Assessment and management of diabetes-related foot infection according to the new International Working Group on the Diabetic Foot guidelines 2023-Multidisciplinary grand rounds

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REVIEW ARTICLE

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Assessment and management of diabetes-related foot infection according to the new International Working Group on the Diabetic Foot guidelines 2023—Multidisciplinary grand rounds

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Abstract

Diabetes-related foot disease is a serious and common complication for people with diabetes mellitus. The gold standard care for a person with diabetes-related foot disease is the involvement of a multidisciplinary foot team engaged in evidence-based care. To date, there are seven International Working Group on the Diabetic Foot (IWGDF) guidelines published to assist healthcare providers in managing diabetes-related foot disease around the world. This review discusses the acute management of diabetes-related foot infection with insights from experts of various specialities (internal medicine, infectious disease, vascular surgery, radiology) with a discussion on the implementation of IWGDF guidelines in real life practice and the challenges that healthcare providers may face.

KEYWORDS
best practice guidelines, diabetes mellitus, diabetes-related foot disease, diabetic foot infection, multi-disciplinary

1 | INTRODUCTION

A 60-year-old man with a history of hypertension presented with a left plantar foot ulcer which had significantly deteriorated over the last 2 days. He first noticed the ulcer 1 month earlier and did not recall trauma as both feet had been numb for the last 3 years. One month earlier, he was diagnosed with type 2 diabetes mellitus during a visit to his general practitioner (GP) when the ulcer developed, his HbA1c was 10.2%, urinalysis showed micro-albuminuria and he had background retinopathy. He was commenced on metformin 1 g twice daily as well as oral flucloxacillin upon suspicion of infection. He was referred to a podiatrist and community nursing for wound dressing, which he failed to attend and he sometimes forgot to take the flucloxacillin. The patient reported that he smoked at least 20 cigarettes a day since the age of 20 and his alcohol intake was <1 standard drink per day.

On examination, the patient looked well. He was mildly tachycardic (108 bpm) and his blood pressure was 150/95 mmHg. He had a temperature of 38°C. A large ulcer (4 × 4 cm) with a sloughy wound base on the left plantar midfoot was noted. The foot appeared to be mildly erythematous with associated oedema. There was more marked erythema, approximately 1.5 cm surrounding the ulcer, despite 2 weeks of previous antibiotic therapy (Figure 1). The wound was deep but did not probe to bone (PTB) (depth of 5 mm). Monofilament testing confirmed loss of protective sensation to both feet. His left foot also appeared to be 'flattened' compared with his right foot. A comment was made by an experienced nurse that the ulcer had a green discharge. Dorsalis pedis pulse was present but posterior tibial artery pulse was not palpable. Ankle-brachial index was >1.4. No abnormalities were noted on the contra-lateral leg.

His blood test showed a high white cell count (26.1 × 10^9/L), elevated serum C-reactive protein (CRP) (260 mg/L) and serum glucose level of 17.2 mmol/L; kidney function was normal. A weight-bearing plain x-rafcalfy of the left foot was performed, which did not show any evidence of osteomyelitis but a flattened midfoot was noted (Figure 2). The patient was admitted to the hospital and immediately received intravenous fluids and an insulin infusion and was started on intravenous amoxicillin-clavulanic acid after blood cultures and a wound swab of the base of the wound had been obtained.

Despite 24 h of intravenous antibiotics and bedrest, the wound did not improve, and the patient had a persistent temperature around 38°C. Additionally, the wound also had increased purulent discharge. He underwent surgical debridement of the wound to remove infected, non-viable tissue, which extended close to the cuboid bone but did not involve it clinically. Debrided tissue was sent for microscopy, culture, and sensitivity. Toe pressure was performed and was 62 mmHg on the left foot with biphasic waveform noted; the Toe:Brachial Index (TBI) was 0.68. Arterial duplex ultrasound of the left leg was performed because of the absent posterial tibial (PT) palpable pulse and the marginal abnormal TBI (preferably it should be >0.7). This did not identify any significant stenosis of major arteries from the aorta to the foot, but calcification of tibial vessels was noted.

