Case report

Diagnostic dilemma of sacral abscess presented with seizure and altered conscious level in a patient with spinal cord injury

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Context: Infection and septicemia may clinically presented with seizure and altered conscious level. In spinal cord injury (SCI) population, they are at risk of having pressure ulcer which can be complicated further with infection and septicemia.

Findings: A 40-year-old man with complete T4 SCI and multiple clean and non-healing pressure ulcers at sacral and bilateral ischial tuberosity regions was initially admitted for negative pressure wound therapy (NPWT) dressing. He had an episode of seizure and subsequently had fluctuating altered conscious level before the diagnosis of deep-seated sacral abscess was made and managed. Prior investigations to rule out common possible sources of infections and management did not resolve the fluctuating event of altered consciousness.

Clinical relevance: We presented an unusual case presentation of septicemia in a patient with SCI with underlying chronic non-healing pressure ulcer. He presented with seizure and fluctuating altered conscious level. Even though a chronic non-healing ulcer appeared clinically clean, a high index of suspicion for deep seated abscess is warranted as one of the possible sources of infection, especially when treatment for other common sources of infections fails to result in clinical improvement.

Keywords: Sacral abscess, Septicemia, Pressure ulcer, Spinal cord injury

Introduction

Infection and septicemia may clinically be presented with seizures and altered conscious level. The spinal cord injury (SCI) population is prone to complications from pressure ulcers. Chronic non-healing pressure ulcers may be complicated by formation of sinus tracts, deep-seated abscess collections, and osteomyelitis, which can lead to septicemia.^{1,2} Therefore, physicians should have a high index of suspicion of these complications in the SCI population who present with chronic non-healing pressure ulcers and septicemia, especially when other common sources of infection have been treated and ruled out. In the event of suspicion of deep-seated abscesses in the SCI population, urgent radiological investigation with computed tomography (CT) or magnetic resonance imaging (MRI) must be performed to confirm the diagnosis and delineate the extent of complications for appropriate management. Most importantly, timely management is crucial to prevent sequelae of septicemia and mortality.

Case report

A 40-year-old man was admitted to the rehabilitation ward with a history of complete SCI at the T4 level and severe traumatic brain injury (TBI) since 2007. He had no history of other medical co-morbidities. He has multiple pressure ulcers over the sacrum, bilateral ischial tuberosities, and left posterior subtrochanteric region. The patient had undergone multiple wound debridements for pressure ulcers over the past 6 years. He had also undergone a bilateral myocutaneous flap reconstruction for his bilateral ischial ulcers in 2009, which was complicated by wound infection and resulted in wound breakdown and a chronic non-healing wound.

The patient was electively admitted to the rehabilitation ward for further management of his chronic non-healing pressure ulcers with negative pressure wound therapy (NPWT). The base of the pressure ulcers over the ischial tuberosities and left posterior subtrochanteric region appeared healthy and healing; therefore, treatment was continued with normal saline wet to dry dressings. The sacral pressure ulcer was 2 cm by 2 cm in size, clean, and healthy looking, but had a 10 cm deep pocket beneath the ulcer edge (Fig. 1). A sinogram

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Figure 1 Site of the pressure ulcers

before admission showed absence of sinus tract extension to other organs and open wounds. NPWT was started on day 2 of admission.

Neurological examination according to the International Standards for Neurological Classification of Spinal Cord Injury showed that the American Spinal Injury Association Impairment Scale (AIS) of the patient was T4 AIS A. He had generalized spasticity of both upper limbs at biceps, triceps, and wrist flexors with Modified Ashworth Scale (MAS) of MAS 1 and 1+. Bilateral hamstring flexion contractures with a popliteal angle of 90 degrees were also noted. These conditions led to the patient needing substantial assistance for pressure relief, mobility, and personal activities of daily living (ADL). His blood pressure upon admission was 100/56 mmHg, and his pulse rate was 70 beats per minute. He was alert, afebrile during admission, and examination of the other systems was unremarkable with baseline systolic blood pressure of 80–90 mmHg and diastolic blood pressure of 50–60 mmHg.

