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Back Pain Following Postoperative Epidural Analgesia: An Indicator of Possible Spinal Infection

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Infection of the spinal epidural space is a rare but potentially devastating disorder. The estimated incidence is between one in 10,000 and one in 400,000[1], or 0.2 to 1.2 cases per 10,000 hospital admissions[2,3]. There is evidence that the incidence is increasing[2,4,5]. Whilst the majority of cases are unrelated to the presence of an epidural catheter, infection is a recognized complication of epidural analgesia. The real incidence associated with epidural catheters is unknown. As the popularity of this form of analgesia is increasing, particularly for the management of postoperative pain, it is to be expected that an increase in the number of cases associated with epidural analgesia will be seen.

Early detection and appropriate management of infection of the epidural space is important if irreversible neurological sequelae are to be avoided. However the diagnosis of epidural space infection can be difficult as the signs and symptoms may be minimal and vague and thus be difficult to distinguish from other sources of infection in the body.

Three cases of suspected epidural space infection presenting with back pain are described below.

All three cases were patients admitted for the elective repair of an abdominal aortic aneurysm. Their perioperative management was similar: general anaesthesia, invasive monitoring with an intra-arterial cannula and a central venous catheter, and antimicrobial prophylaxis with intravenous cephalothin. Prior to the induction of anaesthesia, an epidural catheter was sited either in the lower thoracic or lumbar region, using an aseptic technique. Postoperative analgesia was maintained via the epidural catheter with a continuous infusion of the standard mixture used by the Acute Pain Service (APS) at the Royal Adelaide Hospital (bupivacaine 0.08% and fentanyl 4 μg.ml^{-1}). All patients were visited daily by the APS.

CASE 1

An 85-year-old woman had a past medical history of diverticular disease. Her only medication consisted of aspirin 150 mg daily. A lumbar epidural catheter was inserted at L3-4. Cephalothin was continued for 24 hours postoperatively. There were no problems until the fifth postoperative day when the patient developed a temperature of 37.5°C and complained of pain at the epidural site which was noted to be inflamed and indurated. The epidural and central venous catheters were removed. Culture of the epidural catheter tip resulted in a heavy growth of *Staphylococcus aureus* and *Proteus vulgaris*. On day 7 she complained of backache and in view of the epidural catheter tip culture result, intravenous amoxycillin was commenced.

On day 10 a mild right quadriceps weakness was noted and a neurosurgical opinion was obtained. Magnetic resonance imaging (MRI) was performed which suggested the presence of an inflammatory track at L3-4 and a small epidural collection posteriorly with no mass effect.

A conservative nonoperative course of management was adopted and amoxycillin continued for a total of six weeks. Her course was complicated by a large gastrointestinal bleed on day 14. A repeat MRI on day 23 showed some improvement and the patient was well enough to be discharged home on day 27.

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CASE 2

A 68-year-old man with a history of ischaemic heart disease had had a splenectomy performed a month previously for idiopathic thrombocytopenic purpura. He was on no medication. The epidural catheter was sited at T8-9. Postoperatively he had a low-grade pyrexia despite the continued course of cephalothin and on day 3 the temperature was 38.5°C. The epidural site was noted to be slightly erythematous but he had no pain at the site. On day 4 he complained of buttock pain but there were no focal neurological signs. The epidural and central venous catheters were removed. Culture of the epidural catheter tip resulted in a scant growth of *Pseudomonas aeruginosa* and *Staphylococcus epidermidis*. On day 6 he developed intense back pain and local tenderness. An MRI was performed, which suggested an epidural abscess posteriorly in the upper thoracic region. On day 7 he complained of tingling in his right foot. A neurosurgical consultation was obtained and a two-level laminectomy was performed that day. Although the epidural fat looked abnormal at operation, all microbiological cultures were negative and on histological examination there were no signs of inflammation. Cephalothin was continued for a total of six weeks. The patient was discharged on day 17.

CASE 3

A 61-year-old man had a past history of moderately severe chronic obstructive airways disease, gout and chronic lumbar pain. His medication consisted of a salbutamol inhaler.

