Dr Chandra Kanneganti** National President, BIDA

Dear Members,

This is my first report as National President of our BIDA. I am honoured to have served as BIDA National Chair for 7 years. It's great to see the new Executive team, led by our new National Chair Dr. Ashish Dhawan and National Secretary Dr. Amit Sinha, already having started their hard work in representing and implementing the organisation's vision of justice and equality in our NHS.

As National President, I will be leading in our Constitution review, supporting our Sports Coordinator, leading on the Awards Committee and supporting our Executive in influencing national policies that affect our NHS workforce.

I would like to extend my congratulations to Prof Iqbal Singh, a pioneer in ethnic health and diversity and BIDA member who has been awarded a CBE in H.M. the Queen's New Year's Honours for his enormous contribution to health care and medical regulation in the UK for decades.

We had our AGM in October 2021 when the new Exec has taken over. We hope to organise our National Conference as soon as possible when the regulations allow.

Finally, I wish all our members and their families a very happy New Year.

**Dr Chandra Kanneganti**
*National President, BIDA*

# bida National Secretary's report

**Mr Amit Sinha** National Secretary, BIDA

Dear Members,

The Covid-19 pandemic has not dampened our spirits, as we have remained very active in the past two years. You are already aware of the tremendous work done by the BIDA team in 2020 during the initial Covid-19 pandemic. In 2021 we have continued to work relentlessly. I am pleased to enumerate our main achievements in the past year:

1. Supporting fellow doctor members in difficulty.
2. Highly successful conferences; National Conference in February and the Oncology Conference in June.
3. BIDA's contribution to the Oxygen India Appeal in collaboration with the British Asian Trust.
4. Establishment of an MOU with the American University of Antigua.
5. Successful sale of the ODA House and acquisition of a new office.
6. Participation in National Conferences of MANSAG & APPNE.
7. Elections and Induction of a new National Executive Committee.

BIDA has been progressing towards a milestone of being in force will be reaching fifty years soon. It has achieved recognition of being the voice of the International Medical Graduates and will continue to do so for several more years. It has now begun a fresh journey of collaboration with other Doctors' BAME organisations. In my view, it is a step in the right direction, as this camaraderie and unity will further strengthen our ties and justify of BIDA being called the International Doctors Association.

As your National Secretary, I'll endeavour to ensure our communication reaches all of you. Each divisional Lead will be requested to ask their BIDA friends to become members. We will be setting up a membership committee from our executive committee group from different regions to be the link persons. Their commitments will be to attend the regional meetings and encourage new members to join in.

For the past two years I have served as the Media & Communication Lead. I am pleased to have Sai Pillarisetti, as the Deputy Lead now. If you have any news you wish to share please do not hesitate to write to me.

We look forward to the New Year with hope and pray that we win over the Covid pandemic in the coming year.

**Mr Amit Sinha**
*National Secretary, BIDA*

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# bida National Chairman's report

**Dr Ashish Dhawan** National Chairman, BIDA

Dear Colleagues,

I would like to start by wishing all BIDA members and their families a very Happy New Year. Just when life was returning to some sort of normalcy, we were again reminded by Omicron variant that we are not out of the Covid Pandemic. After spreading like wildfire, this particular variant is now beginning to slow down with cases and hospital admissions in the U.K. now on the decline. Let us all hope and pray that 2022 marks the end of the Covid Pandemic.

Some of you must be aware that due to a sudden Covid surge, GMC on 21st December 2021 announced that they had cancelled all PLAB 2 examinations for January and February. Many stranded international doctors have contacted us for help and support in this regard.

BIDA, along with other organisations, has been in constant talks with GMC. This engagement has led to a satisfactory resolution for most of you. However, we are aware that some of you still haven't had a suitable outcome. We would like to inform you that talks with the GMC are ongoing and hopefully will reach an acceptable outcome for the remaining candidates.

I recently chaired my second EC meeting as BIDA Chair. I am very pleased to share that we are coming up with a lot of ideas that will not only help our members but will also raise BIDA's profile nationally. We also have a very busy events calendar for this year. Among the upcoming events on the horizon are educational events like our National Conference, the President's Cup Cricket Tournament and, last but not the least, our International Conference. It goes without saying that all of these events and activities are dependent upon how the Covid pandemic unfolds this year.

The strength of any organisation is in its membership. In this context, I firmly believe that local divisions play a pivotal role. I am very keen to see our divisions returning to the same level of activity as before the Covid Pandemic. Please continue support to our organisation, and your local divisions, and we can continue to work for the equality and justice for all doctors in NHS.

**Dr Ashish Dhawan**
*National Chairman, BIDA*

# bida G.P. Forum Chairperson's report

**Dr Preeti Shukla** Chairperson, G.P. Forum, BIDA

Dear Members,

It's been an incredibly challenging couple of years for General Practice and I am so proud that General Practice has delivered time and time again, be it at the start of the vaccination drive or to ramp up vaccinations for the Omicron surge, and all at the same time seeing record numbers of patients in the midst of the pandemic with an increasingly tired workforce.

It saddened me that certain sections of the media denigrated General Practice instead of celebrating its achievements. BIDA refuted the false claims by the media and robustly defended General Practice both in the papers and across social media. We also contributed to raising vaccination awareness in ethnic minorities by doing videos in native languages and circulating them in public, and they were well received.

We continue to raise issues pertinent to General Practice and fight its corner alongside other organisations.

I am very grateful for your support as always, and wish you a happy and healthy year.

**Dr Preeti Shukla**
*Chairperson, G.P. Forum, BIDA*

# Racism
## A BANE TO OVERCOME

**Ajit Sinha** MBA MSc CPHR SHRM-SCP RCIC Principal Consultant, Checkpoint Immigration Strategies, Canada

### *The Ugly Reality*

Racism: a phenomenon that raises its ugly head from time to time – is in our face due to one incident or the other, in this part of the world or the other. Leaders everywhere seem to be playing a continuous game of whack-a-mole with it. Some believe it is systemic, some believe it is on its way out, whereas some believe it is exaggerated and is "practiced" in far corners of society – relegated to the background. I posit that it is here, it will take time, and it will take the collective wisdom of the best of us to root it out and lay it to rest or at the very least relegate it to a non-issue.

If you search for racism on the internet, you will find hundreds of articles, research papers, sociological, economic, and historical rationale, scathing criticism of people who were and are the perpetrators and suggested actions that must be initiated to how to deal with both the cause and the aftermath of such incidents. Despite this knowledge being available to everyone including leaders in the community, statesmen, politicians, and leaders in the corporate world, an end to this malaise seems to be elusive. Do not get me wrong - there are strides forward, but then some backwards. Developed countries who are the liberal path-breakers, are the ones who in the recent past have seen the worst incidents. One could argue though that because they are path-breakers, such incidents are always highlighted. However, it is noteworthy that public displays of such racism still occur.

### *The Divide*

It is not difficult to explain to a teenager or an adult the cause, meaning, explanation of such incidents taking place in the world. The electronic media in our homes and what children hear and see provide the information to our kids. There is hurt, insult, injustice, violence, and brutality heaped on an individual or a section of the society. There is abuse, damage, and destruction. The fear of reprisal is also instrumental in numerous cases being never discussed or made public. Children are the worst affected as they do not understand why their skin colour, the way they speak, the food they eat, the way they dress are reasons why others in their group / class do not like them, do things which hurt them. The "divide" begins early.

*"In Canada we witnessed the police kill Chantel Moore, a Nuu-chah-nulth woman and Regis Korchinski-Paquet, a Black woman, and the beating of Chief Allan Adam of the Athabasca Chipewyan First Nation*[1]*"*. Why was George Floyd in Minneapolis ill-treated physically and killed in full public view by Derek Michael Chauvin, a white American police officer. Asians of Chinese descent in Canada and the United States have faced hate crimes harassment or violence – specially on the coat tails of Covid-19, some politicians have painted the Chinese as perpetrators and the brunt of these assertions has been borne by them. The brown Asians in the developed countries are not much better off. Islamophobia has crossed all logical limits of acceptance. *"An exaggerated fear, hatred, and hostility toward Islam and Muslims that is perpetuated by negative stereotypes resulting in bias, discrimination, and the marginalization and exclusion of Muslims from social, political, and civic life*[2]*."*

Each case when evaluated, represents the malaise of systemic or cultural racism which is still embedded in our values and beliefs. There is the deep-rooted fault line between "privileged class and oppressed class". Racism is a complex phenomenon, ugly and deeply personal. It can be both implicit and explicit. Explicit racism is still easier to point out; implicit racism is not. For example, some "whites" still believe that blacks and browns are suited as subordinate or menial staff. Even more surprisingly there are some blacks and browns who have similar thought processes – conditioned over the years to believe this behaviour "normal", this assumption as factual. In offices it should not be surprising to find that some leaders believe it is normal to accept the work of people of colour as inferior to the ones produced by the white people. There is not one thing that you can point to when it comes to this kind of racism – it is subtle, just under the surface – an appraisal missed, an appreciation not provided or an opportunity not given. If you are not at the receiving end of it, you probably would not even realize it happens. Can this be true in 21st century where we "think" that human rights are paramount, and respect and dignity must always be upheld. We are taught we are born equal, but in practice it seems to be that powers that be provide an edge, one way or the other, when they colour you different. Racism has become omnipresent; sometimes overt sometimes hidden.

In 2015, in one of the peer gatherings in an upmarket hotel conference room, I witnessed a kind white colleague asking a brown colleague whether his pronunciation of the English language was ever an impediment in communicating effectively. Instinctively, I wanted to point out that our brown colleague was the one selected to be at the

podium and we had paid to listen to him. Humility in the brown learned man prevailed and it was politely conveyed that he had not faced any such difficulty. A smile was continuously pasted on his face. Such questions from an individual present an immature mind and a biased approach to life. Even at my workplace or in the community, I have been at the receiving end of snide comments and disrespectful references – sometimes overt, sometimes discreet. I have continued to maintain sanity. My goal was to survive in a new country where everyone was not a racist. It is good to remind yourself that you do not cut down a tree if some of the fruits go bad. However, having said that, sometimes it is hard when such "fruits" periodically show up and it leads to you being suspect of the entire tree.

## The discussions and protests

Who amongst us has not discussed racism – practically everyone whom we know? Our neighbours down south in the USA have been reeling with protests and rallies. Across the world, every country has witnessed this uprising in some form or the other. Leaders have provided their support to actions against racism and leading companies have reached out to their employees. In our own conversations, in the community, in our workplaces, among friends, we continue to discuss how racism is all too prevalent under the surface. Racism is brutally ugly, woven into the fabric of our cultures. What surprises me is our non-acceptance that we are squarely to blame for practicing racism in some way. It is important that we drive awareness of equity, on all fronts, above all factors to achieve true engagement of people. Let us do what we can.

One who believes that she/he is superior to another human being, especially on the basis of race, is a weak human being. Their belief assumes that intellect, characteristics, and abilities are defined by race. They do not understand that the attributes and features of any ndividual is unique. Even identical twins are not identical. Thoughts and values do not discriminate on the basis of the colour of skin. The eureka moments for science, technology, humanities, sociology can come to anyone. How do we explain the work of leaders like Nelson Mandela, Mahatma Gandhi, Dalai Lama, Martin Luther King, Barrack Obama and many more? Their nature, values and teaching give us an idea of how to rise above and think of humanity as a whole. Racism is a symptom of how generational "wisdom", archaic traditions, pseudo ethnic science, tribal superiority mars the progress that we as humans have made.

## The Challenge

There are some amongst us who hurt others, insult them, physically assault them and in some cases, kill them. It is advisable that we support them in changing their outlook. *"Throughout the history, racism creates the conflicts and hatred in the minds of men and women, disrupting mutual understanding and trust to build peace in the world* [3]*".*

The Washington Post[4], in an article by Mark Fisher on 15 May 2013, had presented a fascinating map of the world's most and least racially tolerant countries. The intent was to depict racial tolerance in different countries. North America, large parts of South America, Australia, the UK were all tolerant to accept neighbours of a different race. However, numerous incidents of racial violence are still seen in these countries. In the Indian peninsula, these incidents are seen majorly between Muslims and Hindus who have been politically nudged to be "traditional" rivals. Where there is little diversity, the scope of ethnic violence is minimal. Ethnic diversity leads to confrontation and conflict – we need to evolve, mentally, past this.

It is not correct to state that only whites can spew racial hate. Racism is an open game. Indians foster hate against whites, Chinese and Africans. Settlement in housing colonies follows the patterns adopted by the group. Koreans will buy houses where Koreans have settled down. And so will Italians, Indians, Pakistanis, Germans, and Muslims. We, even in the 21st century, seem to feel safer in communities occupied by their creed. Places of worship continue to emerge in areas of concentration of those communities. It seems to be a natural path to settlement. But what this leads to is a tribal outlook to life. If you are surrounded by only one "type" of folks – anything that is different stands out, seems alien, and in extreme situations seems like anathema. Strides are being made to ensure that such auto-segregation does not happen. Singapore as part of their housing policy decrees that all complexes have people from all races in a similar ratio as represented across the country. What this does is ensure that every culture is aware of every other culture. We need to realize that different does not necessarily mean bad – different is well just different. I like chicken, my son likes beef; my wife is religious, my daughter is agnostic, my wife's loves festivals, my daughter in law not so much – different but a difference that is inconsequential – we love each other.

## The Karens

How are stakeholders reacting to this challenge? Governments, corporate giants, businesses, religious leaders, and the administration including police need no reason to become vocal in asking all to collaborate and understand that racial injustice needs to be disappear. That may happen in a utopian world overnight but in the real world, we will continue to see "Karens" in our society – so some more time to come. Fascinating that the term Karen was a term discussed in the New Zealand parliament. Jacinda Ardern, the New Zealand Prime Minister, supported taking to task their citizens spewing racist hatred. In their country it will be legal to call such ladies by the nickname Karen. "Karens," as the internet term goes, are white women who are often mocked by the internet in viral videos for their outlandishly problematic and often racist behaviour[5]. It does not end there. It has been almost a year since Amy Cooper called 911 on bird watcher Christian Cooper in Central Park in New York, falsely claiming her life to be in danger. Such "Karen" incidents have been depressingly on the rise. There have been incidents of banging on doors of homes of immigrants with people yelling racial slurs. Monuments and buildings dedicated to the excellent work of communities have been vandalised. Sixty-year-old Claudia Emanuele of Bayonne, N.J.[6], was arrested by police for intimidation and harassment after she followed 40-year-old Tameka Bordeaux and was filmed shouting racial epithets at her. Sadly, such incidents do not grab attention anymore. Do these incidents happen in the United States of America or Canada only?

