CASE REPORT

Multifocal septic arthritis, gluteal abscess and spondylodiscitis by *Streptococcus dysgalactiae* subspecies *equisimilis* after an intramuscular injection

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SUMMARY

We present the case of a 63-year-old man, admitted for hand cellulitis and acute kidney injury. A *Streptococcus dysgalactiae* subsp *equisimilis* (SDSE) was isolated in blood cultures and despite directed intravenous antibiotherapy, the patient evolved unfavourably, with dorsolumbar spondylodiscitis, multifocal septic arthritis and abscesses. CT also showed densification of the gluteal muscles, multiple air bubbles in the psoas, paraspinal muscles and spinal canal that were associated with an intramuscular injection administered 1 week earlier for a backache. After escalation of the antibiotherapy and intensive supportive measures, the patient showed improvement and was discharged after 8 weeks of antibiotherapy.

The incidence of invasive SDSE infections has been growing, especially in immunosupressed patients. In this case, despite no predisposing factor identified, it evolved to severe sepsis. The intramuscular injection, a trivialised but not harmless procedure, was the assumed port of entry, as previously described in another case report.

BACKGROUND

Streptococcus dysgalactiae subsp *equisimilis* (SDSE) is a coloniser of the respiratory, gastrointestinal and genitourinary tracts which has been recognised as an important pathogen, responsible for invasive and non-invasive infections, particularly of soft tissues.[1] It preferably affects patients with predisposing factors (neoplastic disease, diabetes, cardiovascular disease, immunosuppression and skin integrity problem); 90%–96% of the cases of bacteraemia occur in these patients, showing high mortality (15%–18%).[2] Despite a growing number of reported cases, it remains an infrequent entity.[3]

Intramuscular injections are generally considered a convenient and efficient way to administer therapy, but by its invasive character also carries risks.

CASE PRESENTATION

A 63-year-old man, with known history of hyperuricaemia presented to the emergency room with pain in his left hip, with no other signs or symptoms or history of trauma. Anti-inflammatory therapy was administered intramuscularly in the right buttock, with symptomatic relief. Six days later he returned with signs of inflammation in the right hand and fever 38.7°C. Blood pressure on admission was 120/70 mm Hg, heart rate 90 bpm. Blood tests showed a significant increase in the inflammatory parameters (white blood cells (WBCs) 34100/mcL, C reactive protein (CRP) 365 mg/L) and mild renal impairment (urea 83 mg/dL and creatinine 1.81 mg/dL), glucose 94 mg/dL.

The patient was hospitalised with a diagnosis of hand cellulitis. Antibiotic therapy was initiated empirically with penicillin and clindamycin, after collecting blood cultures. Complementary investigations revealed negative serologies for hepatitis B, hepatitis C and HIV, negative Huddleson's reaction. Transthoracic and transoesophageal echocardiograms showed no vegetations.

The patient showed clinical deterioration, with fever, intense lumbar pain and hypotension. Blood tests with persistently elevated inflammatory parameters, increased cholestasis parameters, γ-glutamyl transferase 156 U/L, alkaline phosphatase 292 U/L; aspartate transaminase 54 U/L; alanine transaminase 38 U/L; hyperbilirubinaemia with total bilirubin 4.50 mg/dL and direct bilirubin 3.06 mg/dL; international normalized ratio (INR) 1.26, hypoalbuminaemia (18.5 g/L) and isolation of SDSE (sensitive to the ongoing antibiotherapy) in two blood cultures. On the fourth day of hospitalisation a computed tomography (CT) scan was performed for clarification of low back pain, which identified densification of the gluteal muscles on the right, multiple air bubbles in the psoas and paraspinal muscles as well as in the spinal canal (figure 1) that were associated with the intramuscular injection. Magnetic resonance imaging (MRI) at day 10 documented dorsolumbar spondylodiscitis from D4 to S1 with multiple intracanalar abscesses (figures 2, 3 and 4) and no indication for surgical intervention.