An epidural catheter was sited at T11-12 and antibiotics were continued for 24 hours. On the fifth postoperative day he developed a low-grade pyrexia and complained of gout-like pain in his left hallux for which indomethacin and colchicine were commenced. In view of the persisting pyrexia, cephalothin was recommenced on day 7. The epidural and central venous catheters were removed and on culture of the latter there was a heavy growth of *S.aureus* which was sensitive to cephalothin. On day 10 the temperature increased to 38°C and he complained of back pain. He was discharged home three days later on oral cephalexin and paracetamol.

Two months later he was re-admitted with very severe thoracolumbar pain. The ESR and C-reactive protein (CRP) were markedly raised despite a normal white cell count. Plain X-rays and MRI demonstrated advanced degenerative disease of the spine. Localized 99m technetium methylene diphosphonic acid (99mTc MDP) bone studies showed no evidence of osteomyelitis or discitis although a gallium scan was suggestive of a lumbar epidural space infection without abscess formation. The possibility of an aortic graft infection was raised. Neurosurgical opinion was that surgery was not indicated but that cephalexin should be recommenced. In view of the result of the culture from the central venous catheter, flucloxacillin was also commenced. He was discharged home on longterm antibiotics because of the possibility of an infected aortic graft or discitis.

Five months later he was again admitted with severe back pain. The MR and CT scans were esssentially unchanged and the whole body 99m technetium hexamethylpropyleneamineoxime (99mTc HMPAO) labelled white cell study was normal. The plain X-rays were now very suggestive of lumbar (L1-4) discitis and osteomyelitis. Aspiration of lumbar disc material confirmed the diagnosis of pseudomonas discitis and osteomyelitis and the patient was prescribed intravenous piperacillin and gentamicin (subsequently changed to tobramycin). An MRI scan a month later confirmed a three-level discitis but no epidural abscess formation. A Hickman catheter was inserted and the patient was discharged on the above intravenous antibiotics and oral ciprofloxacin.

On readmission a month later, he had a lumbar kyphosis and MRI showed indentation of the theca with canal stenosis. He was reviewed by the spinal unit who felt that the risks of surgical intervention far outweighed the potential benefits. A spinal brace was prescribed.

Aspiration of the lumbar discs two months later showed no evidence of infection and the patient was discharged home on ciprofloxacin and ceftazidine, the latter replacing the aminoglycosides due to the development of ototoxicity. A year later the antibiotics were stopped and two years later, the plain X-rays and CT scan showed resolution of the discitis and osteomyelitis. The exact source of the infection is still unclear. The possibilities included the aortic graft, epididymitis (two-month history of pain in the left testis), the epidural catheter and a urinary tract infection due to perioperative catheterization. The Infectious Diseases Unit felt that the last possibility was most likely in view of the initial resistance of the infection to antibiotic therapy.

DISCUSSION

Infection of the spinal epidural space can have devastating consequences. An epidural infection is usually associated with an existing infection elsewhere in the body, organisms gaining access to the

epidural space by haematogenous or direct spread. Haematogenous spread is most commonly from a skin, respiratory or urinary tract infection[2,6]. The infecting agent is most often *S.aureus*, but streptococci, gram negatives and mixed aerobes are also common[7,8]. Pelvic infections may spread by a retrograde venous route to the spinal epidural venous plexus. Direct spread may occur from discitis or vertebral osteomyelitis.

Epidural infection is a recognized complication of epidural anaesthesia[2,9-12] and lumbar puncture[4,13], but must be extremely rare in relation to the number of epidural anaesthetics given[9,14]. Two large prospective series of epidural anaesthesia failed to document a case of infection[15,16]. Possible sources of infection in catheter-related cases include direct inoculation at the time of catheter insertion and infusion of contaminated fluid[10,17]. The infecting organism is almost always *S.aureus* or *S.epidermidis*[12,18]. Immunocompromised and diabetic patients have been shown to have an increased risk of developing spinal epidural infections[1,4,7,9].