## Incidents in the United Kingdom

More than 60,000 cases of racism complaints were registered in UK schools in the last five years[7]. The actual number of incidents may be higher. If racism is in schools, it is most certainly in every nook and


corner of society. Data is available on racism against Chinese. There is rampant disbelief in Muslims across the country in the United Kingdom. Professor Mark Radford, chief nurse at Health Education England, stressed that racism remains a problem in the UK and in the NHS that needs to be addressed, following a controversial government-commissioned report that appeared to downplay the issue[8]. The controversial report by Commission on Race and Ethnic Disparities[9] claimed that the system could withstand against racial discrimination. The reality is the opposite. The European Human Rights Committee has raised fundamental concerns against the approach of the report. The Royal College of Physicians has stated that Doctors of colour and minority ethnic backgrounds have been hindered in their search for senior roles because of widespread "racial discrimination" in the NHS[10]. Their survey has uncovered years of discrimination against Black, Asian and minority ethnic (BAME) doctors. The NHS has allowed to fester this "ingrained" bias making it harder for BAME doctors to become consultants in comparison to their white colleagues. The Royal College of Surgeons has also published an independent review into diversity and inclusion and concluded that the College has not demonstrated itself to be a diverse and inclusive organisation[11].

Let us evaluate the action of the United Kingdom to identify and eradicate racism in one if the premier agencies of their country – The National Health Service. Boris Johnson's government instituted the Commission on Race and Ethnic Disparities (CRED). CRED[9] published its Race report on 31 March 2021 confirming that the NHS does not yet have racial equality in the UK. The main discontent with CRED is its failure to acknowledge structural racism as a key factor in racial inequality[12] among doctors in NHS. The BMA stated that the discontent is widespread across the country[12]. The Independent also reported on 01 July 2021, *"The professional body for doctors refuted the report's overall findings and said the government-appointed Commission on Race and Ethnic Disparities (CRED) missed opportunities to identify effective solutions to tackling racial inequality"*[13]. If racism, inequality, and discrimination is to be addressed, it must be first accepted – you can not get to the root cause, let alone a solution, if you do not acknowledge the problem. Only then can the faith of the affected be earned. If fairness and equity is expected in patient care, the same should be applied to doctors. The pandemic has further proven that irrespective of the treatment the BAME doctors got from their organisation, they continued to uphold the Hippocratic oath they take when they became doctors.

## In the USA

On ABC's Good Morning America show, the US Vice President Kamala Harris stated, *"I don't think America is a racist country."* Yet immediately following this sentence she quickly pointed out, *"But we also do have to speak truth about the history of racism in our country and its existence today."* All politicians are good at making political statement which are normally far from the truth. Her statement is confusing to me, and I am sure it is confusing to most well-meaning Americans and the international community. Deep inside their psyche, Americans realise the benefits of unearned "assets" of whiteness or wealth or position while Biden and Harris make voter base friendly statements.

## Belonging

Some have alluded to horrific incidents in the world which have carved the clear path for hate and racism to grow exponentially. The 9/11 Twin Tower bombing in New York has been instrumental in branding all Muslims "traitors". Covid-19 has branded all Chinese and slavery in the 18th century has branded all blacks inferior to whites. Then there has been a mushrooming of individual pockets of pure hatred. The Taliban against all whites, ISIS against the USA and anything American.

Islamophobia has tainted all innocent Muslim people. All white-skin people are not racist. Chinese and other Asians are as peace loving and hard working as any other race. Blacks are talented and creative. Looking around us will convince us that the best of the best is not defined.

Belonging is essential to humans[14]. We are social human beings and the importance of living with a purpose is critical for our self-esteem. To be at the receiving end of racism creates psychological trauma. The natural reaction for humans is fight or flight. But to give back in these cases results in conflict. On the other hand, when the reaction is flight it leads to accumulated feelings of hate for the perpetrator, and in the worst case – distrust of society at large, and a feeling of dejection. At each level of the community and the workplace, there needs to be a change in the mindset of people. It will take many years or maybe another century to completely eradicate racism. We need to learn more about ourselves. Robin Diangelo in her book White Fragility[15] argues that *"whites in America must face the racist bias implanted in them by a racist society".* Their resistance to acknowledging this, she maintains, constitutes a *"white fragility"* that they must overcome in order for meaningful progress on both interpersonal and societal racism to happen.

## Where is all this leading us to?

This leads us to initiate actions to end Racism. For once there is an urgent need to stop and take stock of where this hate will lead us to. If the goal is to make meaningful progress, sweeping changes need to be implemented. The focus is to inculcate belongingness which is considered at par with need to love in Maslow's hierarchy of needs. When culture of belonging exists, a space is created for all to share, build empathy and inclusion. Inclusivity can be fostered at every level of society, community, and workplace to respect each other and acknowledge inclusive actions. The role models need to be identified, set apart so that the others can identify themselves with them. Leaders in the community need to be responsive, respectful, and supportive. In difficult and stressful times, continue with the work to achieve the commitment to change, to make our world equitable and fair.

The definition of "belonging" needs to be evaluated once again. Many of us are mostly socialized to keep quiet instead of raising the fundamental right to be a part of the change. It is often a survival strategy for our lives during police encounters or economic survival in boardrooms that prevent us from raising concern. Some of us who succeed may experience "survivor's remorse" because we are some of the few to "make it." We embody our dream and become the in-person example to people who do not want to admit that systemic racism exists. We may even convince ourselves that racism is more a citizen of any country, irrespective of the colour of her/his skin, orientation or speech has an undisputed right to receive the same treatment as anyone else. As a society you want the best to do their best work in their area of choosing, not only is the outcome of such equality beneficial to the individual – white, black, blue, purple, pink – it is beneficial to the society at large. As is, we all bleed red. We do not want to lose out on the next potential cure for cancer, a new clean technology because the individual who was most knowledgeable in that area did not have the "right" attributes.

Last week I was waiting to have my purchase processed in a grocery store. A white mum ahead of me at the cash register was asked by her 4-year-old daughter what racism was? The child likely read an education poster in the store carrying a public message. I was speechless when the mum turned around and asked me if she had my permission to involve me in the explanation to her child. I could just nod in agreement. The lady told her daughter that if she is rude or disrespectful to me because I was dark skinned or my clothes or for any reason, she will be a racist. The lady continued explaining that one needs to be polite, respectful, and kind to every human being. At the end of her explanation, she turned around, smiled, and thanked me. I saw the honest smile of appreciation on the child's face and an equally bright cheer on the lady's face. I bowed to thank her. It was a lesson to me that we must not form opinions on racism and, most importantly, one cannot change the world but certainly, the good deed begins with you.

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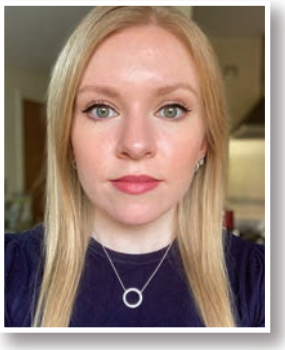
# The role of autophagy in the pancreas and diabetes mellitus

## A SHORT REVIEW



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### Abstract

Diabetes mellitus is a debilitating condition that can limit patients' quality of life. Diabetes prevalence is predicted to rapidly increase in the future, raising concerns about diabetes becoming a public health crisis. The importance of developing treatments for diabetes has been exacerbated by the COVID-19 pandemic, where diabetes has been highlighted as a comorbidity which can increase the mortality risk of patients. Autophagy is a cellular degradative process, involving the lysosomal degradation of damaged cellular constituents, including organelles. The catabolites produced are utilised during periods of cellular stress, such as starvation. Autophagy has been implicated in the pathologies of several intrapancreatic diseases, as well as in diabetic nephropathy. In the exocrine pancreas, autophagy has been implicated in both acute and chronic pancreatitis, as well as pancreatic ductal adenocarcinoma. Autophagy has also been studied in diseases affecting the endocrine pancreas. In the endocrine pancreas, alpha and beta cells are the two most common cell types. In type 2 diabetes, beta cells become dysfunctional, due to cellular exposure to nutrient excess and stressors such as ER (Endoplasmic Reticulum) and oxidative stress. Studies have suggested that the activation of autophagy may act as a pro-survival function for beta cells against cellular stressors. In addition, diabetes can affect the kidney, culminating in diabetic nephropathy. Autophagy is an important process in podocytes, as they cannot divide. Studies have suggested that compounds such as spironolactone and puerarin have a positive effect upon autophagy in mice with diabetic nephropathy. The transcription factor ATF4 has also been highlighted as a potential positive regulator of autophagy during diabetic nephropathy. As such, it is suggested that autophagy may be a desirable target for treatment of several pancreatic diseases as well as diabetic nephropathy.


### Diabetes mellitus – a growing threat to global public health

Diabetes mellitus is a chronic disease, which can lead to the development of severe macro- and microvascular complications for patients[1]. The World Health Organisation estimated that diabetes affected 422 million people globally, in 2014; an increase of approximately 314 million people, from 1980[2]. According to the International Diabetes Federation Diabetes Atlas, this figure has increased to 463 million adults as of 2019[3]. The Federation also suggest that this figure could further rise to 700 million adults, by 2045[3]. It is this growth in prevalence that accentuates the importance of developing novel diabetes treatments, to combat the increasing threat that the disease poses to public health.

Diabetes can be classified into different types; however, the two main classifications include type 1 and type 2. In both cases, glycaemic control is inhibited due to abnormalities in the release of insulin from beta cells, in the pancreas (Figure 1). Type 1 diabetes (T1D) is caused by the idiopathic or autoimmune destruction of pancreatic beta cells and occurs most commonly in children[2,4]. Treatments for T1D include the injection of exogenous insulin, or use of an insulin pump, in order to regulate glucose metabolism in lieu of endogenous insulin production[5].

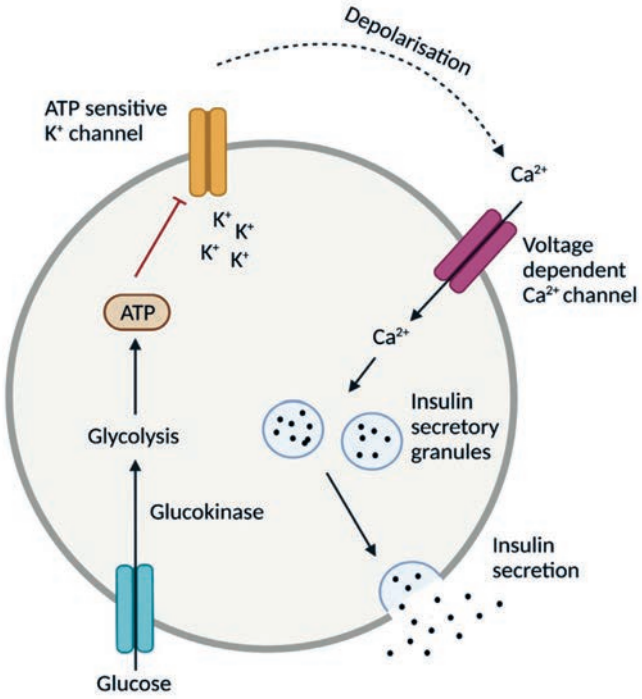


***Figure 1*** *- The process of insulin exocytosis, by beta cells, in response to glucose. Glucose is metabolised during glycolysis producing ATP, which stimulates the closure of ATP sensitive potassium channels[6]. The consequent membrane depolarisation causes an influx of calcium ions to enter the cell, ultimately stimulating the release of insulin from the cell [6]. Figure adapted from Chakravarthy and Semenkovich (2007)[6]. Created with BioRender.com.*

Type 2 diabetes (T2D) possesses a different pathophysiology. T2D is characterised by the development of insulin resistance, and the subsequent beta cell loss in the pancreas[7]. T2D is the most prevalent type of diabetes, accounting for approximately 9/10 of all diabetes cases[8]. Obesity, age, as well as family history, have been highlighted as potential risk factors in the development of T2D[2]. T2D can be managed through a variety of strategies, including lifestyle changes, as well as pharmaceuticals. Changes in diet and exercise habits may aid in the regulation of glucose homeostasis[9]. However, if lifestyle changes alone are not enough to regulate glycaemic control, drugs such as metformin and sulfonylureas can be utilised. Metformin acts to increase insulin sensitivity, whereas sulfonylureas enhance endogenous insulin secretion[9]. Insulin therapy may also be utilised should glycaemic control not be restored via oral pharmaceuticals[10].

The outbreak of the Corona Virus Disease 2019 (COVID-19) pandemic, due to severe acute respiratory syndrome coronavirus 2 (SARS-COV-2) exposure, presented new challenges in the field of diabetes research. Diabetes patients that develop COVID-19 are

more likely to develop severe disease complications, including multi-organ failure[11]. Drugs used to treat COVID-19, such as corticosteroids, also impact upon glucose homeostasis, with hyperglycaemia being associated with increased deaths in all patients[11]. Diabetes has, therefore, been highlighted as a risk factor for mortality in COVID-19 patients[12, 13]. Some studies now suggest that metformin use, prior to COVID-19 hospitalisation, may be protective against intensive care admissions in T2D patients, although there was no observable effect against mortality[12, 13].

## The process of autophagy

Autophagy is a cellular degradation process that regulates the breakdown of damaged, dysfunctional or misfolded protein, organelles and cellular constituents[14]. Three types of autophagy have been classified, including; macroautophagy, microautophagy and chaperone-mediated autophagy[15]. Macroautophagy remains the most studied of the three processes and will be discussed at length in this review.

Macroautophagy, subsequently referred to as autophagy, is a catabolic process, mediated by the presence of lysosomes (Figure 2). Autophagy occurs as a multi-step process consisting of initiation, elongation, maturation, lysosomal fusion and degradation[16]. During the initiation stage, a structure known as a phagophore, develops to surround targeted cellular constituents[16]. The phagophore then closes and matures to form a double-membraned autophagosome[16]. Fusion of an autophagosome to a lysosome causes the release of lysosomal enzymes into a newly formed autophagolysosome[16, 17]. Lysosomal enzymes then breakdown the engulfed cargo into catabolites, such as amino acids and lipids[17]. These building blocks can then be utilised by the cell to adapt to stresses such as nutrient starvation[17].