During admission, the patient was engaged with active rehabilitation therapy to achieve his maximum potential for his ADL and mobility consistent with his impairment level. Unfortunately, on the third day of admission, he had the sudden onset of a generalized tonic-clonic seizure during his physiotherapy session. He had just completed his tilt table therapy session and was lying flat on a plinth when the seizure occurred. The seizure lasted for about 1 minute and ceased spontaneously. The seizure recurred three times within 15 minutes, with each lasted for less than 1 minute and ceased spontaneously.

An immediate clinical evaluation was conducted to rule out possible neurological causes. Table 1 shows

the list of the first few possible neurological causes that needed to be ruled out urgently. The seizures were unlikely to have been caused by autonomic dysreflexia (AD) or orthostatic hypotension because his blood pressure remained stable throughout the tilt table session. His blood pressure ranged from 84 mmHg to 96 mmHg systolic and 46 mmHg to 60 mmHg diastolic, which was similar to his baseline blood pressure. He was tachycardic, with a pulse rate of 110–120 beats per minute. He did not have other signs of AD, such as headache, sweating, flushing, or bradycardia. No drop in his blood pressure was documented during his 45 minutes of tilt table therapy session to suggest orthostatic hypotension. Electrocardiogram (ECG) results showed sinus rhythm without ischemic changes. This finding ruled out a cardiac event as the possible cause of hemodynamic instability that can lead to seizures. His blood electrolytes were normal, and his random blood sugar was 9.1 mmol/L; hence, the seizure was unlikely caused by hypoglycemia or hyponatremia. An urgent CT brain showed an old left basal ganglia infarct, which ruled out intracranial bleeding and acute infarct. The electroencephalography (EEG) conducted a few days later showed only generalized slow wave, without epileptiform discharges. Breakthrough seizure with underlying epilepsy was also one of the differentials because the patient has a history of severe TBI. Nevertheless, no previous history of seizure was recorded, and he was not on any antiepileptic medication. The patient was on regular baclofen and clonazepam to manage his generalized upper and lower limb spasticity for the past few years, which was continued during this hospitalization. No sudden discontinuation of baclofen, which can also induce seizure, was noted.

Blood investigations were normal, except for raised infective markers. White blood cells (WBC), ESR, and CRP were $25.9 \times 10^9/L$, 85, and 17.4, respectively. The patient was not tachypneic and had clear lungs. Urine microscopy suggested urinary tract infection (UTI). The patient was then treated as urosepsis (based on the blood investigations and urinalysis), which may be complicated by seizures. We were unable to rule out meningitis or meningo-encephalitis

Table 1 Differential diagnosis of neurological causes

Differential diagnosis of neurological causes:

1. Autonomic dysreflexia
2. Hypoxic encephalopathy secondary to orthostatic hypotension, cardiac event or hypoglycaemia
3. Cerebrovascular accident
4. Epilepsy

Table 2 Differential diagnosis of infective causes**Differential diagnosis of infective causes:**

1. Meningitis or meningo-encephalitis
2. Pneumoniae
3. Urinary tract infection

as the patient refused to provide consent for lumbar puncture. Table 2 lists the differential diagnosis of infective causes. His pressure ulcers remained clean, without evidence of acute infection.

The patient remained seizure-free, but his consciousness levels were fluctuating. He had confused speech and occasional echolalia. His Glasgow Coma Scale (GCS) fluctuated between 10 and 12. His urine culture and sensitivity showed mixed organism growth. One of his blood cultures grew methicillin-resistant *Staphylococcus aureus*, but it was presumed to be a contaminant as the other blood culture did not grow any organism. The patient was intravenously given ceftriaxone, which was continued for 1 week to cover for urosepsis. He was afebrile throughout. He progressively regained full consciousness, and his GCS was full (scale of 15) after 5 days from the initial onset of seizure, which was on day 10 of admission.

On day 14 of admission, the patient had an episode of low-grade fever. His temperature was 37.6°C. Episodes of altered conscious level and seizure were not noted. His WBC was $10.4 \times 10^9/\text{L}$. His blood culture did not grow any organisms, and his urine culture showed mixed organism growth. The patient was treated as having possible nosocomial infection; hence, he was started on intravenous tazocin to prevent another event of seizure and altered conscious level.