The most common site of spinal epidural infection is reported to be in the thoracic region dorsal to the dura[3,4,19], possibly related to the distribution of epidural fat which is particularly susceptible to infection[10,20]. However other authors report a higher incidence in the lumbar spine[2,21]. Cases related to epidural catheterization occur at the catheter site[17,22], although a predilection for the thoracic region has been reported[12]. In acute infections, granulation tissue and pus extend over an average of four segments[23,24]. Infection in chronic cases is more extensive. The dura is an effective barrier so that concomitant subdural or subarachnoid infection is rare. Neurological deficits result from a combination of pressure from the inflammatory mass and from arterial or venous thrombosis causing infection[4,24]. There can be a precipitous deterioration in acute or chronic cases, possibly related to sudden vascular occlusion[5].

The characteristic clinical presentation is of a febrile illness associated with focal spinal pain, followed by radicular pain, paresis and paralysis[23,25]. The presentation is however quite variable; back pain is the only consistent clinical feature[5,21]. The evolution of symptoms may be acute, over an average of seven days, or chronically over weeks or months. The diagnosis may be difficult in cases presenting with vague symptoms and minimal or no fever[2]. Laboratory tests may indicate the presence of infection but are not diagnostic. Imaging with contrast-enhanced CT will indicate the presence of an epidural mass and may also demonstrate vertebral involvement[24]. MRI, with and without contrast enhancement, is more sensitive at detecting early changes in epidural fat and will also clearly demonstrate the location and extent of the infection[26-29]. The usual findings are an epidural mass with an iso-intense or hyper-intense signal on T1-weighted images and an inhomogeneous hyperintense signal on T2-weighted images[21]. However it may be difficult to differentiate between pus and granulation tissue on MRI[3,4].

Major surgery such as abdominal aortic aneurysm repair provides many possible sources of infection. There may be contamination at the surgical site, leading to graft infection spreading to the intervertebral discs, vertebral bodies and epidural space. There may be haematogenous spread from contaminated or infected intravascular catheters or there may be spread from urinary infections related to bladder catheterization. The epidural catheter related infections presumably result from direct contamination with skin organisms or infusion of contaminated fluids. The three cases presented here had abdominal aortic surgery. Cross-clamping the aorta may interfere with spinal artery blood flow; ischaemia of the supplied tissues may predispose to infection. These cases demonstrate the difficulties in diagnosis of iatrogenic epidural space infections.

Epidural catheter related infections may be with organisms of low virulence, such as *S.epidemidis*[12,14]. Whatever the organism, the presentation of catheter-related infections is often of chronic back pain without fever, rather than the typical acute febrile illness with neurological deficits[22,30-33]. It may be extremely difficult to determine whether the infection is primarily in the epidural space or has spread there from a primary infection elsewhere in the body. An additional compounding factor is that prolonged epidural analgesia may cause MR signal changes in the epidural fat at the site of infusion.

The traditional treatment of choice for epidural space infections has been surgical decompression and debridement, combined with antibiotic therapy[2,21]. Modern series still report mortality rates as high as 25%[4,5,7]. In general, the prognosis depends on the degree and duration of neurological impairment prior to operative decompression[2,19]. Patients with paresis of less than 24 hours duration or with no neurological loss should make an excellent recovery. Paralysis for greater than 48 hours portends no neurological recovery. An epidural abscess causing acute neurological deficits is therefore a neurosurgical emergency[34].

Many authors have recently demonstrated that nonoperative management has a satisfactory out-

come in selected cases[1,5,28,35-37]. Indications for antibiotic therapy alone include[21,36] (1) the presence of concomitant medical problems that increase anaesthetic risk, (2) complete paralysis for more than three days, (3) the absence of severe neurological deficits, (4) an abscess that extends over many segments, and (5) abscesses located in the lumbar epidural space. These are relative indications and the treatment strategy should take into account the factors in each case. The risk of sudden neurological deterioration in patients being treated with antibiotics alone[37] makes close monitoring mandatory, preferably with repeat MR scans[29]. The development of a neurological deficit or failure to improve are indications for surgical treatment.