By day 4 of admission, the patient improved clinically with normalisation of inflammatory markers and clinical appearance of the foot wound. The intraoperative tissue culture showed the growth of *Staphylococcus aureus*. The staphylococcal strain was sensitive to flucloxacillin, cephalexin and clindamycin. The patient's antibiotic was subsequently changed to oral flucloxacillin 1 g 4 times a day for a duration of 2 weeks. The patient was also assessed by a podiatrist

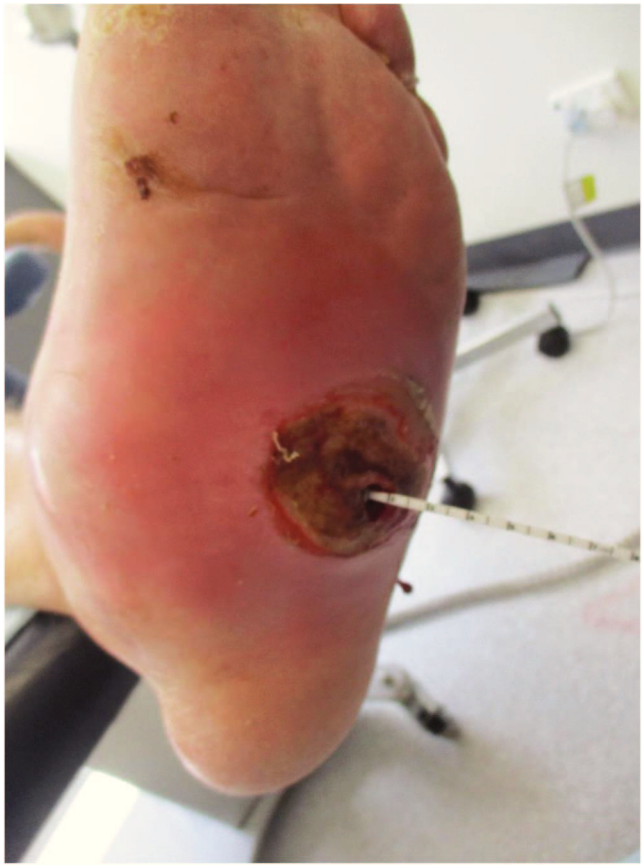

FIGURE 1 Clinical photo demonstrating a large and deep wound over the left plantar midfoot with surrounding erythema.

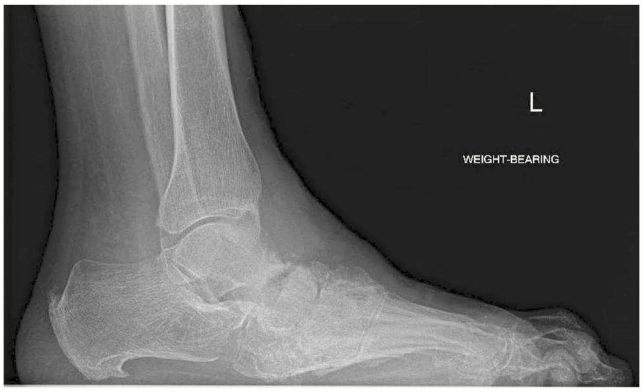

FIGURE 2 A weight-bearing plain radiography did not review any evidence of osteomyelitis of the cuboid.

and was prescribed a DH Offloading Walker (removable knee-high offloading device, Össur®) (Figure 3). He was also assessed by an endocrinologist and was prescribed long-acting subcutaneous insulin in addition to correctional short acting subcutaneous insulin. Both the patient and his family received diabetes education by a diabetes nurse educator on the management of diabetes at home. The patient continued to improve clinically and was discharged home on day 6 with a plan for review in multi-disciplinary foot clinic.

At out-patient review prior to completion of his antibiotic therapy, the ulcer had improved significantly; however, the left foot remained warmer than the right with some erythema and oedema. A repeat X-ray was unchanged. Magnetic resonance imaging (MRI) was performed to clarify whether osteomyelitis of the cuboid (bone underlying the ulcer) or active Charcot neuro-osteoarthropathy was present (Figure 4). MRI demonstrated diffuse bone marrow oedema present within the navicular, all cuneiforms, cuboid and bases of all metatarsals suggestive of Charcot neuro-osteoarthropathy without any fracture. The patient was transitioned into total contact cast (TCC) in consultation with the orthopaedic department as a diagnosis of active Charcot neuro-osteoarthropathy was made.