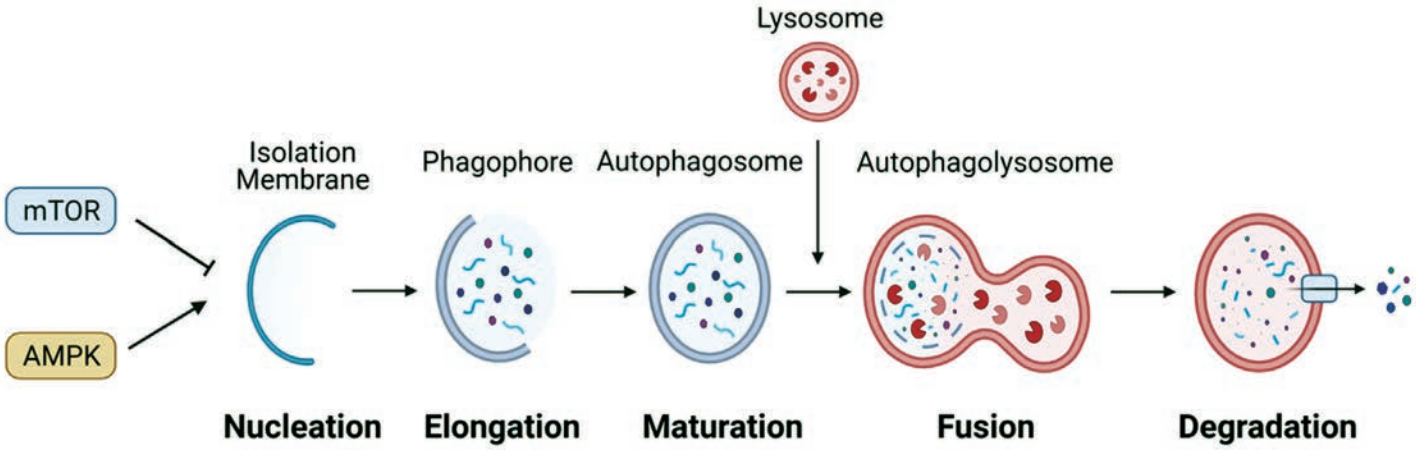


***Figure 2** - The five-stage process of macroautophagy; initiation, elongation, maturation, lysosomal fusion and degradation. Macroautophagy is controlled via the dual action of the AMPK and mTOR signalling pathways, which stimulate and inhibit the process. Catabolites that are produced by macroautophagy are 'recycled' and utilised to maintain cellular homeostasis[17]. Created with BioRender.com.*

Microautophagy and chaperone-mediated autophagy possess different mechanisms of action that occur upon the lysosome, and do not require the formation of an autophagosome[17]. Microautophagy occurs through the engulfment, and consequent breakdown, of targeted cargo by lysosomes[15, 18]. In contrast, chaperone-mediated autophagy delivers specific proteins to the lysosome, via interaction with heat shock-cognate chaperone 70 kDa (HSC70) and lysosomal associated-membrane protein 2A (LAMP-2A)[15, 18].

The process of autophagy is regulated by the synergistic action of the AMP-activated protein kinase (AMPK) and mammalian target of rapamycin (mTOR) signalling pathways[19]. AMPK signalling negatively regulates mTOR signalling; AMPK signalling initiates autophagy, whereas mTOR signalling inhibits autophagy[20]. The AMPK pathway is a known mediator of cellular homeostasis, whereas mTOR aids in the regulation of cellular growth[20]. Nutrient stress can impact upon the activation of both AMPK and mTOR signalling[21]. In eukaryotic cells, starvation can induce AMPK signalling, causing an inhibition of mTOR signalling, and aid in the production of catabolites that can then be metabolised for energy[22]. Alternatively, nutrient excess can activate mTOR signalling, inhibit autophagy, and can aid in the stimulation cellular growth[23].

## Introduction to the pancreas

The pancreas is the organ responsible for maintaining glucose homeostasis within the human body. The pancreas is comprised of two major constituents; exocrine cells, which possess a digestive function, and endocrine cells, which secrete hormones[24]. The exocrine pancreas comprises approximately 98% of total pancreatic mass and primarily consists of acinar and ductal cells[24, 25]. Interestingly, ductal cells are the most likely of all exocrine and endocrine pancreatic cells to develop a malignancy, whereas acinar cells are the least likely[25].

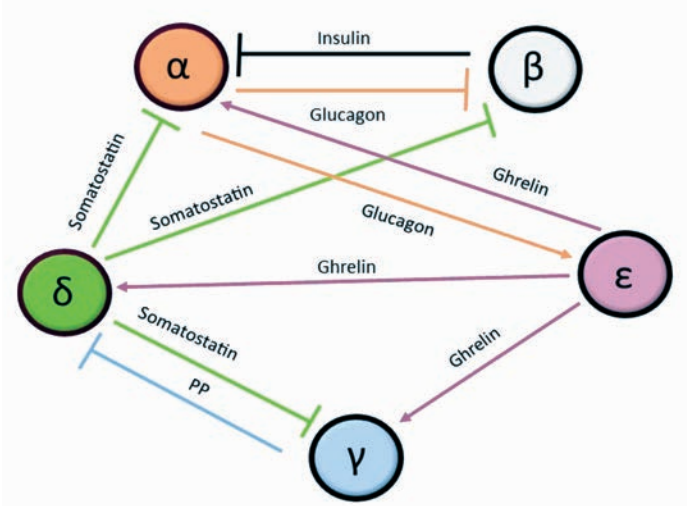


***Figure 3** – Five cell types make up the islets of Langerhans: alpha cells, beta cells, delta cells, epsilon cells and pancreatic polypeptide/gamma cells. Alpha and beta cells secrete glucagon and insulin, which act antagonistically to maintain glucose homeostasis [27, 28]. Glucagon can also induce ghrelin release from epsilon cells[29]. Delta cells secrete the hormone somatostatin which can prevent insulin, glucagon and PP secretion[30]. In turn, PP secretion can inhibit somatostatin secretion[31]. Finally, the hormone ghrelin, as secreted by epsilon cells, can induce glucagon, somatostatin and PP secretion [32, 33].*

The endocrine component makes up the remaining 2% of the pancreas[24]. The endocrine pancreas consists of the islets of Langerhans, containing five different cell types, primarily responsible for secreting hormones involved in maintaining glucose homeostasis (Figure 3)[24, 26]. Alpha and beta cells are the most common islet cells, comprising approximately 90% of islet mass[26]. Delta, pancreatic polypeptide (PP) and epsilon cells comprise the outstanding 10% of islet mass[26]. The alpha and beta cells are responsible for secreting glucagon and insulin, respectively, allowing for tight control blood glucose levels[26]. Ghrelin-secreting epsilon cells, pancreatic polypeptide-secreting PP cells, and somatostatin-secreting delta cells also play a role in mediating glucose homeostasis, via the regulation of insulin and glucagon secretion, among other functions[26].

## Autophagy and the exocrine pancreas

As stated previously, the exocrine pancreas consists of cells including acinar cells and ductal cells. The main function of acinar cells is to secrete digestive enzymes, such as amylase, and therefore possess an elevated biosynthetic capacity[34]. However, increased production of digestive enzymes dictates that acinar cells remain highly susceptible to the development of endoplasmic reticulum (ER) stress, due to a build-up of misfolded proteins[34]. Previous research has implicated autophagy within the preservation of acinar cell function; a basal rate of autophagy has been suggested to be beneficial for acinar cells, as to reduce the development of ER stress and thus protecting against potential loss of function[34].

Furthermore, autophagy has been linked with several diseases in the exocrine pancreas. Pancreatitis can be classified as both acute and chronic, however, impaired autophagy has been linked with both types. In acute pancreatitis, impaired autophagy has been linked to the activation of trypsin in the pancreas, which plays an important role in the development of the disease[35]. It has also been observed that a double-knockout of lysosomal proteases Cathepsin B and Cathepsin D in mice, caused acinar cell cytoplasmic vacuolization, accompanied with an increase of the autophagy markers microtubule-associated protein 1 light chain 3 (LC3) and sequestosome 1/p62 (p62), culminating in the development of chronic pancreatitis and the implication that autophagy had become defective[36].

Pancreatic ductal adenocarcinoma (PDAC) is the most common type of pancreatic malignant tumour, accounting for approximately 90% of cases[37]. PDAC is a disease possessing a high mortality rate, with a 5-year survival rate of approximately 8%[37]. PDAC can be cured using treatments involving pancreas resection, but treatment is dependent on the stage of the disease; it is estimated only around 1/5 PDAC patients can be treated via resection[37, 38]. It has been noted that autophagy is elevated within late-stage PDAC; increased autophagy in cancer cells allows for the maintenance of a high metabolic rate[39]. Interestingly, it has also been demonstrated that when autophagy is inhibited, PDAC can then promote macropinocytosis, in order to scavenge nutrients from extracellular deposits[39]. Therefore, more research is required into the effects of autophagy on PDAC, and how to mitigate the pro-survival effects of autophagy inhibition on the cancer.

## An introduction to pancreatic beta cell autophagy

The primary function of beta cells is to secrete insulin in response to changes in blood glucose concentrations, in order to maintain glucose homeostasis[26]. Due to their function, beta cells are exposed to a variety of stressors, which may impact upon survival, unless relieved by processes such as autophagy. For example, the high energetic demand placed upon beta cells to synthesize the required amounts of insulin, results in the induction of ER stress[40]. It is known that the process of autophagy and the ER are interconnected; it has been suggested that the ER is involved in the generation of isolation membranes, during the preliminary stages of autophagy[41, 42]. Furthermore, ER stress has also been demonstrated to be an inducer of autophagy[41].

In addition, the unfolded protein response (UPR) has also been linked with autophagy[41, 43]. Studies have noted that UPR-associated genes are downregulated in autophagy-related 7 (Atg7) deficient mouse beta cells, suggesting that autophagy deficiency negatively impacts upon UPR functionality[41]. In addition, islet cells derived from mice with Atg7 deficient beta cells displayed increased levels of cell death, in contrast to healthy controls, due to induced ER stress, and potential UPR malfunction[41]. Autophagy, as a basal process, could therefore act as a pro-survival process in beta cells.

Due to their function in insulin secretion, beta cells are unique in how they respond to nutrient stresses, such as starvation. For most eukaryotic cells, starvation is a potent inducer of autophagy[44, 45]. However, research suggests that in beta cells, starvation acts to inhibit autophagy. As described previously in this review, autophagy is a degradative process where catabolites are 'recycled' by the cell and can then be metabolised for cellular energy[46]. However, it is this autophagic production of cellular energy, that can abnormally promote insulin secretion in a nutrient starved environment, exacerbating further hypoglycaemia[46]. In a 2015 study by Goginashvili et al., it was suggested that beta cells employ the process of starvation-induced nascent granule degradation (SINGD), in order to inhibit autophagy during periods of starvation. In essence, SINGD involves the targeted degradation of nascent insulin secretory granules by lysosomes during periods of starvation, resulting in the degradation of proinsulin[46]. SINGD also allows for the inactivation of autophagy, via stimulation of the mTOR signalling pathway, ultimately suppressing insulin release during starvation[46].

## Pancreatic beta cell autophagy and diabetes

Beta cells are the most common pancreatic cell type studied in relation to autophagy and diabetes. The role of beta cell autophagy has been studied within both T1D and T2D, with most research focussing upon T2D, although some recent studies highlight a potential role in T1D.

Nutrient excess, in the context of T2D, can trigger the development of insulin resistance, and culminate in beta cell dysfunction and demise[47]. More specifically, it is lipotoxicity that has been observed to contribute towards the beta cell dysfunction observed in T2D[48]. Chronic exposure to free fatty acids, including palmitate, can induce cellular injury, however, autophagy has been observed to protect beta-cells against the detrimental cellular effects induced by palmitate exposure[48]. Furthermore, pharmacological inhibition of autophagy, via chloroquine, in INS-1 cells exacerbated palmitate-induced cellular death[48]. In this sense, autophagy can be viewed as a 'pro-survival' pathway for beta cells exposed to high-fat environments.

Lipotoxicity can also trigger other cellular stressors, such as oxidative stress[49]. Oxidative stress is present within the pathogenesis of both T1D and T2D and can contribute to the development of beta cell dysfunction[50, 51]. It has long been noted that beta cells possess low levels of antioxidant enzymes, such as catalase, which ultimately contributes towards the susceptibility of beta cells to oxidative stress, via reactive oxygen species (ROS)[50]. A 2018 study by Marasco et al., demonstrated an interaction between the antioxidant response and autophagy within beta cells, via the cytokine interleukin 6 (IL-6)[51]. IL-6 knock out mouse beta cells displayed both an increase in oxidative stress, via 4-hydroxynononeal staining, and a reduction in beta cell mass[51]. IL-6 was also observed to incite autophagy within beta cells, demonstrated by elevation of LC3-II and a reduction in p62 levels, after fifteen minutes[51]. Ultimately, Il-6 stimulated a reduction of cyclic adenosine monophosphate (cAMP) levels and an increase in transcription factor nuclear factor erythroid 2-related factor 2

(NRF2), resulting in mitophagy induction, thus combating oxidative stress[51].

The development of ER stress can also be associated with diabetes. ER stress has previously been linked with dysfunctional autophagy as a driver of beta cell dysfunction[52]. A 2012 study by Quan et al., revealed that crossing ob/ob mice with beta cell specific Atg7 knock out mice resulted in the development of diabetes; Atg7 knock out mice alone did not develop diabetes[52]. Similarly, a different study noted how T2D human islets that were exposed to the autophagy inducer rapamycin ameliorated the disadvantageous changes to ER ultra-structure caused by palmitate exposure[53]. It is worthy of note that other studies have suggested that chronic ER stress alone does not directly correlate with diabetes development, it is rather that inadequate UPR responses contribute towards beta cell dysfunction and ultimately diabetes development, under high-fat diet (HFD) conditions[54]. As such, the role of ER stress coupled with autophagy, remains an interesting topic to research within the field of diabetes.

In the future, beta cell autophagy could become an attractive target in the treatment of T2D. Manipulation of autophagy in order to combat factors such as nutrient excess, as well as cellular stressors, may provide a mechanism to preserve and protect functional beta cell mass, that may have been lost during normal disease progression.

## A link between diabetic nephropathy and autophagy?

Diabetes is a disease that is known to adversely affect many organs within the body, including the kidneys. Diabetic nephropathy (DN) can ultimately lead to the development of end-stage kidney disease, which can be fatal for patients if left untreated[55]. DN involves the presentation of both microalbuminuria and proteinuria[55]. Podocytes aid in the prevention of proteinuria development via their function in the glomerular filtration barrier[55,56]. It has previously been noted that the basal rate of autophagy in podocytes is high, compared with many cell types, possibly due to their need to maintain cellular homeostasis in lieu of cell division[55,56]. It has been suggested that if homeostasis is not maintained, and podocytes become dysfunctional, proteinuria may occur, which is associated with DN[55,56].

A 2018 study determined that spironolactone treatment, in combination with insulin, significantly restored LC3B and Beclin1, in DN mice[56]. This suggests that spironolactone treatment possesses a stimulatory effect upon podocyte autophagy in mice with DN, ultimately resulting in the reduction of podocyte injury, as evidenced by factors including a reduction in the thickening of glomerular basement membranes[56]. Similarly, the compound puerarin has also been studied to determine any potential affects that the treatment may possess with regards to podocyte autophagy, in the context of DN[57]. Puerarin is a compound derived from radix puerariae, and it has been demonstrated that heme oxygenase 1 (HMOX-1) expression can be increased in diabetic mice kidneys, via puerarin treatment[57]. HMOX-1 is an enzyme that can stimulate the AMPK signalling pathway, and induce autophagy, in times of cellular oxidative stress[57]. Puerarin treatment restored LC3B levels, as well as reducing p62, in DN mouse podocytes[57]. It could therefore be suggested that puerarin treatment stimulates autophagy in DN mouse kidneys[57].

ATF4 is a transcription factor involved in the PERK-eIF2alpha-ATF4 signalling pathway, which is known to initiate autophagy[58]. Apoptosis was observed to be elevated in ATF4 knockdown podocytes, subjected to serum of mice with DN[58]. Similarly, ATF4 knockdown in podocytes, also exposed to serum from mice with DN, caused a decrease in LC3 punctate, compared to podocytes treated with the serum alone[58]. The previously observed decrease in LC3 punctate, via ATF4 knockdown and serum treatment, was restored by treatment with rapamycin, suggesting ATF4 aids in the regulation of autophagy induction[58].