Faced with the possible diagnosis of epidural space infection, the clinician should attempt to confirm the diagnosis with urgent MRI or contrast-enhanced CT scanning. Antibiotic treatment should be commenced immediately, preferably based on the results of cultures and sensitivities: epidural catheter tips should be cultured whenever the catheter has been in place for more than 24 hours. Immediate surgical drainage combined with antibiotics is the treatment of choice for patients with neurological deficits. Other patients can be treated with a course of intravenous and oral antibiotics for six to twelve weeks, but should be monitored closely.

Epidural analgesia is increasing in popularity as a technique for the management of acute pain, particularly postoperative pain. Although the risk of an epidural space infection following epidural analgesia is very small, the signs and symptoms are often minimal or vague, especially when associated with the presence of back pain, with or without an accompanying pyrexia. A high index of suspicion, early and detailed investigation and neurological assessment are therefore essential if the risk of permanent neurological sequelae is to be minimized.

At our hospital, management protocols have been formulated by the Acute Pain Service in conjunction with the Department of Neurosurgery (see Appendix). These are aimed at early detection and appropriate management of suspected or proven epidural space infections.

Every patient with an epidural catheter in situ is visited daily by the APS and Section A of the protocol implemented. Since clinical practice may vary between institutions and as all positive epidural catheter tip culture results will not necessarily require antibiotic therapy, it is recommended that each institution develop a protocol for the management of suspected epidural catheter related infection (for example Section B of Appendix). Early detection will increase the chances of successful, and possibly conservative management of an epidural space infection.

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APPENDIX

ROYAL ADELAIDE HOSPITAL ACUTE PAIN SERVICE—POSTOPERATIVE EPIDURAL ANALGESIA PROTOCOL

A. Early Detection of a Possible Catheter-Related Infection

1. Daily check for:
   - inflammation or induration of the epidural catheter insertion site (if present, remove the epidural catheter)
   - back pain at the epidural catheter insertion site or radicular pain
   - tenderness at the epidural catheter insertion site
   - pyrexia
   - neurological deficit (sensory, motor, sphincter tone)
2. All epidural catheter tips to be sent for culture if the catheter has been in situ for more than 24 hours.
3. All patients to be given the Acute Pain Service information sheet outlining the course of action to take, if, when discharged home, the patient notices any numbness or weakness of their legs, experiences difficulty with voiding or has increasing back pain.

B. Management of a Suspected Catheter-Related Infection

1. Localized infection at the epidural catheter insertion site

- culture any exudate and treat with antibiotics as appropriate
- consider surgical incision and drainage

2. Suspected epidural space infection

a. Presence of back pain and/or neurological symptoms and signs
   - seek an urgent neurosurgical consultation
   - organise an urgent MRI
   - antibiotic therapy alone may be appropriate in the absence of significant neurological deficit
   - immediate surgical intervention if neurological signs and symptoms are present and there are no contraindications to surgery

b. Positive culture of the epidural catheter tip
   - since not all positive cultures will necessitate treatment, especially in the absence of clinical symptoms and signs, microbiological advice is recommended for individual cases.