In spite of directed antibiotic therapy, the patient showed an unfavourable evolution with emergence of a new abscess in his left foot, followed by massive bilateral pleural effusion with significant dyspnoea. A thoracentesis was performed, draining blood-stained fluid compatible with exudate (leucocytes 1400 /uL; proteins 31.3 g/L; lactate dehydrogenase 646 U/L); direct examination for bacteria and mycobacteria and cultural exam of the pleural fluid were negative. Aspiration of the gluteal collections and right sacroiliac arthrocentesis were performed, the fluids collected were sterile. Due to the pain worsening, a new CT was held on the 15th day of hospitalisation, to exclude new focus of infection,

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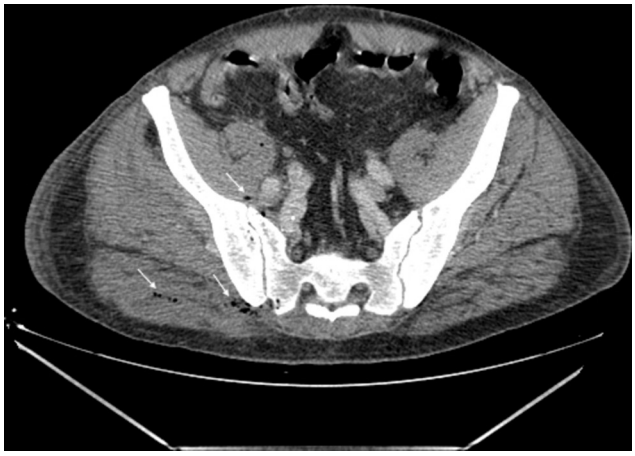

Figure 1 CT scan: densification and oedema of the gluteal and right psoas muscles with air bubbles inside (marks).

which showed signs of right sacroiliitis with pseudoerosive aspects and reduction of joint interface; extensive oedema in the gluteus muscle region with two poorly defined intramuscular collections, and disappearance of the previously identified air bubbles.

In addition, the patient passed fresh blood per rectum with circulatory compromise (demanding the transfusion of a total of 10 red cell concentrate units). The first colonoscopy only documented diverticula without active bleeding; the second colonoscopy held identified a diffuse haemorrhage in the ileocaecal valve. Embolisation by angiography was successfully performed, without further visible blood losses and haemodynamic and haematological stability. Faced with severe sepsis, with cultural exams persistently negative, antibiotic therapy was scaled up for meropenem and linezolid (keeping clindamycin) on the 12th day of antibiotherapy, with progressive clinical improvement. Intense pain was controlled with opioid therapy. The bone scintigraphy confirmed inflammation of dorsolumbar and

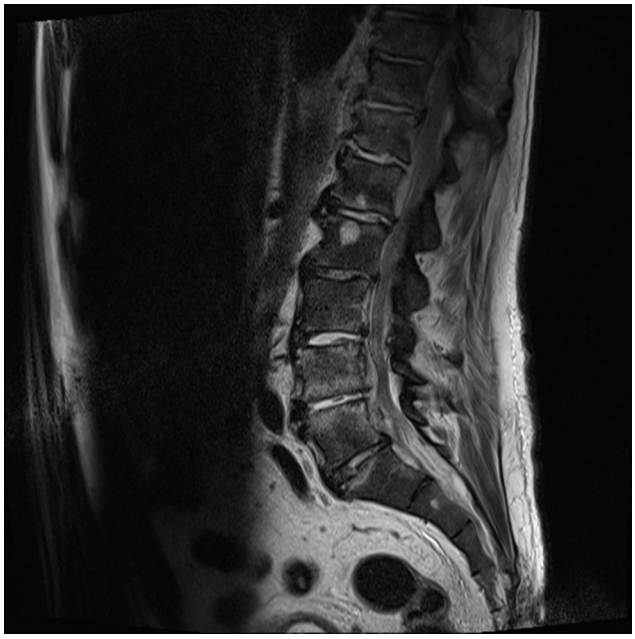

Figure 2 MRI of the column: irregularity of the vertebral platforms and decreased disc height with signal uptake, suggestive of inflammatory/infectious process.