## Conclusions

To conclude, it is evident that autophagy is an important cellular process that is required to maintain the basic functionality of many cell types, including those in the pancreas and kidney. It has been demonstrated that the dysregulation of autophagy may exacerbate cellular dysfunction in several pancreatic diseases, including pancreatitis and diabetes mellitus [35,36,52]. Manipulation of autophagy in various pancreatic cell types could, therefore, provide an attractive target in the treatment of several diseases, including T2D. It is predicted that 5.5 million people will have diabetes in the U.K. by 2030 [59]. Therefore, it is important that future research is conducted into the potential therapeutic effects of autophagy manipulation, with focus placed upon pancreatic disease.

*Acknowledgement: This research was kindly funded by Diabetes UK.*

### Learning Points

- The prevalence of type 2 diabetes is rising at a rapid rate and is on course to become a major public health concern over the next 30 years.
- Autophagy is a cellular recycling process that is regulated by the AMPK and mTOR signalling pathways.
- Dysfunctional autophagy has been associated with several intrapancreatic diseases, including pancreatitis, pancreatic ductal adenocarcinoma and type 2 diabetes.
- More research is required into the effects of autophagy manipulation to treat diseases, such as diabetes mellitus and pancreatic ductal adenocarcinoma, for such treatments to be utilised in clinical settings.

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A complete listing of all the references in this article is available from the Editor upon request, or please e-mail the author.

# Health Management *in times of Disaster*

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*Disasters and other emergencies often result in significant impacts on people's health, including the loss of many lives. Every new threat reveals the challenges for managing health risks and effects of emergencies and disasters.*

Natural, biological, technological and societal hazards put the health of vulnerable populations at risk and the potential to cause significant harm to public health. Examples of these hazards are as follows:

1) Natural: *earthquake, landslide, tsunami, cyclones, flood or drought*
2) Biological: *epidemic disease, infestations of pests*
3) Technological: *chemical substance, radiological agents, transport crashes*
4) Societal: *conflict, stampedes, acts of terrorism*

Disasters, emergencies, and other crises may cause ill-health directly or through the disruption of health systems, facilities and services, leaving many without access to health care in times of emergency. They also affect basic infrastructure such as water supplies and safe shelter which are essential for health.

Many lives can be saved in the first hours after an emergency through community response before external help arrives.

Health systems are composed of public, private and nongovernmental facilities which work together to serve the community; these include primary health care centres, field hospitals, general hospitals, speciality hospitals, laboratories, pharmacies and blood banks. Safe hospitals programmes ensure health facilities are safely built to withstand hazards, remaining operational in emergencies.

In order for the health of the population to be protected during and after a disaster, wider determinants of health such as water, sanitation, nutrition, shelter and security also need to be adequately addressed through multi-sectoral working.

Various risk factors for human vulnerability to disaster-related morbidity and mortality include the following – low income, low socioeconomic status, lack of home ownership, single-parent family, older than 65 years, younger than 5 years, female sex, chronic illness, disability, social isolation or exclusion.

## An all-hazards health emergency and disaster risk management programme could be expected to have the following component:

Capacities in health and related technical disciplines / domains for disaster risk management at community, sub-national and national levels, for example: child health, maternal and new-born health, management of communicable diseases, chemical incidents, radiation emergencies, mass casualty management, mental health and psycho-social support, mass fatality management, nutrition, sexual and reproductive health, trauma care, water, sanitation and hygiene.

The senior author has spent 20 years training in Royal Army Medical Corps in wars in establishing the Field Hospitals. He has tried to establish Field Hospitals in Civilian Major Disasters. In the Covid-19 pandemic globally, Field hospitals have been established to save civilian patients.

## Field Hospitals

All major disasters need medical cover. Field hospital is a temporary hospital or mobile medical unit that takes care of casualties on-site before they can be safely transported to more permanent facilities.[1] During wars Field hospitals supported the battle line and served in every capacity, from disease hospital to resuscitation centre to acute care (with surgical reinforcement) to reserve and rest status. Their job was triage, stabilization, and evacuation to the base hospitals. This term was initially used in military medicine (such as the Mobile Army Surgical Hospital, or MASH), but it is inherited to be used in disasters and other emergency situations like what has happened during the Covid-19 pandemic. [2]

Field hospitals should ideally be established on plain ground and should remain within one to two miles distance from the major disaster, or if suitable large buildings are available.

Field hospitals have canvas tents and pre-prepared containers which provide all kinds of necessary equipment according to the need of the individual department. These containers are dropped near each department. Prefixed providers for field hospitals can be the Army, Governments and Charities.

## Pre-prepared containers

Each field hospital has an accident and emergency department which includes 10-15 recess bays for triage, to provide first aid and resuscitation, and a plain X-ray facility within the A & E.

Depending upon the nature of the major disaster, Field hospitals need a large number of staff to cover both the day and night duties, including surgeons, physicians, orthopaedics, anaesthetics, facio-maxillary surgeon, neuro surgeons, neurologists,

*Field Hospitals*

psychiatrists, gynaecologists, pathologists, radiologists, urologists, dentists, paediatricians, nurses, priests , stretcher bearers, porters, theatre assistants, radiographers, physiotherapists, lab technicians, and a support team of other assistants for allied works - engineers, electricians, cooks and security.

A validation exercise for training is essential before establishing any field hospitals.

My own experience for validation is that I found lack of manpower for 200 bedded hospitals. I trained double manpower to make the duty schedules for day and night shifts.

Flexible and compressive medical and diagnostic services are required in any major disasters or any army and civilian causalities.

**Rule one:**
- Immediate care by colleagues

**Rule two:**
- More advanced care with resuscitation and some surgical and diagnostic facilities by the close support from general hospitals.
- Health management in times of disaster using own available resources
- Evacuation of facilities for serious casualties to any general and speciality hospitals

**Rule three:**
- Advanced treatment provided by the field hospital

The introduction of digital radiographic imaging and hand held ultrasound equipment has meant that use of diagnostic imaging can now be utilised ensuring causalities injuries diagnosed and treated within the golden hour after injury. Now CT and MRI are also available in field hospital and also in the most general hospital.

Disasters occur unexpectedly and cause chaos, physical, psychological illness, financial loss and loss of life. Any disaster remains a nightmare throughout our lives; the loss of life of our beloveds and friends, and especially the loss of parents and children.

In a disaster you need prompt medical cover that saves lives. First golden hour resuscitation and basic life support save 60% of life through advanced trauma life support and airway management, for example in shock, thoracic, abdominal, head, maxillofacial, spine and spinal cord, limbs, burn and ophthalmic injuries.

## Mass Casalities Triage

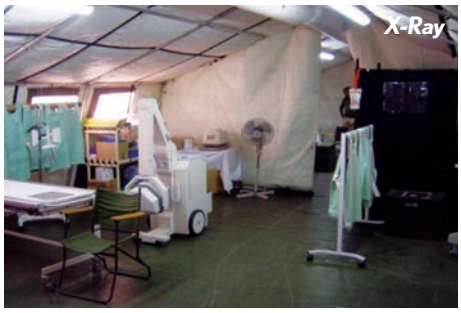
**T1 P1 Immediate Treatment** - Those needing emergency life-saving treatment procedure should not be time consuming and concern only those with a high chance of good quality survival. Examples are remedial airway obstruction, accessible haemorrhage and emergency amputation.

**T2 P2 Delayed Treatment** – Those needing major surgery (after sustained treatment such as intravenous fluid, anti-biotics and splinting) or medical treatment but where conditions permit delay without endangering life. Examples are open fracture long bones, large joint dislocation and burns covering 15-30% body surface area.

**T3 P3 Minimal Treatment** – Those with relatively minor injuries who can effectively take care of themselves or be helped by untrained personnel.


Pre-prepared Containers


Surgical Theatre


X-Ray


Ultrasound


Ward

Examples are minor laceration and uncomplicated fractures.

**T4 P1 Hold - Expected treatment** - Those with serious multiple injuries needing extensive treatment or with poor chance of survival. Those causalities receive supportive treatment compatible with resources for example, analgesia. Examples are severe head and spinal injuries, extensive burns and large dose of radiation.

Dead is still dead.

**Type of shock** - Hypovolaemic, cardiogenic, neurogenic, anaphylactic and septic shocks

Hypovolaemia is the cause of shock in most major disaster. Management require immediate control of haemorrhage either by direct compression, splintage, the application of a tourniquet or where necessary urgent surgery.

**Evacuation:** When primary treatment has been completed, evacuate the causality to a specialist unit of definitive treatment. Causalities without a definitive airway should be evacuated in the three quarter prone and must be carefully supervised.

## Conclusion

From a military perspective, field hospitals were sites for the re-construction and further "improvement" of soldiers' bodies, minds and identities. Their main military purpose was to send as many patients as possible back to the front or to find ways of enabling them to contribute to the war effort in a civilian job or they were sent back to hospitals at home for further treatment, convalescence and rehabilitation. This ethos has paved the way for establishment of an International Strategy[3]. The global Covid-19 pandemic has highlighted the importance of these field hospitals

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## Biography – Dr Deep Chand

**Dr Chand had a distinguished career, as a consultant radiologist and the Territorial Army (TA) where he rose through the ranks to become a lieutenant colonel. He and his team created a radiology department in the 202 Field Hospital, Birmingham. Dr Chand served in both Gulf wars. During the latter, he ran a 200-bed military hospital over six months in Basra. Working on the frontline Dr Chand and his colleagues provided life-saving care by introducing the use of ultrasound.**

**Dr Chand has received several awards recognising his outstanding career and for his service to Queen and Country. These awards include being admitted into the Order of St. John, the Territorial Decoration Award for long service, as well as being appointed as a Fellow of the Royal Society of Medicine.**

## From the Editor's Desk

The on-going COVID-19 pandemic led to the establishment of field hospitals in many parts of the world, especially in the developing world. In the UK, as we all know, "Field hospitals" were designed to support the NHS during the coronavirus pandemic. There were 19 in Wales in 2020. In England there were seven critical care NHS Nightingale Hospitals, as part of the response to the COVID -19 pandemic, the first one being in London, which opened first on 3 April 2020.

They provided extra bed capacity but they also helped normal hospital services to restart and support social care services. It is a testament to the NHS field hospital staff that the majority of patients have received positive outcomes and reported positive patient experiences, saying they felt safe and involved when decisions were made about their care and treatment.

# Technological developments in minimally invasive spine surgery for metastatic spinal tumours

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**Pragnesh Bhatt** Department of Neurosurgery, Aberdeen Royal infirmary, Aberdeen, AB25 2ZN Scotland.

## Introduction

Spinal tumours are the most common bony metastasis, usually affecting the vertebral body that spread from the breast, lung, or prostate. They are more likely to involve the thoracic or lumbar spine compared to the cervical segments [1]. The tumour will generally involve the vertebral body with possible extension posteriorly into the pedicles. The common indications for surgery are spinal instability, intractable back pain and high-grade spinal cord compression. The need for total tumour resection has been greatly reduced since the introduction of stereotactic body radiation therapy as it reliably reduces tumour volume [2]. Consequently, separation spinal surgery is being performed to create a circumferential boundary between the spinal cord and tumour of at least 6 mm for more effective postoperative radiotherapy dosimetry to limit damage to the spinal cord [3]. Breakthroughs in radiation therapy have driven the need for minimally invasive spine surgery (MISS) as more procedures are being performed on an outpatient basis and there are increasing demands for shorter hospital stays [4].

Several comparative studies have shown MISS to be as effective as open spinal surgery for tumour decompression with benefits such as reduced operative blood loss, shorter operating time, reduced postoperative pain and shorter hospital stay [5-7]. This could allow patients to be started on postoperative radiotherapy earlier than with open surgery as there are fewer wound related complications leading to better cancer care [2, 5]. Patients with short term survival are often not considered for surgery. As a result, decision making should be guided by prognostic scoring systems such as Tokuhashi, Tomita, spinal instability neoplastic score (SINS) or the neurologic, oncologic, mechanical, and systemic (NOMS) decision framework to select appropriate patients for surgery [2, 8]. This review aims to highlight the technological developments that have taken place in MISS with regards to spinal decompression, spinal stabilisation and surgical navigation for the management of metastatic spinal tumours.

## Spinal Decompression

### Tubular retractor technology

Tubular retractors have become a major tool for MISS techniques as they can be used completely on their own as the working channel or they can be expandable allowing for mini-open approaches. Tubular retractors are cylindrical tubes that have a fixed diameter. After making a paramedian incision, the Wilste approach is used to separate paraspinal muscles along the surgical plane using progressively larger tubes, until the desired view of the lamina is gained. The tube with the appropriate length and diameter for that space is docked to keep the tissues retracted maintaining the surgical corridor [5]. The adoption of tubular retractors has increased as it can be used in conjunction with conventional surgical instruments like drills and rongeurs that are used in open surgery [5, 9].

Spinal metastases are focal pathologies affecting specific vertebrae. Open spinal surgery with posterior approaches requires long midline incisions and extensive paraspinal muscle dissection, which exposes unnecessary spinal segments. By using tubular retractors smaller incisions can be made thereby reducing soft tissue injury and maintaining the normal anatomy of the site. This reduces wound related complications like infections, blood loss and inadvertent durotomy causing cerebrospinal fluid leaks [5]. The tubes can be used to expose specific vertebrae reducing muscle detachment, which allows for quicker healing and reduction in postoperative pain [5, 10]. In cases where metastasis affects multiple spinal levels, tubular retractors can be used at each site. These tubes can be tilted along the lamina for instruments to access both sides of the vertebrae from a single midline incision such as for bilateral screw placement [9].

### Endoscopic decompression

Endoscopic techniques are not commonly used in the treatment of spinal tumours as there is limited ability to control bleeding [11]. At present, endoscopy can be performed using full endoscopic systems where the same tubular device contains channels for optics, irrigation and instrument to be used for transforaminal approaches. A less commonly used method is the micro-endoscopic system, where a thin rigid endoscope attached to tubular retractors can be used to visualise the narrow surgical corridor in posterior approaches. Endoscopes present an opportunity to further decrease the invasiveness of surgery through smaller ports, which could allow for tumour removal under local anaesthesia making it more practical for elderly patients at risk of general anaesthesia and further shortening hospital stay [11, 12].

## Spinal Stabilisation

### Bone augmentation

Osteoporosis from a metastatic tumour invading the vertebral body decreases the load-bearing capacity of the vertebrae leading to vertebral compression fractures (VCF). This causes severe pain and spinal instability that limits the quality of life. Open surgery to stabilise VCFs is extensive, requiring internal fixation which carries a high risk of failure as these patients already have poor bone quality due to advanced age. Percutaneous vertebroplasty is a minimally invasive technique where polymethylmethacrylate bone cement is directly injected at the fracture site by a transpedicular approach guided under fluoroscopy. As the cement hardens, it increases the height of the vertebral body resisting further compression [13, 14].