Six months following his presentation to the emergency department, the ulcer had completely healed without further antimicrobial therapy. Charcot neuro-osteoarthropathy was in remission without further development of foot deformities and the TCC treatment had been stopped. Given that he has a high-risk foot, he continues to receive regular podiatry care and has customised footwear with an customised insole to ensure ongoing offloading.

FIGURE 3 Removable knee high offloading device.

2 | ACTIVATE CODE DFI

Diabetes-related foot disease is associated with significant morbidity, reduced quality of life but also reduced survival compared with many patients diagnosed with cancer.[1,2] Delayed diagnosis and subsequent referral to multi-disciplinary foot services remains a concern.[3] In our case, this patient's first contact with the specialist team was a month following his diagnosis of diabetes-related foot infection (DFI) and likely resulted in significant clinical deterioration. One may wonder if this deterioration could have been prevented with timely implementation of multidisciplinary care. A DFI with rapidly progressive deep infection can escalate to a limb- and even life-threatening condition within a few days or even hours. Immediate recognition and urgent treatment with debridement (surgically if necessary), systemic antibiotics and clinical stabilisation of impending sepsis are essential.[4] 'Code STEMI' is in several countries the phrase used to activate an emergency clinical pathway which has significantly improved outcomes for patients with suspected myocardial infarction. The integrated pathway facilitates immediate management and prompt referral to the cardiology team if there is a reasonable suspicion of myocardial infarction. Terminology such as activate 'Code DFI' or 'Diabetic foot attack', and its clinical pathway, should be developed for patients with DFI to emphasise the urgency in the management of diabetes-related foot complications to improve clinical outcomes.[5]

3 | IS THERE A FOOLPROOF RECIPE TO MANAGEMENT OF ACUTE DFI?

When it comes to the management of emergency presentation of DFI, no two feet are alike. The presentation can vary from being clinically well to critically unwell, mild DFI or systemic sepsis.

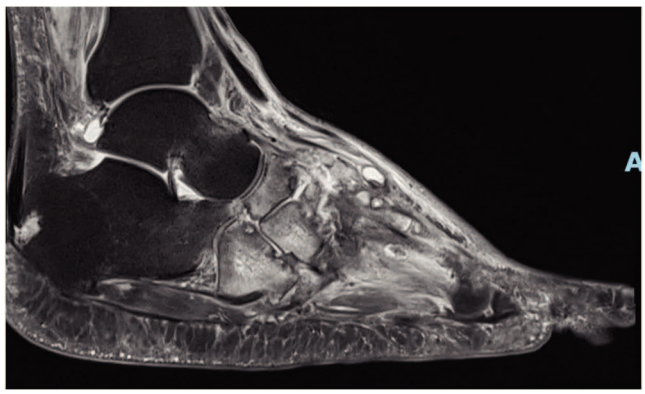

FIGURE 4 Magnetic resonance imaging demonstrating diffuse bone marrow oedema present within the navicular, all cuneiforms, cuboid and bases of all metatarsals suggestive of Charcot foot.

Regardless of the presentation, there is a consistent strategy with a holistic approach that one can utilise to allow effective assessment and treatment.

The management of DFIs should start with full evaluation of the person to assess for any systemic signs of sepsis, dehydration, or exacerbation of underlying comorbidities (such as heart failure or chronic kidney disease) as a result of acute infection. Immediate treatment such as fluid resuscitation and intravenous antibiotics should be administered in a timely manner to prevent further deterioration. It is also important to immediately assess blood glucose level as severe hyperglycaemia, with or without diabetic ketoacidosis or hyperosmolar hyperglycaemic state, must be addressed promptly, as in our patient.

All DFIs should be classified using a validated classification system such as the International Working Group on the Diabetic Foot (IWGDF)/IDSA infection classification and the wound ischaemia foot infection (WIfI) classification system to facilitate communication and help guide ongoing treatment.[1] Based on the clinical presentation of an infected deep ulcer, our patient had a moderate foot infection according to the IWGDF/IDSA infection classification and his WIfI clinical stage was 3 (Table 1).[4,6] It is important to bear in mind that the assessment of DFI is dynamic and evolving as more information becomes available.

The inflammatory signs at presentation and the appearance of the flat foot at presentation also raised concern for the diagnosis of Charcot neuro-osteoarthropathy, but no bony destruction was seen on the X-ray. Initial radiological examination for DFI requires three standard views on plain x-ray, namely dorsoplantar, lateral and medial oblique views, as was done in our patient. The location of the ulcer should also be indicated with a radio-opaque marker to allow accurate assessment of the structures adjacent to the ulcer.