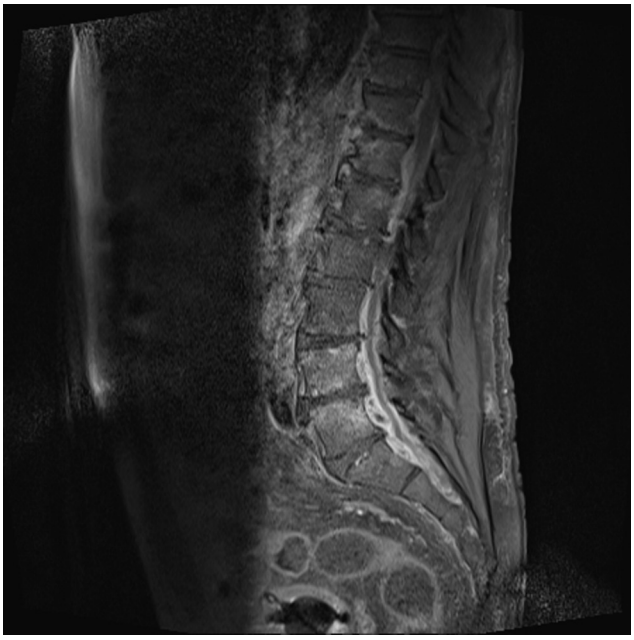

Figure 3 MRI of the column: fusiform dorsolumbar epidural collections.

right gluteal regions, carpi, right elbow, sacroiliac joints and left tarsus, without osteomyelitis (figure 5).

OUTCOME AND FOLLOW-UP

The patient completed the antibiotherapy—3 weeks of linezolid, 8 weeks of meropenem and 10 weeks of clindamycin—with gradual regression of the inflammatory parameters (WBC 7000/mcL, CRP 53.7 mg/L at discharge) and good clinical outcome under physiotherapy. In the follow-up appointment, 8 weeks after discharge, the patient showed significant symptomatic and functional improvement of the joints involved, tolerating the suspension of opioids. Control MRI still documented plurisegmentar

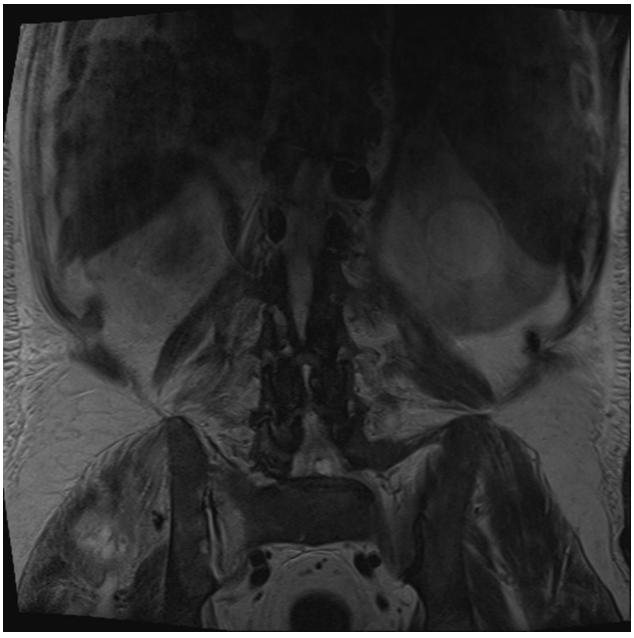

Figure 4 MRI of the column: right sacroiliitis signs and ipsilateral gluteal collections.

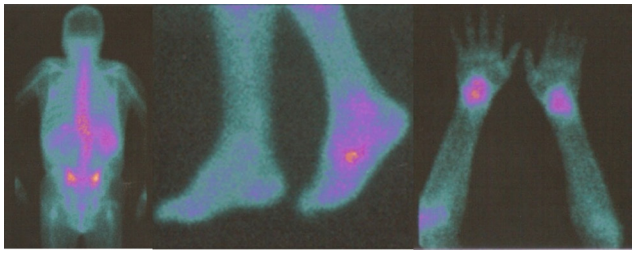

Figure 5 Gallium scintigraphy: pathological osteoarticular hyperfixation with involvement of both carpi, sacroiliac joints and left tarsus.

spondylodiscitis with reabsorption of the dorsolumbar collections, and regression of the posterior epidural empyema at L3/L4.