The patient remained well with antibiotic on board (intravenous tazocin) until after 6 days (day 20 of admission), when he had another episode of altered consciousness. His GCS was 12, and he had confused speech and echolalia. He was afebrile, and seizure was not noted. Repeated WBC, ESR, and CRP were $8.3 \times 10^9/\text{L}$, 83, and 6.3, respectively. Intravenous tazocin was still continued. Organism growth was not observed in his blood culture, but his urine culture had mixed organism growth. A repeat CT brain was conducted, which showed similar findings as the first brain CT. An old left basal ganglia infarct was noted, but no intracranial bleeding. He regained full GCS, and his speech became coherent after 2 days. Intravenous tazocin was continued.

On day 28 of admission, the patient had the first episode of multiple spikes of high-grade fever. His temperature was 38°C, and his GCS dropped to 12. No

episode of seizure was noted, and the apparent cause of fever and altered conscious level could not be established again. On the same day, copious pus discharging from an opening in a healed surgical scar above the current sacral ulcer was observed. A provisional diagnosis of a deep-seated abscess was given, and an urgent pelvic CT was conducted (Fig. 2). A 2.3 cm (AP) $\times$ 9.7 cm (W) $\times$ 10.6 cm (H) rim-enhancing fluid collection with air pockets within was observed at the sacral region, which confirmed the diagnosis. His tazocin treatment was changed to intravenous imipenem on the same day.

The patient was referred to the orthopedic team for urgent wound debridement and incision and drainage (I&D) of the sacral abscess. Figure 3 shows the surgical wound at the sacral area post I&D. Microbacterial culture from the pus grew *Proteus mirabilis* and ESBL-producing *Klebsiella pneumoniae*. Both organisms were sensitive to imipenem. He completed intravenous imipenem for 14 days. The patient recovered well after surgery and completion of antibiotic. No more episodes of fluctuating altered conscious level, seizure, or fever were noted. Table 3 shows the summary of clinical events and investigations conducted throughout his hospitalization.

The patient was referred to the plastic surgery team for further management of his sacral ulcer. The team

Figure 2 CT scan of the pelvis with rim-enhancing fluid collection at sacral region.

Figure 3 Post incisional drainage of abscess at sacral region.

proceeded with bursectomy of the infected ischial bones (chronic osteomyelitis) and primary closure of the sacral ulcer with a myocutaneous flap at four months after the detection of sacral abscess.

Discussion

The SCI population is at high risk for developing pressure ulcers because of their sensory impairment and immobility. Those with chronic non-healing ulcers may also have underlying deep-seated abscesses or osteomyelitis, which may remain undetected until the patients present with sepsis or overt wound breakdown. Patients with SCI with chronic ulcers will most likely have intermittent infection of the ulcer; bacteremia is a common complication of non-healing chronic ulcer.¹ The infection may track deep into the subcutaneous tissues causing pockets of infection or abscesses. The abscesses can spread hematogenously over time, causing systemic infection or sepsis including meningitis.²

Simpson *et al.* reported a case of bacterial meningitis associated with complex ischiorectal abscess in a 37-year-old man with a history of groin abscess 4 years prior.³ They reported that the bacterial meningitis was