Kyphoplasty is a variation upon the percutaneous vertebroplasty to reduce complications related to cement extravasation and cement toxicity. In this procedure, balloons are inflated after insertion into the bone to improve the kyphotic angle and the cavity is filled with bone cement. This results in better restoration in the height of the vertebral body and fewer complications [13, 14]. Both techniques have been proven to be safe and efficacious for pain relief in several high-quality randomised trials, but the choice is down to surgeon or centre preference [13].

### Percutaneous stabilisation

Spinal stabilisation is necessary when mechanically unstable fractures extend into the posterior elements of the vertebrae or if vertebral fragments are present in the vertebral foramen. SINS has become a widely adopted scoring system to determine spine instability for treatment algorithms. Total scores can be classified into stable (0- 6), potentially unstable (7- 12) and unstable (13- 18) with surgery being favoured for scores above 7. Mechanically unstable fractures that do not affect the posterior elements can be treated with bone augmentation alone. When vertebral fractures extend into the posterior elements then extensive support is required by using percutaneous stabilisation and kyphoplasty. When vertebral fragments are displaced posteriorly there is a higher risk of canal compromise and neurological deficit from further displacement of the fragment, therefore bone augmentation should be avoided (Figure 1) [2, 10].

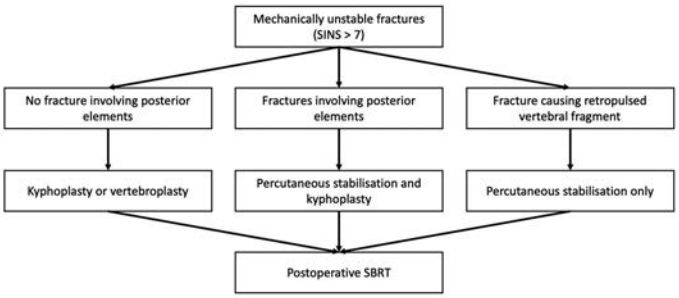


***Figure1:*** *Schematic of the use of bone augmentation procedures and percutaneous stabilisation based on fracture ,orphology. Abbreviations: SINS: Spinal instability neoplastic score, SBRT: Stereotactic body radiation therapy. Adapted from Barzilai et al* [2].

During percutaneous stabilisation, pedicle screws are inserted through small incisions. This creates parallel stab incisions over each vertebral segment. The screws will be inserted through the pedicles into the vertebral body with appropriate trajectory checked using pedicle probes and radiographic confirmation (Figure 2). Next, the rods can be threaded through the screws and displacement of the spine can be reduced by tightening the screws and bending the rods to fit the shape of the spine. The ideal length of screw and rod constructs can be difficult to determine as it needs enough segments above and below the point of the fracture to provide robust support from compressive forces while avoiding stiffness [4,10]. Severely unstable fractures can cause adjacent vertebrae to lie in different orientations, requiring different trajectories into the pedicle screw on each side. This presents a challenge as instrumentation can cause further displacement during fixation. This problem has been reduced with the use of pedicle screw systems like Sextant (Medtronic) and Mantis (Stryker) that hold multiple screws as a single fixed structure while the rod is being threaded through and tightened to reduce displacement achieving better spinal fixation [4,10,11]. Over time screws have been designed with new features and material such as fenestrated screws that can be injected with bone cement after placement to increase pull-out strength and carbon fibres screws that are radiolucent on MRI and CT imaging for better monitoring of tumour recurrence [8,11].

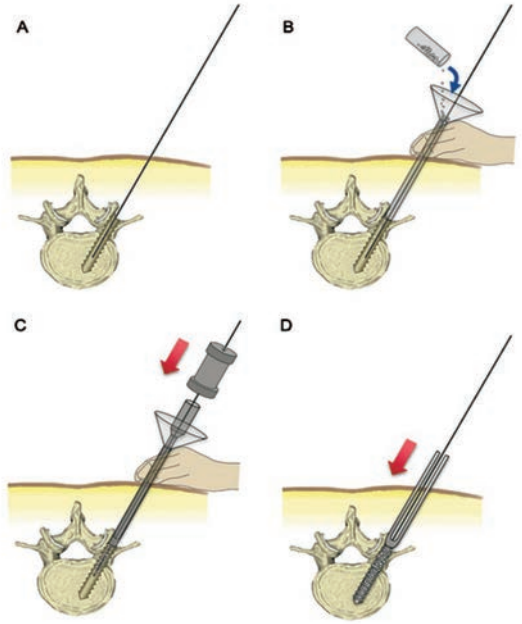


***Figure2:*** *Surgical procedure of percutaneous pedicle screw insertion. Adapted from Kanno et al* [15].

## Surgical Navigation

As minimally invasive procedures have become more popular, the surgeon is able to visualise less of the surgical site. Therefore, systems are needed that can allow for accurate calculations to be made in real-time to understand the local anatomy for instrumentation [2]. Currently, intraoperative image guidance through fluoroscopy using the C-arm or computed tomography using the O-arm can be used to show the likely trajectory of the pedicle screw from multiple views reducing the risk of misplacement [10,16,17]. These navigation systems have gained widespread adoption with studies demonstrating accuracy in screw placement of 98%, reduction in screw placement times and reduction in reoperation [2,10]. Similar to neuronavigation used in intracranial pathologies more tools like drills, probes and curettes could be guided to the site of the spinal metastasis for a smaller surgical footprint [2].

Even with the appropriate use of image-guidance systems, human error and technical challenges of the procedure could still lead to neurovascular injury from misplacement of instruments [17]. Combined innovations in robotics, artificial intelligence and image guidance systems could lead to more products like the SpineAssist (Mazor robotics) and Rosa One Spine (Zimmer Biomet) for robotic-assisted spine surgery [2,10]. The robotic arm would allow for a 3D approach to be planned preoperatively that can be autonomously executed, while still giving the surgeon the ability to take control. This could reduce human error caused by mental and physical fatigue to the surgeon in difficult cases achieving better surgical outcomes [16]. The 3D tracking technology could make surgery more accurate in tumour excision with smaller margins and reduce intraoperative radiation exposure [17].

## Conclusion

The technological advancements that have taken place in MISS have allowed for sick patients that would previously be unsuitable for open surgery to receive treatment improving their quality of life. Even though the adoption of minimally invasive techniques remains slow the benefits of MISS to the patient like reduced postoperative pain and shorter hospital stay will likely increase its demand and drive innovation [10]. As a result, surgeons will need to be exposed to these tools early on in their training to develop the manual skills to work in a narrow space and be able to keep precise surgical orientation in a limited field of view [9].

## Take-home message

- MISS is beginning to be as effective as open spinal surgery for metastatic spinal tumours with benefits such as reduced operative blood loss, reduced postoperative pain, earlier postoperative radiotherapy and shorter hospital stays.
- Reduced disruption to local anatomy and image-guided surgical navigation tools allow for frailer patients to undergo surgery who otherwise would be unfit for open spinal surgery improving their quality of life.
- MISS could become the most effective technique for managing vertebral compression fractures through percutaneous stabilisation rather than needing extensive muscle dissection.
- Open spinal surgery is preferred over MISS when there is extensive tumour damage causing severe spinal cord compression to achieve a higher degree of decompression.

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# Supervision: Have we got it right?

**Professor David Brigden** University of Bolton. Undergraduate Tutor, School of Medicine, University of Liverpool.

***This article discusses what supervision is and the role of clinical supervisors, educational supervisors and senior colleagues in supporting students, trainees and colleagues in a range of contexts.***

Supervision has been defined in many ways but it is essentially a conversation between professionals aimed at promoting learning, reflective practice and improving patient safety and quality of care.

In medical education, a distinction is often made between clinical and educational supervision - namely performance and development.

Educational supervision is 'the provision of guidance and feedback on matters of personal, professional and educational development in the context of a trainee's experience of providing safe and appropriate patient care' (Kilminster et al, 2007). All doctors are required to have educational supervision across their whole training period (DOH, 2007). It should be aimed at helping learners develop self-sufficiency in acquiring skills and knowledge through meetings, observation of practice, assessments and the provision of pastoral care. It is important an educational supervisor flags up any concern at an early stage.

Clinical supervision, on the other hand, relates to the everyday supervision of a trainee's performance. It involves being there, looking over their shoulder and teaching 'on the job' with developmental conversations, regular feedback and the provision of rapid response to issues as they arise (DOH 2007)

All trainees should have a named clinical supervisor for each post who should tailor the level of supervision to competence, confidence and experience of the trainee. Clinical supervision is a core aspect of personal and professional development and lifelong learning. It should help practising professionals develop complex skills in the context of real practical issues and situations, which may include a variety of face to face professional encounters such as mentoring and coaching

In the day-to-day clinical context, educational supervision necessarily includes some aspects of clinical supervision because issues discussed by the educational supervisor and supervisee relate to issues in the clinical arena. Although educational supervision may cover some technical aspects of work, clinical supervision is the place where a wider range of issues around specific patients can be raised and addressed.

Most supervision conversations address three inter-related domains - cases, contexts and careers. The role of supervisor is clearer in some of these domains than others.

Clinical supervision can be particularly helpful in cases, which involve:

- ethical issues
- complex decision making
- dealing with angry and distressed patients (and their families?)
- handling complaints
- handling significant events
- patients presenting with somatisation
- patients with conditions where there is no clear diagnosis

Educational supervisors also need to be able to discuss clinical cases and know to whom the learner can be referred to discuss clinical issues requiring expert knowledge. Discussing clinical cases may identify / highlight behaviours through which educational needs are identified. These can then be included in a learning contract.

Clinical scenarios depend on the context in which they occur - who is involved and their interactions. Issues relating to context might include:

- professional / inter professional difficulties
- communication problems
- difficulties relating to teamwork
- conflicts about roles or boundaries
- differing expectations about care
- power, authority, money or politics

Educational supervisors need to understand the supervisee's work contexts as they relate to his / her learning needs, educational objectives and professional development.

Supervision conversations can often raise issues about careers, including further training needs, work conditions, job prospects and career aspirations and how to manage and delegate work. The educational supervisor's role is key here and he/ she should support the learner along their 'learning journey'.

Constraints to effective supervision include:

- lack of time
- worrying about the enormity of a problem
- need for appropriate training to carry out supervision
- embedded cultural attitudes (some clinicians traditionally work alone, taking individual responsibility,)
- training and supervision being given low priority
- fear of showing areas of weakness or need
- anxiety about appraisal and professional revalidation
- attitudes about 'policing' the profession

The process of supervision gives an opportunity for supervisees to reconstruct their view of a particular situation or issue through the supervisor asking questions to try to help them see things from a different perspective. It is essential in promoting professional development and ensuring effective clinical performance. Good and effective supervision is based on the core principles of mutual respect, a good working relationship and developing an open and honest conversation centred around the supervisee's educational and professional needs.

# BIDA Quiz January 2022

Answers on page 26

**Alireza Sherafat** National Academic & Research Lead, BIDA Student Wing

### Case 1:

A 68-year-old patient was diagnosed with advanced prostate cancer in a tertiary urology department. Due to severe underlying health conditions, the patient has not been deemed fit to undergo surgery. Hence, he is commenced on medical treatment with goserelin therapy. A few days after starting this treatment, he presented with a flare up of lower urinary tract symptoms. Following an initial assessment by the urology on-call doctor, it becomes apparent that he should have been prescribed another medication alongside goserelin to prevent this complication. What is the name and use of the missing medication?

a) Prednisolone- Oral steroid to reduce inflammation
b) Diclofenac – Non-steroidal anti-inflammatory medication
c) Paracetamol- Antipyretic
d) Flutamide- A synthetic antiandrogen
e) Oxybutynin- Inhibit muscarinic action of acetylcholine on bladder muscle

### Case 2:

A 69-year-old lady called her GP practice to be see seen by a GP as her husband was concerned that her skin colour had turned "yellow". Following an initial telephone assessment, she was booked to have a face-to-face appointment with the GP. As she walked in, the GP was able to elicit that she was presenting with jaundice. She did not complain of having any pain. Her past medical history was not significant, and her alcohol consumption was within the recommended limits (less than 14 units/week). On examination, her abdomen felt soft and non-tender, but her gallbladder was distended. She was referred to hospital urgently (under two weeks). She was diagnosed with a particular type of cancer and Whipple's procedure was performed to treat her cancer. Which tumour marker below can be used to monitor her cancer in the follow-up?

a) CEA b) AFP c) CA19-9 d) CA15-3 e) PSA

### Case 3:

A 17-year-old lady has presented for the fourth time in the last year with symptoms of sinusitis, nasal congestion and otalgia. She has also noted some blood in his sputum for a few times in the last week. Yesterday evening she noted some streaks of blood in her urine. Her family history is significant for rheumatological conditions. The GP suspects the potential diagnosis to be a type of vasculitis condition. What is the most likely diagnosis and what is the most specific test to detect it?

a) Wegener's granulomatosis; p-ANCA
b) Churg-Strauss syndrome ; c-ANCA
c) Wegener's granulomatosis; c-ANCA
d) Henoch-Schonlein Purpura ; CRP
e) Henoch-Schonlein Purpura ; ESR

### Case 4:

A 45-year-old gentleman presents with constipation and fatigue. He also complains of headache and loss of appetite. His past medical history is not significant. He is a non-smoker and drinks alcohol socially. He works as a plumber for the last 4 years. On examination, his abdomen is distended and non-tender. Bloods are sent and results are not available yet. What is the most likely cause of his symptoms?

a) Chronic constipation due to low-fibre diet
b) Lead poisoning
c) Colorectal cancer
d) Sigmoid volvulus
e) Hypercalcaemia

### Case 5:

A 23-year-old lady presents to A&E with excessive nausea and vomiting. She is currently eight weeks pregnant. She is referred to gynaecology team for assessment and admission. What is the most important follow-up appointment which needs to be offered after discharge?

a) Ultrasound scan to check for molar pregnancy
b) Blood test to check for iron deficiency anaemia
c) Urinalysis to check for protein
d) Blood test to check for sodium and potassium
e) ECG to check for heart block

### Picture Quiz:

The radiograph below belongs to a 91-year-old patient who lives in a care home. Her WHO performance status is 3. What is the best management option that we can offer?

A) Total arthroplasty B) Hemiarthroplasty C) Intramedullary Nail
D) External fixation E) Conservative management


# bida President's Cup Cricket Tournament 2021

**Dr Raghu Hegde** National Sports Coordinator, BIDA

Hosted by Wigan Division on 21st August, the final was played on a sunny afternoon between the present cup holders, North East Division and previous three-time winners Wigan Division.

North East Division batted first and, after losing two quick wickets to Wigan opening bowler Jawad Ahmed for eleven runs, made 91 all out in 28 overs. Their top scorers were their captain, C. Kalluri, who made 29 runs (not out), and P. Sonaware who made 28. Wigan's Ravi Badge took 3 wickets for 23.

With some brilliant batting, in reply Wigan made their target of 92 in just 18 overs, losing only two wickets. Top scorer was the fifteen-year-old opening batsman Adithya Badge, who scored 39 runs and , and Ravi Badge, who made 33 not out. For NE Division the standout bowler was teenager Rafi Ahmed, who took two wickets for thirty runs.