4 | SO, IS IT INFECTED?

DFIs are one of the most frequent diabetes-related complications requiring hospitalisation and the most common precipitating events leading to lower extremity amputation.[7,8] Infections can be acute or chronic, localised or systemic, involving soft tissue, bone, or both. Signs and symptoms of acute inflammation may be masked by the presence of peripheral neuropathy, Peripheral artery disease (PAD), or immune dysfunction.[4] Our patient displayed clinical signs suggestive of evolving infection, including cellulitis, fever, mildly elevated heart rate and significant hyperglycaemia. As an adjunct to the clinical diagnosis, the patient also had leucocytosis and raised CRP levels to further support the diagnosis of an infection. Additionally, on clinical examination, the ulcer was sloughy and extended close to joints in the midfoot, but the PTB test was negative. If the PTB test was positive, it would strongly support a presumptive diagnosis of osteomyelitis. A normal x-ray does not rule out osteomyelitis, but this can be repeated in 2–3 weeks at which time changes may be noted. Additionally, plain x-ray also plays an important role in the acute setting to detect foreign body not suspected due to the loss of protective sensation.[4] While advanced imaging for diagnosing diabetes-related foot osteomyelitis (DFO) is not needed in many patients, MRI is a sensitive tool that gives good imaging of soft tissues, bones, and joints, aiding in detecting pre-operatively any purulent collections or the extent of bone involvement, as well as clarifying whether active Charcot may be present.

TABLE 1 WIfi classification of foot wound, for details on WIfI please refer to Fitridge et al. (2023).[6]

Wound	2
Ischaemia	0
Foot infection	2
WIfI clinical stage	3 Moderate risk of amputation at 1 year

Abbreviation: WIfI, wound ischaemia foot infection.

5 | IT IS INFECTED

Once a diagnosis of infection has been made, it is time critical to commence appropriate antimicrobial therapy based on the likely isolated causative pathogens(s) and their antibiotic susceptibilities while taking into consideration the clinical severity of the infection. The choice of oral flucloxacillin as the initial treatment in our patient was appropriate as it was the patient's first presentation of ulceration, and the patient was not clinically unwell. Additionally, oral flucloxacillin is low cost and not commonly associated with severe adverse events.[9] However, some clinicians might opt for prescribing broader spectrum antibiotics such as amoxicillin-clavulanic acid because of concerns that oral flucloxacillin has presumed low oral absorption and consequently low bone penetration. In case of mild DFIs, the most likely causative organisms are gram-positive pathogens (*S. aureus* and beta-haemolytic streptococci). Broader spectrum antibiotics with activity against gram-negative bacteria and obligate anaerobes should be considered in patients with moderate or severe infections.[4] The decision to change antibiotics to empirical intravenous amoxicillin-clavulanic acid for beta-lactamase coverage was appropriate as the patient had failed initial management and progressed to moderate infection, with recent antibiotic use which warrants coverage for gram-negative rods. There was a comment made on the initial assessment that the discharge has a 'green tinge'. Historically, clinicians have made decisions to prescribe antibiotics with *Pseudomonas* spp. coverage based on visual and olfractory cues. However, a recent prospective evaluation has found that even in experienced clinicians, the predictive ability is only moderate with better specificity than sensitivity.[10] Nevertheless, *Pseudomonas aeruginosa* infection in community-acquired DFI is rare, and the IWGDF guidelines recommend a pre-emptive or empirical anti-Pseudomonas medication only in life-threating infections or special epidemiological settings with a very high prevalence of Pseudomonal infections.[4]

Obtaining a specimen for culture is value adding as it provides useful information on the causative pathogen(s) and their antibiotics susceptibility. A common pitfall in the management of DFIs is the reliance on superficial wound swab to guide treatment as wound swab is easier to perform compared to soft tissue culture or curettage of the wound base.[4] There is a higher risk of contamination with normal skin flora with superficial wound swab, even from the wound base, and consequently if treatment was based on the results from the superficial wound swab there is a risk of therapy failure or antibiotic resistance due to inappropriate choice of antibiotics. If there is a suspicion of osteomyelitis, bone sampling (either surgically or percutaneously) should be considered to ensure appropriate antimicrobial therapy directed at the causative pathogen.[4]