During the 1 year of follow-up, the patient maintained a positive evolution without intercurrent diseases or development of signs or symptoms of diabetes, cardiovascular or neoplastic disease, with an excellent performance status and resolution of the lesions in the RMI control at 1 year (figures 6 and 7).

DISCUSSION

SDSE is a β-haemolytic, pyogenic streptococcus classically belonging to Lancefield groups C and G.[4] In more recent taxonomic studies, a new division was suggested by Vandamme *et al*, based on the origin of the streptococci: *S.* dysgalactiae were to be separated between large colony-forming groups C and G streptococci of human origin—SDSE—and the veterinary pathogens group C streptococci—*S. dysgalactiae* subsp *dysgalactiae*.[5] For many years, SDSEs were considered non-pathogenic, mere colonisers of the human upper respiratory, gastrointestinal and female genital tracts, also often identified in skin lesions. More recently, SDSE has been recognised as an important bacterial pathogen resembling *Streptococcus pyogenes* in the clinical presentation of the invasive infection and sharing many of its

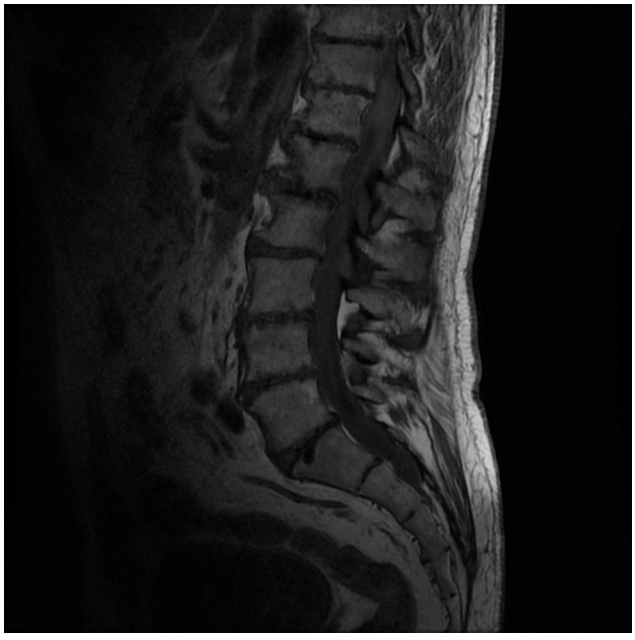

Figure 6 MRI of the column: improvement of the multicompartmental infectious process with disappearance of the epidural and paravertebral collections.

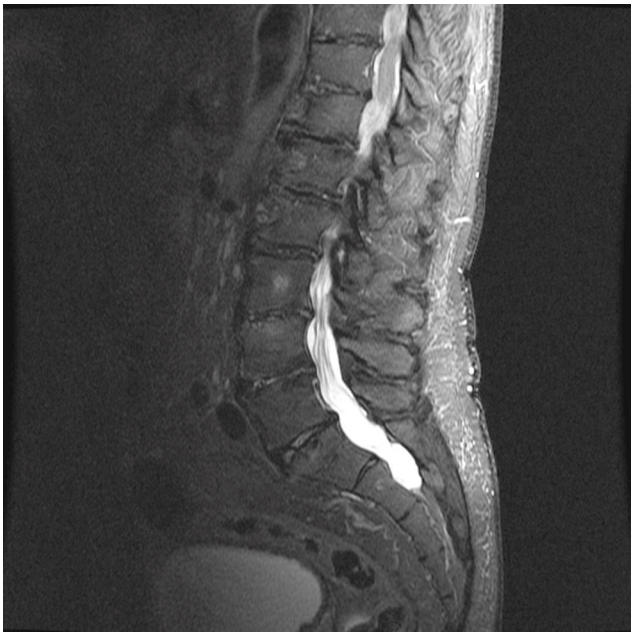

Figure 7 MRI of the column: small areas of subchondral bone oedema with discrete hypersignal T2 uptake of the disc and still with intense contrast uptake where there was previously a spondylodiscitis process.