After the match BIDA National President, Dr. B K Sinha praised both teams for playing good cricket with good sportsmanship. President Sinha then presented the trophy to the Wigan captain, Amit Anand. The man of the match on Wigan's team was Adhitya Badge for scoring 39 runs and taking two excellent catches. Man of the Match from North East team was Rafi Ahmed..

# Malignant Otitis Externa: A Summary

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## Introduction

Malignant Otitis Externa (MOE) is a rare [1,2,3] but potentially life threatening[4-7] infection of the external auditory canal (EAC) which spreads to temporal bone[3,5-7] and progresses into skull base osteomyelitis.[5,7] Although it is called malignant otitis externa, it is not a malignancy. It is also known as Necrotising Otitis Externa (NOE). Incidence of MOE is estimated to be about 10 cases per million per year.[8]

A high index of suspicion, early diagnosis and aggressive management are the keys for reducing morbidity and mortality associated with MOE.[1,6,7] It is therefore essential that all healthcare professionals involved in the assessment and management of patients with otitis externa are aware of MOE.

## Anatomy

On average, the EAC is 24 mm in length. The outer third (8mm) of the EAC is cartilaginous and the inner two thirds (16mm) is bony (Figure 1).[9,10] MOE is known to spread via the osseo-cartilagenous junction of the EAC[4] and Fissures of Santorini, in the cartilaginous EAC.[3,6]

The infection can spread anteriorly towards the cervico-fascial spaces, parotid gland and temporomandibular junction.[4] It can spread medially into the tympanomastoid suture,[6] petrous apex and skull base.[4] Posterior spread is towards the mastoid process.[4] Infection can also spread along fascial planes and venous canals.[6]

## Pathophysiology

The EAC is lined by stratified squamous epithelium above a layer of dense connective tissue[4]. MOE can lead to loss of this epithelium or ulceration, with extension of inflammation and bacteria into the underlying connective tissue layer that can be seen on histopathology.[4]

MOE is commonly caused by Pseudomonas aeruginosa[1-7] which is not a commensal of the EAC.[6] Other bacterial causes include methicillin-resistant Staphylococcus aureus[1,7] and Staphylococcus aureus.[2,3,5,6] Fungal[1] causes include candida species and aspergillus species,[4,5] particularly Aspergillus fumigatus.[6]

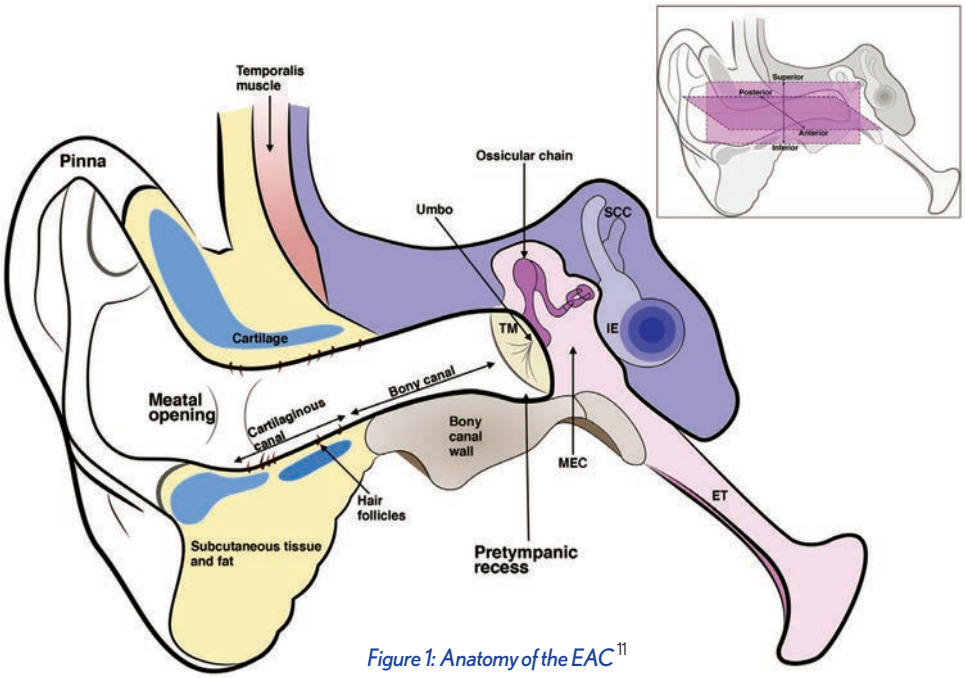


*Figure 1: Anatomy of the EAC*[11]

## Risk Factors

Diabetes is the most common risk factor for MOE.[1-7] Other risk factors include immuno-suppression,[1-4,6,7,12] advanced age[1,2,4,6] (aged over 50),[12] previous malignancy,[1,12] previous head and neck radiotherapy,[1] arteriosclerosis[1] or the presence of EAC polyps (on the inferior floor) or granulation tissue.[1,12]

## History

Otitis externa and MOE essentially share the same history and presenting symptoms: unilateral otalgia,[1] otorrhoea,[1] and, in later stages, some hearing loss.[1,6,7] Herein lies the difficulty deciding between the two conditions. Table 1 details red flag clinical symptoms suspicious for MOE.

| |
|---|
| Age above 50 years [1,2,4,6,12] |
| Immunocompromised state (diabetes mellitus, previous cancer treatment, history of immunotherapy)[1-7,12] |
| Chronic severe otalgia[4,7] disproportionate to examination findings[3,6] |
| Infection completely refractory to usual treatment[1,12] |
| Nocturnal otalgia[1,-6] |
| Headache[3,5,6] |
| Ongoing ear discharge[1-7] |

*Table 1. Red flag clinical symptoms suspicious for MOE*

Otoscopy and full cranial nerve examination is fundamental in any patient with suspected MOE.

Table 2 details ear examination findings suspicious for MOE.

| |
|---|
| Disproportional pinna and tragal tenderness[3,6] |
| EAC oedema[1,5-7] and discharge[6,7] |
| EAC polyps[1,4] and granulation tissue[1-7] |
| Exposed bone[6] on the floor of the EAC, especially at the osseo-cartilagenous junction[1,4,6,7] |

*Table 2. Ear examination findings suspicious for MOE*

## Differential Diagnosis

The main differential diagnosis is malignancy (squamous cell carcinoma) of the ear canal.[4,6] Other differentials are osteomyelitis, otomycosis,[4] Paget's disease, clival lesions, nasopharyngeal malignancy or granulomatous diseases.[6]

## Complications

Spread of infection to the skull base foraminae results in cranial nerve palsies.[3,6] The facial nerve[1,2,4,5,7] is the most commonly affected cranial nerve as the EAC is close to the stylomastoid foramen.[1, 3-6] Cranial nerves IX,[3,6,7] X,[3,6] and XI[3,6,7] are affected with spread of disease to the jugular foramen.[3,6] Medial spread involves the petrous apex and therefore cranial nerves II, III and VI.[6] Cranial nerve XII can also be affected.[2]

Other complications include ipsi- and contralateral skull base osteomyelitis,[1, 3-6] intracranial abscess,[1,2,6] meningitis,[1,2,6] Bezold's abscess[1,2] and venous thrombosis (internal jugular vein, sigmoid sinus or lateral sinus).[1,2,6]

## Investigations

***Ear swab:*** A deep sample should be taken for microbiology (culture & sensitivity).[1,4,6,12] If cultures are negative for micro-organisms, polymerase chain reaction assays could be used to try to detect bacterial or fungal components.[4] Fungal MOE should be suspected in patients with MOE symptoms with negative cultures who do not respond to appropriate antibiotics, or in patients who suffer recurrence after an initial response to antibiotics.[6]

***Histology:*** Any visible ear canal granulations / polyp should be biopsied and sent for histology to rule out underlying malignancy.[4,6,12] This can often be performed in the clinic under local anaesthesia.

***Serology:*** All patients should have full blood count, C-reactive protein and erythrocyte sedimentation rate (ESR),[1] which should be checked twice weekly.[1,2] Following a diagnosis of MOE, serial ESR can indicate response to treatment[1,6] and should be checked once weekly.[12] Renal function and liver function should be checked twice weekly[12] as high doses of antibiotics can be nephrotoxic and hepatotoxic.

***Imaging:*** CT scan of the temporal bones is the first-line imaging for diagnosing MOE [1,3,6,12] as it is good for visualising bony erosion of the skull base and EAC (Figure 2),[3,4,6] inflammation extension into adjacent structures and abscesses.[4,6]

Contrast MRI scan of the skull base should also be considered as it aids visualisation of soft tissue involvement [1,3,6] (Figures 3 and 4) and is particularly useful to assess osteomyelitis.[1] Patients should therefore have a baseline contrast MRI scan at the time of initial diagnosis.[1,2]

SPECT CT with technetium-99m methylene diphosphonate bone scan can be used for disease staging. Gallium nuclear imaging is useful for monitoring treatment response[3,6] and can aid decision-making regarding treatment completion.[1,6] Combined PET and CT imaging has been shown to be highly sensitive and specific for diagnosing osteomyelitis.[1]

CT and MRI scan should be repeated after 6 weeks to see response to treatment. However, we should bear in mind that radiological

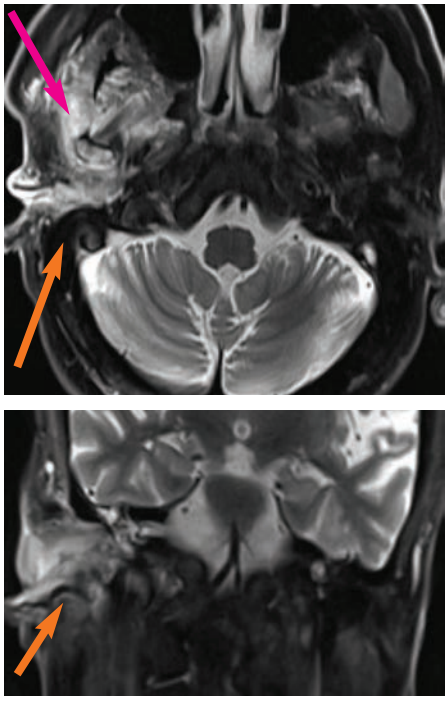

*Figure 3: MRI T2W image (axial and coronal): Appearances are similar to CT in keeping with malignant otitis externa (orange arrows mark oedematous soft tissue in the middle and external ear cavities), most likely inflammatory in aetiology, with possible involvement of the mandibular condyle (marked by pink arrow) in keeping with osteomyelitis.*

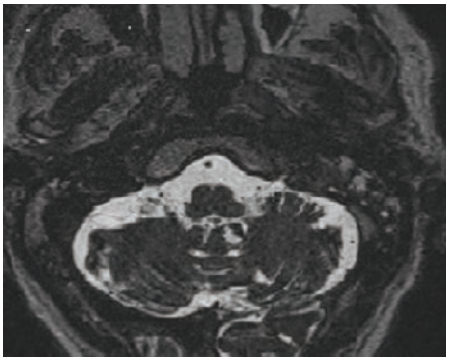

*Figure 4: Post-treatment MRI T2W image (axial): Significant interim resolution of inflammatory changes in the right middle ear cavity. Incidental fluid in left mastoid air cells.*

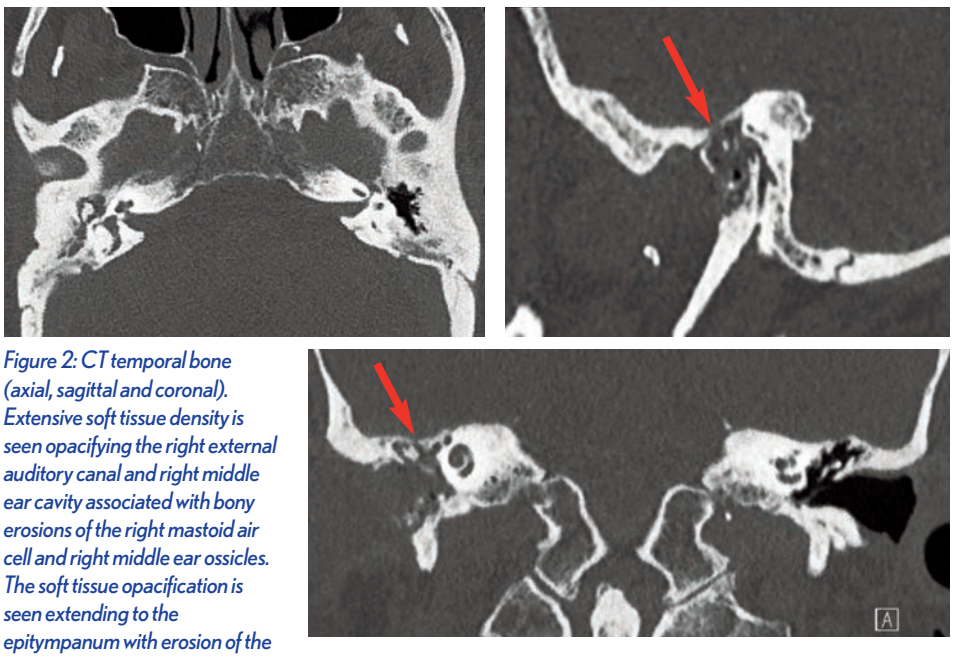

*Figure 2: CT temporal bone (axial, sagittal and coronal). Extensive soft tissue density is seen opacifying the right external auditory canal and right middle ear cavity associated with bony erosions of the right mastoid air cell and right middle ear ossicles. The soft tissue opacification is seen extending to the epitympanum with erosion of the right scutum. Rarefaction of the right tegmen tympani (marked by red arrows). Unremarkable left external and middle ear.*

changes take longer to normalise and can still be apparent despite disease resolution.[6]

## Treatment

Patients with MOE should be managed by a multidisciplinary team of ENT surgeons, microbiologists, diabetologists, acute and chronic pain teams, radiologists and histopathologists.[1,6,12] Skull base MDT discussion is advisable.

If the patient is diabetic, early involvement of the diabetes team is crucial as strict glycaemic control is essential.[1,2,4,6] If swallowing is affected due to cranial nerve involvement, the speech and language team[12] and dietician should be involved to ensure the patient is receiving adequate nutrition via an appropriate route. Ophthalmology and physiotherapy should be involved in patients with a facial nerve palsy.[12]

Patients with MOE often require several weeks of intravenous antibiotics, therefore early referrals should be made for long-term vascular access and, where available, community antibiotic team involvement.[12]

## Medical management

The mainstay of management of MOE is long term systemic and topical antibiotics,[1,6,12] Empirical antibiotics suggested by latest ENT UK guidelines (see Appendix 1) include dual treatment with IV ceftazidime and oral ciprofloxacin.[12] Pseudomonas aeruginosa is susceptible to fluoroquinolones, which have high oral bioavailability, good bone penetration and a smaller side effect profile in comparison with other antibiotic agents.[6]

Antibiotics should be tailored to the microbiology culture and sensitivity report[1,4,6] and all cases of MOE should be discussed with the microbiology team for decision-making about antibiotic choices.