6 | 'DON'T LET THE SUN SET ON PUS'

This is a common saying amongst surgeons when it comes to the management of infection—when there is pus, complete drainage of purulent and likely infected material is important for sepsis control. As the patient developed signs of persistent infection with increased purulent discharge, a decision was made for surgical sepsis control with the goal of removing any infected tissue and allowing pus drainage. Fortunately, the intraoperative assessment suggested unlikely bony involvement and debridement was carried out with a tissue sample sent to laboratory for culture and sensitivity. The culture returned positive for *S. aureus*. A total duration of 2 weeks of antibiotics is usually prescribed for severe soft tissue DFI. Due to low clinical suspicion of osteomyelitis, further imaging such as MRI was not performed at that time.

7 | SHOULD WE CALL THE VASCULAR SURGEON?

Clinicians should evaluate wound perfusion and the potential need for revascularisation as soon as practical in all patients with diabetes-related foot ulcer (DFU). Peripheral artery disease (PAD) is present in about 50% of DFU and is associated with delayed wound healing and amputation.[6] The presence of one or more palpable foot pulses does not reliably rule out PAD. The history of smoking also increased the likelihood of PAD. Ankle-brachial index was likely falsely elevated due to medial artery calcification (MAC). MAC is associated with peripheral neuropathy and an elevated risk of cardiovascular mortality and morbidity.[11] As the foot arteries are less likely to be affected by MAC, the measurement of toe pressures (TP) with calculation of a TBI is often preferred; a TBI >0.7 makes PAD less likely.[6]

Assessing peripheral perfusion is also important to predict the likelihood of healing. A TP <30 mmHg or an ankle pressure <50 mmHg are associated with greater likelihood of impaired healing and require consideration of revascularisation. The WIfI classification system grades the severity of the wound, the presence and severity of ischaemia, and the presence and severity of infection to predict the likelihood of major amputation. It can also be used to assess whether there is likely to be a benefit of revascularisation.[12] Anatomical assessment of the arteries from the aorta to the affected foot should only be undertaken if ischaemia is found to be present on bedside testing and deemed severe enough to warrant consideration of revascularisation. In this case, the initial clinical assessment of perfusion was according to the vascular surgeon inconclusive with a TBI that was somewhat below the threshold value of 0.7 (and the absent PT pulse) but with a biphasic waveform that rendered significant PAD less likely. This led to further vascular imaging to assess if there was any evidence of flow limiting arterial stenosis that warranted revascularisation. Overall, the final assessment was that the perfusion was adequate, and that revascularisation was not indicated. Additional reassessment of foot perfusion would have been indicated if no significant improvement in the wound area was found after 4 weeks of appropriate therapy.[6]

8 | TAKE THE PRESSURE OFF

The patient's ulcer was probably caused by repetitive biomechanical stress and the foot deformity (pes planus) had probably resulted in increased plantar pressures in his midfoot during walking and standing. Due to the loss of protective sensation, he did not perceive the repetitive trauma and in order to enable healing, his ulcer should be protected against this elevated weight-bearing pressure. A non-removable knee-high offloading device such as a TCC or Walker is strongly recommended for neuropathic plantar ulcers.[13] However, his infected wound required frequent dressing changes and wound monitoring; therefore, a TCC was initially not appropriate and a removable below knee walker was used. Once infection is under control, this treatment can be changed to a non-removable device. However, many patients prefer a device that they can take off, but due to poor compliance removable devices do not promote healing as well as non-removable TCCs or Walkers.[13] Of note, and importantly, offloading should go in parallel to professional and regular wound care, especially in the presence of DFI.