virulence factors.[1 2 6] Colonisation sites and focal infections are the main reservoirs for transmission that occurs from person-to-person. SDSEs are easily disseminated by aerosols from the nose and throat of colonised individuals and also through direct contamination of wounds. SDSE infections are mainly community-acquired. SDSE causes a broad spectrum of superficial, deep, toxin-mediated or immunologically mediated diseases. Pharyngitis is a classic presentation, as are skin and soft-tissue infections, including cellulitis/erysipelas, wound infections, pyoderma and abscesses.[7] Severe invasive infections are almost always associated with immunosuppressed patients, skin breakdown or primary sites of colonisation/infection as port of entry (eg, intravenous drug users).[8] The disruption of the cutaneous barrier has been enhanced as a very important factor in the development of bacteraemia and invasive infections, which comprise arthritis, osteomyelitis, myositis, necrotising fasciitis, pleuropulmonary infections, peritonitis, intra-abdominal and epidural abscesses, meningitis, endocarditis, puerperal septicaemia and neonatal infections.[3 9 10] In recent years, SDSEs have also been isolated in cases of streptococcal toxic shock syndrome (which was previously only associated to *S. pyogenes* and *Staphylococcus aureus*) with a reported mortality rate of 66% in 15 cases from the literature.[10–13]

The intramuscular administration of medication per se disrupts the cutaneous barrier, causing tissue damage and creating a port of entry to the bacteria from the skin flora, even when correctly executed and in aseptic conditions.[11] In the last decades, several health institutions, namely WHO have drawn attention to the risks associated to intramuscular injections, especially in the transmission of infections, suggesting skin preparation and disinfection with 60%–70% alcohol.[14–17] Local infectious complications such as cellulitis, subcutaneous abscesses and necrotising fasciitis have not been infrequently described, especially related to the administration of non-steroidal anti-inflammatory drugs (NSAIDs).[18 19] Haematomas, nerve damage, quadriceps contracture, among others, are also possible hazardous effects of intramuscular injections, sometimes not taken into consideration

when deciding the medication administration route. Some pharmacokinetic characteristics of intramuscular administration may provide an advantage in specific scenarios—for example, the faster onset of action than subcutaneous injections or the prolonged duration of the drug's effect, which is important in antibiotic, hormonal or neuroleptic depot formulations. It is also an alternative when the patient cannot tolerate an oral medication, or when compliance is uncertain.[20 21] In addition to this is the patients' perception that an injection is more potent than standard oral treatment, which may lead physicians to favour this route.[22] However, some experts defend that in most cases, current evidence does not support the intramuscular route over the oral route for commonly intramuscular administered drugs.[23] Regarding the effects of intramuscular administration of NSAIDs, studies that compared an oral NSAID (ibuprofen or indomethacin) to intramuscular ketorolac have not demonstrated a significantly better response to the intramuscular route.[24–28] The choice of a drug's administration route should therefore take all these factors into account, weighing the benefits and risks to each individual patient—in this case, the injection of NSAIDs probably did not bring much benefit to the initial hip pain.

An increase in cases of bacteraemia and of severe infection due to group G β-haemolytic streptococci in humans has lately been recognised as associated with a mortality rate of up to 18%.[7 9 10 29–31] The literature suggests an association between SDSE bacteraemia or other invasive infection and underlying conditions, like diabetes mellitus, cardiovascular disease, chronic skin condition, neoplastic diseases, alcoholism or use of immunosuppressive medication.[7 10 32] The increase in the number of patients presenting with these conditions, due to longer survival and broader therapeutic options, may be an explanation for the increase of the incidence of SDSE infections, in addition to its virulence factors.[2 33]

In the present case, despite exhaustive research, no immunosuppressive or predisposing factor for SDSE infection was identified, neither during the hospital stay nor during the 1-year follow-up. The locations of the focal infections also stand out. Primarily presenting with cellulitis (the most frequent presentation of bacteraemia) and multiple abscesses, the involvement of intervertebral discs complicated the clinical picture and prolonged the antibiotherapy. SDSE is often associated to soft tissue infections, and despite less frequently, the intervertebral discs may also be a preferred territory of SDSE, as described in previous reports.[34–36] Parallel to the growing incidence of this infection, SDSE will probably be more frequently encountered in atypical settings, for example, the three cases of vertebral osteomyelitis described by Kumar *et al*.[37] The manifestations of spondylodiscitis are often non-specific and a high level of suspicion may therefore be necessary.