In addition to long term systemic antibiotics, regular ear microsuction clearance[4,12] and usage of topical antibiotics is very important.[1,6,12]

One of the main presenting symptoms of MOE is significant otalgia which is disproportionate to clinical findings and poorly responsive to usual paracetamol/ibuprofen. Patients often wake up in the middle of night due to pain. Adequate analgesia should be one of the top priorities and should be prescribed as per the WHO pain ladder, with involvement of the acute or chronic pain teams if pain remains uncontrolled.[12]

Once the patient's pain and blood sugars are under control and inflammatory markers stabilise, they can be discharged home on intravenous antibiotics[12]. The community IV

team and microbiologists should be involved in this decision-making. Serial inflammatory markers should be checked routinely in the community.[12] Patient should also regularly attend ENT outpatients for review and aural care[12]. Clear and ongoing communication between the community team, ENT surgeons and microbiologists is paramount for a good outcome.[12]

Systemic antibiotics should be continued for at least 6 weeks[1,4,6,7,12] and only stopped once otalgia subsides and inflammatory markers normalise.[12] CT and MRI scans should be repeated at this stage. It is not uncommon for patients with MOE to receive 12-14 weeks of systemic antibiotics.

If the patient fails to respond to systemic antibiotics or starts deteriorating, ear granulations should be biopsied again to rule out underlying malignancy and CT/MRI scans may be repeated.[12]

Adjunctive hyperbaric oxygen therapy, if available, may be considered in patients with refractory[5,12] or advanced MOE,[5] or patients with intracranial complications.[6]

## Surgical Management

Surgery has a very limited role in management of MOE[1] and is reserved for local debridement, removal of bony sequestrum and abscess drainage.

## Conclusion

Malignant otitis externa has high mortality and morbidity if not treated promptly and aggressively. There should be a high index of suspicion of MOE in elderly, diabetic or immunocompromised patients who present with otitis externa with excruciating pain that often wakes them up in the middle of night and is out of proportion to the clinical findings. Multidisciplinary team approach towards managing patients with MOE is crucial for a good outcome.

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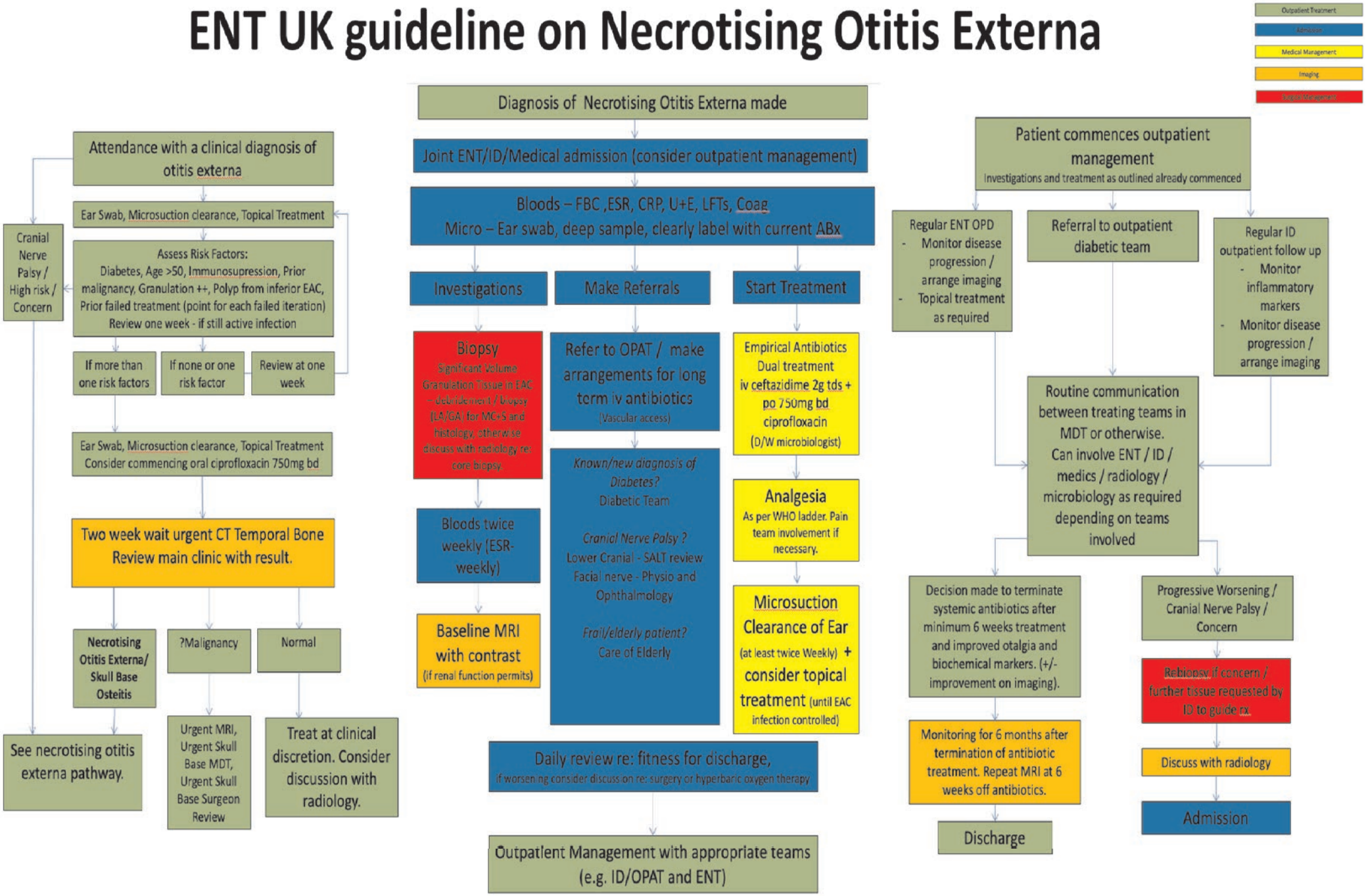
# ENT UK guideline on Necrotising Otitis Externa

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# Celebrating the first year of BIDA Student Wing

**Sai Ram Pillarisetti** Final Year Medical Student & President, BIDA Student Wing

Dear BIDA Members,

This issue marks 1 year since the inception of the BIDA Student Wing – the very first organisation dedicated to representing the voice of International Medical Students studying in the United Kingdom.

Within the past year, I am proud to say we have grown into truly international community of over 350 BIDA Student Wing members who come from 39 different countries! These figures highlight the diversity of the international medical community in the UK, which is representative of our ever-growing, multicultural NHS.

*"No one can whistle a symphony; it takes an orchestra to play it'*
H E Luccock

This quote rings true in the formation of BIDA's newest division - the BIDA Student Wing

I would like to extend my heartfelt gratitude to the entire BIDA Executive Committee for their unwavering support throughout the past year, particularly when I proposed the idea to start the student wing. I would also like to thank the entire Student Wing Committee for their hard work and diligent work ethic because without their incredible efforts, this initiative would not have been possible.

I would like to especially thank 3 members of the EC - Mr Amit Sinha, Mr Bhattacharyya and Dr Preeti Shukla for their hands-on support and guidance during our first year of operations. The entire student wing committee has thoroughly enjoyed working with them and we look forward to working on many more projects together in the future.

We started this organisation with the aim of providing a platform to unite all the international medical students in the UK while providing academic and wellbeing support. Within the span of just 1 year, we have conducted more than 30 free webinars for medical students on a range of topics from getting involved in academic research, OSCE revision as well as providing a structured exam revision series presented by junior doctors under the guidance and supervision of consultant doctors from the BIDA EC. We have several other webinar series planned this year and will be launching them closer to exam season.

Last year, we launched a fundraiser to support the families of health-care workers who died during the pandemic. This initiative was a grand success with several media outlets such as BBC Lancashire and LancsLive promoting our initiative.

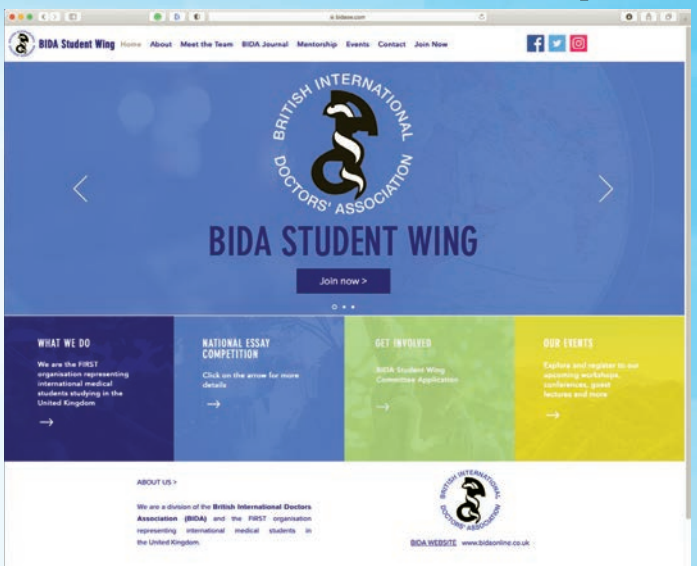
We have also formed a mental health sub-committee within the BIDA Student Wing with the aim of better supporting our community of international medical students with wellbeing support as they may be particularly vulnerable living so far away from home for the first time.

Inspired by the success of the 2021 BIDA National Conference, we are organising our own student conference later this month and are honoured to have Dr Chaand Nagpaul CBE confirmed as the Chief Guest to inaugurate our first conference.

On a personal note, my learning from serving as Founder-President of the BIDA Student Wing has been immense, particularly after working closely with individuals who come from across the world. I look forward to working with them over the next few months and hope to bring my energy, enthusiasm and ideas to BIDA after I graduate from medical school later this year.

**Sai Pillarisetti** *President, BIDA Student Wing*

## Learn, Advocate & Inspire!




# BIDA A.G.M. 23 October 2021 via Zoom

***Dr BK Sinha*** (President) began by welcoming all attendees and informing everyone that the meeting is being recorded.

***Dr Chandra Kanneganti*** gave his last report as BIDA Chairman. Dr Kanneganti has been the Chairman for the last seven years and wants to make sure BIDA thrives and continues to survive. He pointed out that we have worked closely with all the major organisations and will continue to do so. Dr Kanneganti thanked the EC and officers who worked so hard to raise funds firstly, for the stranded students during the Covid pandemic and then for funding to provide oxygen concentrators and other vital emergency medical equipment to India. Along with this we set up a special telephone consultation service for medical personnel in India who were dealing with the crisis. This year alone we have supported many campaigns including the removal of surcharges. Dr Kanneganti wished to thank ***Mrs. Alison Sherratt*** for all her support and a special thanks to all EC members and officers, particularly ***Mr Amit Sinha*** for all he has been doing for BIDA in his role as BIDA media and communication lead.

***Dr Ashish Dhawan*** thanked all the EC committee for their support over the last four years that he has been in the role of BIDA General Secretary, special thanks to ***Dr BK Sinha***, ***Mr Pranab Sarkar*** & ***Dr Chandra Kanneganti***. This last year has been challenging but as an organisation we have managed to carry on with holding highly successful virtual meetings both nationally and divisionally. The BIDA Journal has carried on being produced as normal and we are also sending regular newsletters out to our members. The sale of ODA House has now gone through, and we are renting a temporary office until the new Executive Committee take over and decide whether to carry on renting or buy a new office.

***Mr PK Sarkar*** (Treasurer) began by thanking the President, Chairman, General Secretary, and the EC committee for their support during his time in the role. He also thanked ***Alison*** and thanked ***Mr Zahur*** for all his advice. Mr Sarkar wished good luck to his successor ***Dr Vinod Gadiyar***.

***Mr Zahur*** presented the accounts and thanked ***Mr Sarkar*** and ***Mrs Sherratt***. The accounts for 2020-2021 will be completed soon and it was unanimously decided the EC will have a meeting to check and approve once done.

***Dr BK Sinha*** read out an amendment to the constitution, which was discussed in the last constitution committee meeting, this is: ***'Notwithstanding the paragraphs 42-47 of by-laws, NEC, if it considers appropriate and subject to adequate technical advice and support available, may decide to hold the whole election process including nominations and voting electronically'***. This would be added as 47A in the constitution (pages 40-42 under the heading Elections). This was unanimously passed.

***Dr BK Sinha*** then moved on to announce this year's fellowship awards. ***Dr (Mrs) Kalpana Upadhyay***, ***Dr Ravi Sharma*** and ***Mr Amit Sinha*** were the recipients of the highest award of BIDA.

***Dr Sinha*** then reported on the Presidents Cup Cricket Tournament. This year Wigan Division were the winners against Blackburn Division. Next year ***Dr Raghu Hegde*** would like to step down as sports co-ordinator and a replacement was suggested. Dr Hegde was thanked for all his hard work in his time in this role.

***Dr BK Sinha*** went through the list of the new EC members (previously published in the last BIDA Journal) and wished them all the best.

This being done, the meeting was finished.

# BIDA Fellowship Awards 2021

## Dr (Mrs) Kalpana Upadhyay

**MS, FRCOG**

Consultant and Clinical Director, Obstetrics & Gynaecology, Wrexham Maelor Hospital, BCUHB since 2008.

Dr Upadhyay is an accredited menopause specialist and member of the medical advisory council of the British Menopause Society, UK. She is an expert witness for the GMC and heavily involved in clinical governance and risk management both at local and national levels.

She has always been actively engaged in delivering educational lectures and small group teaching sessions for primary care colleagues and junior doctors. She has been a member of BIDA since 2008 and a member of BIDA Journal editorial board since 2019 with contributions in the form of articles, quiz and peer review of submitted manuscripts.

She is philanthropic by nature and passionate about improving women's health globally.

## Dr Ravi Sharma

**MD FRCP**

Consultant Physician and Gastroenterologist at Rochdale Group of Hospitals, now part of Northern Care Alliance Trust, since 1983.

Dr Sharma is a highly respected physician with a vast experience and contribution towards education, management and a trained mentor and coach.

He has chaired the Senior Medical Staff Committee, the Medical Executive Committee, the Research and Ethics Committee at Rochdale amongst other management roles.

His contribution towards education includes being on the STC in Gastroenterology and Internal Medicine, Tutor for the Royal College of Physicians London, Post graduate Tutor at Rochdale and an Examiner for MRCP PACES for RCP, UK. He is the current Programme Lead for M.MeD in Gastroenterology and a Senior Lecturer at Edge Hill University.

He has been a BIDA member since 1984 and has played vital roles in both the regional and national committees. He is currently President of Bury & Rochdale Division. He remains a member of the Hospital Consultants and Specialist Committee of BIDA and serves as an assessor for CEA applications/ nominations. His support to the ethnic doctors and trainees and colleagues of all ethnic backgrounds is well appreciated by one and all.

## Mr A Sinha

**MS MCh FRCS G FRCS (Tr& Orth)**

Consultant Orthopaedic Surgeon, Glan Clwyd Hospital, Betsi Cadwaladr University Health Board (1999 - 2018).

He has served the NHS for more than 30 years and been involved in education, research and training as well as the management of orthopaedic services. He has been the Past President of the Welsh Orthopaedic Society and the British Indian Orthopaedic Society UK, Chairman of the Welsh Orthopaedic Sub Advisory Committee and an Ex-Officio Member of the British Orthopaedic Association.