Table 3 Summary of clinical events and investigations

Timeline (Day of admission)	Clinical Event	Investigations
Day 3	First episode of generalized clonic-tonic seizure Lasted for 1 minute, aborted spontaneously Recurred 3 times in 15 minutes No fever documented Treated as urosepsis with IV ceftriaxone for 1 week Investigation of cerebrospinal fluid (CSF) with lumbar puncture was not consented	Random blood sugar – 9.1 mmol/L WBC – $25.9 \times 10^9/L$ ESR – 85 CRP – 17.4 Electrolyte normal Urine microscopy – Leukocyte positive – Nitrite positive – Protein positive CT brain – old left basal ganglia infarct
Day 5	GCS fluctuated between 10 to 12/15 No new episode of seizure	EEG – generalized slow wave and no epileptiform discharges
Day 10	Regained full consciousness with GCS of 15 Completed 1 week of IV ceftriaxone, afebrile throughout	Urine C&S – mixed organism growth Blood C&S – no significant bacterial growth
Day 14	Had episode of low grade temperature – 37.6° Celsius GCS full No seizures Started on IV Tazocin treated as nosocomial infection	WBC – $10.4 \times 10^9/L$ Blood C&S – no growth Urine C&S – mixed organism growth
Day 20	Another episode of altered conscious level with GCS of 12/15 He had confused speech and echolalia He was afebrile and had no seizure He regained full conscious level after 2 days	WBC – $8.3 \times 10^9/L$ ESR – 83 CRP – 6.3 Electrolytes were normal Blood C&S – no growth Urine C&S – mixed organism growth CT brain – old left basal ganglia infarct, no ICB
Day 28	Multiple spiking of fever – 38° Celsius GCS of 12/15 No seizures Overt pus discharges from a deep seated abscess Started on IV imipenem Referred to orthopedic for wound debridement and incisional and drainage of pus	CT pelvis – rim enhancing collection measuring 2.3 (AP) $\times$ 9.7 (W) $\times$ 10.6 (H)cm with air pockets within was seen at the sacral region Pus C&S – <i>Proteus mirabilis</i> and ESBL-producing <i>Klebsiella pneumonia</i> , sensitive to imipenem

a result of direct extension of a spontaneously occurring ischiorectal abscess into the intradural space. This complication may also be the case for our patient. Given that the location of the sacral abscess was close to the sacrum, the abscess could have eroded into the intradural space, lead to bacterial meningo-encephalitis, and may clinically presented with seizure and altered level of consciousness. Unfortunately, this hypothesis could not be established in our case because lumbar puncture was not performed. Other causes of septicemia that were considered in our case were pneumonia and UTI. Given that the urine microscopy of the patient suggested UTI, his seizure and subsequent altered conscious level may possibly be due to that. Hence, the treatment was initially aimed at the diagnosis of urosepsis, which can be complicated with seizure. Initially, the chronic non-healing pressure ulcer of the patient was not deemed as a source of infection because his ulcer was clean and no pus or any discharges from the deep 10 cm pocket beneath the ulcer edge were observed. The patient also did not have high spiking temperature during the first 27 days of hospitalization. The patient had high-grade fever and noted copious pus discharges from an opening in a healed surgical scar above the current sacral ulcer only on day 28 of admission. The CT of the pelvis confirmed the diagnosis of deep-seated sacral abscess. Notably, in the event that no other common sources of infection are discovered, further radiological investigation with MRI or CT is warranted to explore possible complications (e.g. deep-seated abscess, osteomyelitis, and fluid collections) in a clean-appearing chronic non-healing pressure ulcer.⁴

Preventing wound breakdown and chronic pressure ulcers that may lead to severe infection is important. Poor motivation and lack of knowledge of medical complications related to pressure ulcers make management more difficult to ensure successful wound healing and prevention of wound breakdown after a surgical closure with myocutaneous flap. Chronic non-healing

ulcers in SCI may be complicated with underlying osteomyelitis and can be detected with high sensitivity and specificity with MRI.⁵ One of the main learning points from this case report is the importance of investigating the possible underlying osteomyelitis with MRI earlier in a case of chronic non-healing ulcers for early detection and surgical removal of osteomyelitic bone to prevent other complications, such as abscess and septicemia.

Conclusion

Pressure ulcers are still a common complication in SCI despite updated knowledge, awareness, and devices that support pressure ulcer prevention. Chronic pressure ulcers are difficult to manage. Therefore, many cases are left with open pressure ulcers for many years. Chronic non-healing pressure ulcers are prone to recurrent infections, which can eventually lead to abscesses, septicemia, and risk of mortality. When a patient with SCI with a chronic non-healing pressure ulcer presents with septicemia, a high index of suspicion for deep-seated abscess is warranted to proceed with appropriate investigations and management, especially when treatment for other common sources of infection fails in achieving clinical improvement.

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