In spite of the apparently benign clinical presentation, the patient evolved unfavourably, despite adequate antibiotic treatment, highlighting the need for close monitoring of these infections. The decision to change the antibiotherapy was taken based on the clinical deterioration of the patient under penicillin and clindamycin, although the antibiogram showed sensitivity to those antibiotics. There is no evidence to presume a different sensitivity in vivo than in vitro, as studies performed so far showed strains of SDSE to be almost uniformly susceptible to penicillin and other β-lactam agents.[38] Therefore, the recommended first-choice treatment of spondylodiscitis caused by penicillin-sensitive streptococci is penicillin G, ceftriaxone or cefazolin.[39 40] In a study about molecular characterisation of group G streptococci, antibiotic testing of 290 isolates showed susceptibility to ampicillin, penicillin, ofloxacin and cefotaxime.[41] As expected, SDSE also showed sensitivity to vancomycin and linezolid.[42] Resistance to clindamycin, erythromycin, azithromycin and tetracycline was detected in 6.6%, 8.6%, 9.7% and 37.6% of isolates, respectively—[41]which is concordant with other studies, with a reported resistance as high as 60% for tetracycline and 71% for clindamycin.[13 38 42–44] Regarding the use of fluoroquinolones, a study characterising the molecular mechanisms of levofloxacin resistance in 314 isolates of SDSE responsible for infections in humans in Portugal found a high proportion of levofloxacin-resistant isolates of 12%.[45]

With the exception of the initial blood cultures, all the other cultures (pleural fluid, fluid of the gluteal collections, of the sacroiliac joint and subsequent blood cultures) were sterile, so we could not confirm the suspicion of a superinfection. A potential factor for the improvement after changing from penicillin to meropenem, and linezolid (keeping clindamycin) could be the low penetration of β-lactam agents into the vertebral disc.[46–49] Linezolid has good penetration into the bone and has proven to be efficient in the treatment of spondylodiscitis,[50] but further studies are needed to confirm these findings. Taking into account the severity of the clinical situation, the decision was made to keep meropenem, which also penetrates well in almost all body tissues.[39 47] A swift and aggressive therapeutic intervention enabled the recovery of the patient. One year after completion of antibiotic therapy the patient shows complete resolution of the lesions on MRI and a very good functional status.

The intramuscular injection was the only point of entry identified, as previously described in one case report of the literature reviewed.[11] Since no immunosuppressive factor was identified, one hypothesis is that the inoculum entering through the intramuscular injection was great enough to overcome the immunological defensive mechanisms of the host, causing a multifocal invasive disease with severe sepsis. This points out the possible iatrogenic complications related to a procedure so trivialised in clinical practice as administering a drug intramuscularly, which led to a severe invasive infection, with a long hospitalisation, multiple exams, invasive techniques, a prolonged broad-spectrum antibiotherapy, and, most importantly, jeopardised the patient's life.

As stated by WHO: 'An injection should only be given if it is necessary—and each injection that is given must be safe'.[16]

Learning points

- *Streptococcus dysgalactiae* subsp *equisimilis* (SDSE) infection, despite commonly associated with immunosuppression factors, can occur in immunocompetent patients with severe presentations.
- SDSE often causes invasive soft tissue infections. The intervertebral discs may also be a preferred territory.
- The administration of intramuscular therapy is a possible form of inoculation of this pathogen.
- Infection prevention practices are important to avoid iatrogenic complications, as well as the evaluation of the benefit of intramuscular injections as a drug administration route.

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Competing interests None declared.

Patient consent Obtained.

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Unexpected outcome (positive or negative) including adverse drug reactions

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