His contribution to training has been through his involvement in the Specialist Training Committee of Orthopaedics in Wales and as the Regional Adviser of Orthopaedics in North Wales. He was an honorary clinical teacher at Cardiff University, Honorary Senior Lecturer Edge Hill University, Royal College Examiner for MRCS and Intercollegiate Fellowship examiner for Trauma and Orthopaedics.

He has been a long-standing member of BIDA and is currently the Media & Communication Lead, Editor of BIDA Journal and Supervisor of the BIDA Student Wing.

# Congratulations! Prestigious Awards for BIDA Members

## Professor Iqbal Singh C.B.E.

Consultant Physician, Royal Blackburn Teaching Hospital East Lancashire Hospitals Trust

Huge Congratulations to Professor Singh, who has been awarded the CBE on the New Years Honours List of 2022. This symbolises recognition of his tremendous contribution to the welfare of the NHS healthcare and medical regulation in the UK.

He is a Medical Leader with great credibility in the wider community, a pioneer in ethnic health and diversity, combined with board level GMC and Healthcare Commission Service. He holds the Chair of the global Centre of Excellence in Safety for Older People (CESOP). He is a member of the National Platinum Awards Committee and medical vice chair of Advisory Committee for Clinical Excellence Awards (ACCEA) North West and is in the Health Honours Committee. He has contributed to BIDA over several years by promoting equality, diversity and human rights within the NHS.

## Dr Saima Niaz recognised for her Voluntary Work during the Covid-19 pandemic

**Dr Saima Niaz**, Consultant Psychiatrist based in London has been recognised for her voluntary work to create mental health awareness and support to the community during the unprecedented times of Covid-19 pandemic. Her work has been appreciated and shared across multiple print and social medias creating awareness related to mental health, corona virus, covid vaccine Myth Busters and to encourage Covid-19 vaccine uptake.


She received acknowledgement for her work from the Mayor of London for reaching out to the diverse communities. The Pakistan High Commission, London prominently shared her work on their website and media handles.

She was felicitated with the 11th Pakistan Achievement Award on 22nd October 2021 in the recognition of her contributions. She also received Award from the Association of Pakistani Physicians and Surgeons UK (APPS).

## Professor Sanjay Arya awarded the 2021 Doctor of the Year Award by BAPIO (UK)

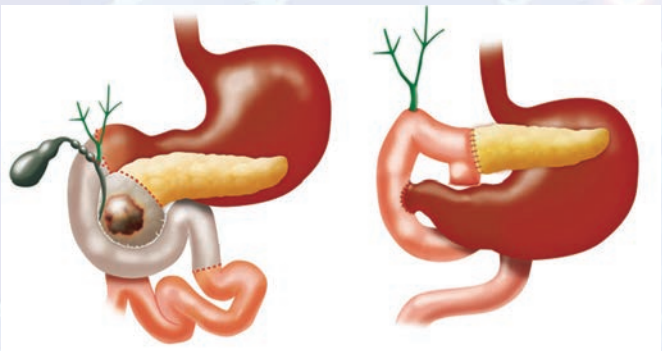
**Prof Sanjay Arya**, Chairman BIDA Hospital Doctors Committee was awarded 2021 Doctor of the Year Award by BAPIO (UK). He is the Medical Director & Consultant Cardiologist at Wrightington, Wigan & Leigh Teaching Hospitals.

Prof Arya was given the award for the leadership he has demonstrated during the Covid pandemic. His work on risk assessment of all frontline staff, availability of enhanced PPE, re-deployment of high-risk clinical staff and elective recovery programme has been widely acclaimed. He was earlier this year awarded Covid Hero for his work during the pandemic. Prof Arya was also commended for his contribution in providing leadership to the BAME healthcare workers in the NHS through his work on embedding fairness and equality and ensuring BAME doctors have fair and equitable access to remediation which is consistent across the NHS.

# BIDA Quiz Answers

***Case 1:*** d) Flutamide - A synthetic antiandrogen. Goserelin often results in flare up of lower urinary tract symptoms once it is started. An antiandrogen should be prescribed along it to prevent the flare up.

***Case 2:*** c) CA19-9. It is one of the tumour markers that usually increase in the head of pancreas cancer. Painless jaundice is often due to head of pancreatic cancer. Whipple's procedure is the gold standard surgical management of head of pancreas malignancies.



***Case 3:*** c) Wegener's granulomatosis; c-ANCA. It is a vasculitis condition that can affect nasal sinus and small to medium vessels in lungs and kidneys resulting in nasal congestion, coughing up blood and seeing blood in urine. C-ANCA is positive in Wegener's granulomatosis.

***Case 4:*** b) Lead poisoning. Constipation, fatigue, headache and loss of appetite are classic symptoms of lead poisoning. He has an occupational exposure to lead as well.

***Case 5:*** a) Ultrasound scan to check for molar pregnancy. It is important to rule out molar pregnancy in patients presenting with hyperemesis gravidarum.

***Picture Quiz:*** B) Hemiarthroplasty. It is the treatment of choice for Garden 3&4 neck of femur fractures in frail elderly patients.

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ALWAYS Rx FULTIUM-D$_3$ BY BRAND NAME

**Abbreviated Prescribing Information.**

**Fultium-D$_3$ 800 IU, 3,200 IU & 20,000 IU Capsules Abbreviated Prescribing Information**. Please refer to the appropriate Summary of Product Characteristics (SmPC) before prescribing Fultium-D$_3$. Use care when prescribing in pregnancy, as high doses of colecalciferol may affect the foetus. **Fultium-D$_3$ capsules:** Each Fultium-D$_3$ 800 IU capsule contains colecalciferol 800 IU equivalent to 20 micrograms vitamin D$_3$. Each Fultium-D$_3$ 3,200 IU capsule contains colecalciferol 3,200 IU equivalent to 80 micrograms vitamin D$_3$. Each Fultium-D$_3$ 20,000 IU capsule contains colecalciferol 20,000 IU equivalent to 500 micrograms vitamin D$_3$. **Indication:** Fultium-D$_3$ 800 & 20,000 IU capsules. Prevention and treatment of vitamin D deficiency. As an adjunct to specific therapy for osteoporosis in patients with vitamin D deficiency or at risk of vitamin D insufficiency. Fultium-D$_3$ 3,200 IU capsules only. Treatment of vitamin D deficiency. **Dosage and administration:** Adults and the elderly. Treatment of Vitamin D deficiency (serum levels <25nmol/l (<10ng/ml)). **Depending on the severity of the disease and the patient's response to treatment:** 1-4 Fultium-D$_3$ 800 IU capsules daily for up to 12 weeks or 1 Fultium-D$_3$ 3,200 IU capsule daily for up to 12 weeks or 2 Fultium-D$_3$ 20,000 IU capsules per week for 7 weeks. Prevention of vitamin D deficiency 1-2 Fultium-D$_3$ 800 IU capsules (800-1600IU) daily or 1 Fultium-D$_3$ 20,000 IU capsule per month. Long term maintenance therapy following deficiency treatment or vitamin D insufficiency (serum levels 25-50nmol/l (10-20 ng/ml) 1-2 Fultium-D$_3$ 800 IU capsules daily. Children over 12 years. **Depending on the severity of the disease and the patient's response to treatment:** 1 Fultium-D$_3$ 800 IU capsule daily (for prevention/ treatment), or 1 Fultium-D$_3$ 3,200 IU capsule daily for up to 12 weeks (treatment), or 1 Fultium-D$_3$ 20,000 IU every 6 weeks (prevention), or 1 Fultium-D$_3$ 20,000 IU every 2 weeks for 6 weeks (treatment). Should only be given under medical supervision. Not recommended for use in children under 12 years. For oral use. Swallow capsules whole with water. **Contraindications:** Hypersensitivity to vitamin D or any of the excipients in the product; hypervitaminosis D; nephrolithiasis; diseases or conditions resulting in hypercalcaemia and/or hypercalciuria; severe renal impairment. **Warnings and Precautions:** Use with caution in patients with impaired renal function or sarcoidosis and monitor the effect on calcium and phosphate levels. In patients with severe renal insufficiency, vitamin D in the form of colecalciferol is not metabolised normally and other forms of vitamin D should be used. In cases of long-term daily doses exceeding 1,000 IU, monitor serum calcium levels. Use caution in patients receiving treatment for cardiovascular disease. Consider vitamin D supplementation from other sources. **Interactions:** Concomitant treatment with phenytoin, barbiturates and glucocorticoids can decrease the effect of vitamin D. Effects of digitalis and other cardiac glycosides may be accentuated. Absorption of vitamin D may be reduced by ion exchange resins and laxatives. **Pregnancy and lactation:** Use only under medical supervision. Studies have shown safe use up to 4,000 IU daily but reproductive toxicity has been seen in animal studies. The 20,000 IU dose should not be used during pregnancy. Vitamin D is excreted in breast milk, when prescribing additional vitamin D to a breast-fed child consider the dose of any additional vitamin D given to the mother. **Undesirable effects:** Allergic reactions are possible. Uncommon adverse reactions include hypercalcaemia and hypercalciuria. **Rare adverse reactions include:** pruritus rash and urticaria. **Overdose:** Refer to SmPC. **Legal Category:** POM. **Pack size:** Fultium-D$_3$ 800 IU capsules x 30 – NHS Price £3.60. Fultium-D$_3$ 800 IU capsules x90 – NHS Price £8.85. Fultium-D$_3$ 3,200 IU capsules x30 – NHS Price £13.32. Fultium-D$_3$ 3,200IU capsules x90 – NHS Price £39.96. Fultium-D$_3$ 20,000 capsules x15 – NHS Price £17.04. Fultium-D$_3$ 20,000 capsules x30 – NHS Price £29.00. **MA Number:** 40861/0002 [Fultium-D$_3$ 800 IU capsules] 40861/0003 [Fultium-D$_3$ 3,200 IU capsules] 40861/0004 [Fultium-D$_3$ 20,000 IU capsules] **MA Holder:** Internis Pharmaceuticals Ltd. Linthwaite Laboratories, Linthwaite, Huddersfield, West Yorkshire HD7 5QH, UK. Full Prescribing Information is available from Internis Pharmaceuticals Ltd. **Date of preparation:** August 2020. **Unique ID no:** FUL-543. **Fultium-D$_3$ Drops Abbreviated Prescribing Information**. Please refer to the Summary of Product Characteristics (SmPC) before prescribing Fultium-D$_3$ Drops. Use care when prescribing in pregnancy, as high doses of colecalciferol may affect the foetus. **Fultium-D$_3$ Drops:** 1 ml of oral solution contains 2740 IU (68.5 mcg per ml) colecalciferol; 3 drops contains 200 IU colecalciferol. **Indications:** Prevention and treatment of vitamin D deficiency in adults and children, and as an adjunct to specific therapy for osteoporosis in patients with vitamin D deficiency or at risk of vitamin D insufficiency. **Dosage and administration:** For oral use. Can be taken directly or mixed with a small amount of food. **Adults:** Treatment of deficiency: 12 - 60 drops (800-4000 IU) daily; During pregnancy and breast-feeding: 6-60 drops (400-4000 IU) daily; Osteoporosis adjunctive therapy: 12 drops (800 IU) daily. **Maintenance or prevention of deficiency:** 12 - 24 drops (800-1600 IU) daily; During pregnancy and breast-feeding: 6-30 drops (400-2000 IU) daily. **Children:** Treatment of deficiency: 0-2 years: 6-15 drops (400-1000 IU) daily; 2-11 years: 6-30 drops (400-2000 IU) daily; 12-18 years: 6-60 drops (400-4000 IU) daily. Maintenance or prevention of deficiency: 0-2 years: 3-15 drops (200-1000 IU) daily; 2-11 years: 6-15 drops (400-1000 IU) daily; 12-18 years: 6-24 drops (400-1600 IU) daily. **Contraindications:** Hypersensitivity to vitamin D or any of the excipients; hypervitaminosis D; nephrolithiasis; diseases or conditions resulting in hypercalcaemia and/or hypercalciuria; severe renal impairment. **Warnings and Precautions:** Use caution in patients with impaired renal function or sarcoidosis. Monitor effect on calcium and phosphate levels in these patients. Consider risk of soft tissue calcification. Use other forms of vitamin D in cases of severe renal insufficiency. Consider the need for calcium supplementation in individual patients. Where calcium supplementation is necessary, close medical supervision is required. Use caution in patients receiving treatment for cardiovascular disease. Make allowances for vitamin D supplementation from other sources. Monitor to prevent hypercalcaemia. **Interactions:** Concomitant phenytoin, barbiturates and glucocorticoids can decrease the effect of vitamin D. Ion exchange resins, laxatives, actinomycin and imidazole may also reduce the effect of vitamin D. Oral calcium and vitamin D potentiates the effect of digitalis and other cardiac glycosides. **Pregnancy and lactation:** Limited clinical data in pregnancy. Animal studies have shown reproductive toxicity. RDI in pregnancy is 400 IU. Pregnant women who are vitamin D deficient may need a higher dose. Pregnant women should follow the advice of their GP, as their requirements may vary depending on disease severity and response to treatment. Vitamin D and metabolites are excreted in breast milk. Overdose in nursing infants has not been observed, however, when prescribing additional vitamin D to a breast-fed child, consider the maternal dose of any additional vitamin D. **Undesirable effects:** Hypercalcaemia and hypercalciuria. Refer to the SmPC for the full list of side effects. **Legal Category:** POM. **Pack size:** Fultium-D$_3$ Drops, 1 x 25 ml – NHS Price £10.70. **MA Number:** 40861/0005. **MA Holder:** Internis Pharmaceuticals Ltd. Linthwaite Laboratories, Linthwaite, Huddersfield, West Yorkshire HD7 5QH, UK. Full Prescribing Information available. **Date of preparation:** July 2020. **Unique ID no:** FUL-542.

Adverse events should be reported. Reporting forms and information can be found at: www.mhra.gov.uk/yellowcard or search for MHRA Yellow Card in the Google Play or Apple App Store. Adverse events should also be reported to 01484 848164.

References. 1. Hypponen E, Power C. Am J Clin Nutr 2007; 85: 860-868. 2. Lanham-New SA, Webb AR, Cashman KD, et al. Vitamin D and SARS-CoV-2 virus/COVID-19 disease. BMJ Nutrition, Prevention and Health. 2020; bmjnph-2020-000089. doi:10.1136/bmjnph-2020-000089. Available at: https://nutrition.bmj.com/content/bmjnph/early/2020/05/13/bmjnph-2020-000089.full.pdf. 3. Data on File Thornton & Ross Ltd, D1. 4. Data on File Thornton & Ross Ltd, D2. 5. Fultium-D3 800IU capsules SmPC: https://www.medicines.org.uk/emc/product/2813/smpc#gref- accessed July 2021. 6. Fultium-D3 3200IU capsules SmPC: https://www.medicines.org.uk/emc/product/5355/smpc#gref- accessed July 2021. 7. Fultium-D3 20KIU capsules SmPC: https://www.medicines.org.uk/emc/product/3659/smpc#gref- accessed July 2021. 8. Fultium-D3 Drops SmPC: https://www.medicines.org.uk/emc/product/6861/smpc#gref- accessed July 2021.

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