9 | CARPE DIEM: SEIZE THE OPPORTUNITY TO MANAGE RISK

Our patient had a delayed diagnosis of T2DM, which came to light due to a complication of the disease, and apart from the neuropathy he also had signs of eye and kidney damage. He clearly is at a very high risk of future cardiovascular complications and progressive loss of kidney function; his presentation to the hospital should be considered as a golden opportunity to address any modifiable risk factor that could contribute to further complications. The Steno-2 study showed that in patients with type 2 diabetes mellitus and early kidney damage, such as in our patient, a combination of lifestyle

modification, glycaemic control, blood pressure control and further cardiovascular risk management can dramatically reduce cardiovascular complications.[14]

Glycaemic control in patients with advanced DM must be managed carefully with strict monitoring. It is not just a matter of reducing blood glucose levels quickly and aggressively as that can have detrimental effects.[15] Too rapid reduction in HbA1c in someone who has had very high glycaemia for a long time can cause a paradoxical flare-up of microvascular complications, such as retinopathy, nephropathy or neuropathy. Treatment-induced neuropathy occurs especially if HbA1c drops >3% in a short period of time and blood glucose levels should be gradually lowered over several weeks.[16] In addition, too aggressive blood glucose reduction to near-normal values can be harmful in patients with advanced complications with an increase in cardiovascular events and mortality.[17] These observations were made with the use of older drugs such as sulfonylureas, glitazones and insulin and it is uncertain if aggressive reduction of HbA1c using more recent drugs such as SGLT-2 inhibitors or GLP1-receptor agonists would have the same effect. Regardless of their effect on glycaemia, SGLT-2 inhibitors should be considered in our patient once the ulcer has healed given their cardiovascular and renal benefits.[6]

Best medical therapy, comprising evidence-based strategies for smoking cessation and other active lifestyle changes, cholesterol reduction, and treatment of hypertension must be recommended to this person as it is known to reduce risks of major adverse cardiovascular events and major adverse limb events.

10 | SO, IT IS ALSO CHARCOT

A diagnosis of active Charcot neuro-osteoarthropathy should have been considered at the hospital presentation as he had a red, hot and swollen foot. But, the patient also had signs of active infection and the plain x-ray did not demonstrate any evidence of fracture and therefore the diagnosis of active Charcot neuro-osteoarthropathy was not considered. However, with hindsight, this reasoning was not correct as the presence of ulceration and active infection do not preclude underlying active Charcot. The IWGDF 2023 guidelines recommended that this diagnosis should always be considered in a person with diabetes with neuropathy and a hot swollen foot with intact skin, but of course this condition can also occur in an infected/ulcerated foot, as in our patient.[18] Fortunately, the diagnosis of stage 0 Charcot neuro-osteoarthropathy was considered after resolution of the DFI and the diagnosis was confirmed based on the outpatient MRI that showed typical abnormalities, including bone marrow oedema without fractures.[19] Although this imaging technique is very useful in the diagnosis of active Charcot, it can be difficult to differentiate this disease from DFO as bone marrow oedema can also be a feature of osteomyelitis.[20] Expert radiologist opinion is therefore recommended to assess the MRI results. When in doubt, further investigations such as dual energy CT, some scintigrahy techniques and/or bone biopsy are warranted until the diagnosis is made or excluded.[21,22] Treatment was in our patient modified from a removable knee-high offloading device to the gold standard TCC to safely immobilise and offload the foot to halt any further progression of joint and bony destruction.

With the benefit of hindsight, it appeared that the patient had developed active Charcot-neuro-osteoarthropathy likely prior to his presentation to his GP with a plantar foot ulceration. The changes in his foot shape (flat foot) due to ligamentous subluxation in his midfoot would have led to increased plantar pressure and the subsequent skin breakdown and ulceration. This ulcer subsequently became infected, which finally prompted his presentation to the emergency department. As the infection was more prominent and deemed to be a more acute issue, the diagnosis of Charcot foot was not considered until the infection had settled. Despite the diagnostic delay, the patient was initially appropriately prescribed a knee-high removable walking device, which was likely sufficient to prevent further deformities.

11 | CONCLUSION

Much is currently known about the management of patients with diabetes-related foot disease, including diagnosis, surgical and medical treatment. The IWGDF guidelines are useful tools in the management of persons affected by diabetes-related foot disease; however, we must also acknowledge that there will be challenges with the day-to-day application of the recommendations due to factors relating to the person, resources, and the institution that will require clinicians to troubleshoot or even come up with creative solutions.

AUTHOR CONTRIBUTIONS

Kay Hon, Frank Nobels, Éric Senneville, Ilker Uckay, Mario Maas and Robert Fitridge have made substantial contributions to the conception and design of the review article; been involved indrafting the manuscript and revised it critically for important intellectual content. All authors have given final approval for the version to be published and have agreed to be accountable for all aspects of the work.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analysed in this study.

ETHICS STATEMENT

Not applicable.

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