Fatal streptococcal necrotizing fasciitis as a complication of axillary brachial plexus block

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A 74-yr-old diabetic woman developed necrotizing fasciitis of the right upper limb after axillary brachial plexus block for carpal tunnel decompression. Clinical signs included oedema, diffuse swelling and bullae; rapidly followed by toxic shock syndrome and multiorgan failure. The patient died 48 h after hospital admission, despite broad-spectrum antibiotics, surgical treatment and supportive measures for the management of shock and multiorgan failure. Cultures yielded group A Streptococcus. Delay in antibiotic and surgical treatment probably affected the outcome. Early diagnosis and treatment are essential to improve the outcome of streptococcal necrotizing fasciitis.

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The risk of infection during regional anaesthesia is very low, epidural catheter-related infections are the major cause of this problem after neuroaxial block.1 We present a case of fatal necrotizing fasciitis as a result of group A Streptococcus complicating axillary brachial plexus block.

Case report

A 74-yr-old woman with a history of insulin-dependent diabetes mellitus, hypertension, and chronic atrial fibrillation was admitted for right carpal tunnel decompression. A single incision arthroscopic technique was used to release the carpal tunnel.2 Anaesthesia was provided by an axillary brachial plexus block. The procedure was performed without neurostimulation in the operating room using a sterile technique including wearing of theatre cap, sterile gloves, and gown. Povidone iodine 10% was used for skin disinfection. A single injection (30 ml) of bupivacaine 0.25% was administered with a disposable needle. The patient’s diabetes was well controlled before the procedure. No perioperative antibiotics were given, in keeping with the Société Française d’Anesthésie et de Réanimation recommendations. The patient was discharged the same day.

Four days later, she complained of axillary pain associated with erythema and oedema localized to the regional anaesthesia puncture site. The next day, she was admitted to hospital with oedema extension, increasing pain, and fever. The patient had not taken non-steroidal anti-inflammatory agents, and the incision related to carpal tunnel syndrome was healing well. Her temperature was 38.5°C, heart rate 122 beats min⁻¹, arterial pressure 120/80 mm Hg, the ventilatory frequency 20 bpm and the coma Glasgow score was 15. There was swelling, oedema, diffuse erythema, and haemorrhagic bullae covering 20% of the right upper limb and axilla.

Laboratory findings showed mild renal impairment (creatinine 142 μmol litre⁻¹, urea nitrogen 12 mmol litre⁻¹), elevated creatine kinase (1091 iu litre⁻¹), hyponatraemia (128 mmol litre⁻¹), hyperglycaemia (10 mmol litre⁻¹), and elevated C-reactive protein (428 mg litre⁻¹) without polynucleosis (white blood cell count 9.6×10⁹ litre⁻¹, neutrophils 6.8×10⁹ litre⁻¹). Other laboratory results were normal. ECG showed sinus tachycardia at a rate of 130 beats min⁻¹. Chest radiograph was normal. Radiograph and Doppler ultrasonographic examination of the right upper extremity were normal.
Shock and tachypnea developed 4 h later. Dopamine (15 µg kg⁻¹ min⁻¹) and dobutamine (20 µg kg⁻¹ min⁻¹) infusions were started after a large fluid infusion (2000 ml of hydroxyethylstarch and 1000 ml of saline 0.9%), the trachea was intubated and ventilatory assistance commenced. Piperacillin/tazobactam and amikacin were the first antibiotics to be administered 3 h after hospital admission and more than 24 h after first symptoms. The patient was transferred to our intensive care unit. Her haemodynamic status worsened, lactic acidosis, diffuse intravascular coagulation, anuric acute renal failure, and life-threatening multiorgan failure and the isolation of group A Streptococcus pyogenes. 5 New methods of diagnosis have been pursued with computerized tomography and magnetic resonance imaging (MRI). However, there are marked systemic symptoms, which may include shock and organ failure.3 The early onset of shock and organ failure and the isolation of group A Streptococcus from a normally sterile site are the defining characteristics of the streptococcal toxic shock syndrome.5 New methods of diagnosis have been pursued with computerized tomography and magnetic resonance imaging (MRI). However, confusion can occur with MRI between cellulites and necrotizing fasciitis. Because the sensitivity of MRI exceeds its specificity, MRI can overestimate the extent of deep facial involvement.6

Treatment of streptococcal necrotizing fasciitis includes broad spectrum antimicrobial regimens, prompt and aggressive exploration and debridement of suspected deep seated infection, and supportive measures for the management of shock and multiorgan failure.4 Piperacillin/tazobactam and amikacin were administrated to the patient more than 24 h after first symptoms. In addition, surgical intervention was performed 24 h after hospital admission because of profound shock. This delay in antibiotic and surgical treatment probably affected the outcome. New approaches to treatment include combination therapy with penicillin and clindamycin for an enhanced bactericidal effect. I.V. immunoglobulin has been shown to reduce mortality if the necrotizing fasciitis is associated with toxic shock syndrome.6

A portal of entry from the patient’s skin or from the anaesthetist’s oropharyngeal cavity was suspected. Unfortunately, we were unable to determine whether the anaesthetist was wearing a facemask or not during the regional anaesthesia. Furthermore, no swab was performed to determine the anaesthetist’s carrier status. However, it was not the anaesthetist’s usual preference to wear a facemask. In a postal survey of members of the obstetric anaesthetists association in the UK and Ireland, 273 of the 539 respondents (50.6%) did not wear masks for either spinal or epidural blocks, only 83 out of 259 mask wearers changed their masks between cases, and 50 out of 240 (21%) of those who routinely wore masks did not believe that wearing a mask reduced the risk of infection.7 However, surgical facemasks have been demonstrated to be effective in reducing bacterial contamination caused by dispersal from the upper airway.8 Povidone 10% iodine was used for the patient’s skin preparation. However, there was no record of how long it was allowed to dry. Since this case, usual strict asepsis measures have been continued in the operating room. In addition, wearing a facemask and allowing time to ensure effective antibacterial action of antiseptics have been recommended for the practice of regional anaesthesia.

Other potential sources of infection are contaminated anaesthetic solutions or syringes. However, local anaesthetics have bactericidal activity against commonly encountered skin organisms.9 The bactericidal effect is concentration-related and has been shown to be most marked with bupivacaine 0.5%, and less with lower concentrations.10 The axillary approach compared with other approaches to the brachial plexus may be associated with more risk of infection as a result of hidradenitis and general hygiene issues. However, to our knowledge, no study has demonstrated a significant difference in infection complications between the axillary approach and others.

Streptococcal necrotizing fasciitis is a rare but potentially lethal acute complication that should be acknowledged and
recognized by all anaesthetists involved in the practice of regional anaesthesia. Early diagnosis and treatment are essential to improve the outcome of streptococcal necrotizing fasciitis. Rigorous adherence to the principles of asepsis is the foundation of regional anaesthesia-related infection prevention.

References