REFERENCES

1. Slade WR, Lonano F. Acute spinal epidural abscess. J Natl Med Assoc 1990; 82:713-716.
2. Danner RL, Hartman BJ. Update of spinal epidural abscess: 35 cases and review of the literature. Rev Infect Dis 1987; 9:265-274.
3. Teman AJ. Spinal epidural abscess. Early detection with gadolinium magnetic resonance imaging. Arch Neurol 1992; 49:743-746.
4. Hlavin ML, Kaminski HJ, Ross JS, Ganz E. Spinal epidural abscess: a ten year perspective. Neurosurgery 1990; 27:177-184.
5. Nussbaum ES, Rigamonti D, Standiford H, Numaguchi Y, Wolf AL, Robinson AL. Spinal epidural abscess: a report of 40 cases and review. Surg Neurol 1992; 38:225-231.
6. McGee-Collett M, Johnston IH. Spinal epidural abscess: presentation and treatment. Med J Aust 1991; 155:14-17.
7. Redekop GJ, Del Maestro RF. Diagnosis and management of spinal epidural abscess. Can J Neurol Sci 1992; 19:180-187.
8. Baker AS, Ojemann RG, Swartz MN, Richardson Jr EP. Spinal epidural abscess. N Engl J Med 1975; 293:463-468.
9. Mahendru V, Bacon DR, Lema MJ. Multiple epidural abscesses and spinal anesthesia in a diabetic patient. Reg Anesth 1994; 19:66-68.
10. North JB, Brophy BP. Epidural abscess: a hazard of spinal epidural anaesthesia. Aust NZ J Surg 1979; 49:484-485.
11. Scott DB, Hibbard BM. Serious non-fatal complications associated with extradural block in obstetric practice. Br J Anaesth 1990; 64:537-541.
12. Ngan Kee WD, Jones MR, Thomas P, Worth RJ. Extradural abscess complicating extradural anaesthesia for caesarean section. Br J Anaesth 1992; 69:647-652.
13. Bergman I, Wald ER, Meyer JD, Painter MJ. Epidural abscess and vertebral osteomyelitis following serial lumbar punctures. Pediatrics 1983; 72:476-480.
14. Ericsson M, Algers G, Schliamser SE. Spinal epidural abscesses in adults: review and report of iatrogenic cases. Scand J Infect Dis 1990; 22:249-257.
15. Scherer R, Schmutzler M, Giebler R, Erhard J, Stocker L, Kox WJ. Complications related to thoracic epidural analgesia: a prospective study in 1071 surgical patients. Acta Anaesthesiol Scand 1993; 37:370-374.
16. Tanaka K, Watanabe R, Harada T, Dan K. Extensive application of epidural anesthesia and analgesia in a University Hospital: incidence of complications related to technique. Reg Anesth 1993; 18:34-38.
17. Dawson P, Rosenfeld JV, Murphy MA, Hellyar AG. Epidural abscess associated with postoperative epidural analgesia. Anaesth Intens Care 1991; 19:569-572.
18. Mamourian AC, Dickman CA, Drayer BP, Sonntag VKH. Spinal epidural abscess: three cases following spinal epidural injection demonstrated with magnetic resonance imaging. Anesthesiology 1993; 78:204-207.
19. Statham P, Gentleman D. Importance of early diagnosis of acute spinal extradural abscess. J R Soc Med 1989; 82:584-587..
20. Carey ME. Infections of the spine and spinal cord. In: Youmans JR, ed. Neurological Surgery. Philadelphia, W. B. Saunders Co. 1990; 3759-3781.
21. Curling OD, Gower DJ, McWhorter JM. Changing concepts in spinal epidural abscess: A report of 29 cases. Neurosurgery 1990; 27:185-192.
22. Sowter MC, Burgess NA, Woodsford PV, Lewis MH. Delayed presentation of an extradural abscess complicating thoracic extradural analgesia. Br J Anaesth 1992; 68:103-105.
23. Huesner AP. Nontuberculous spinal epidural infections. N Engl J Med 1948; 239:845-854.
24. O'Sullivan R, McKenzie A, Hennessy O. Value of CT scanning in assessing location and extent of epidural and paraspinal inflammatory conditions. Australas Radiol 1988; 32:203-206.
25. Hulme A, Dott NM. Spinal epidural abscess. Br Med J 1958; 1:64-68.
26. Erntell M, Holtås S, Norlin K, Dahlquist E, Nilsson-Ehle I. Magnetic resonance imaging in the diagnosis of spinal epidural abscess. Scand J Infect Dis 1988; 20:323-327.
27. Bertino RE, Porter BA, Stimac GK, Tepper SJ. Imaging spinal osteomyelitis and epidural abscess with short T1 inversion recovery (STIR). AJNR-Am-J-Neuroradiol 1988; 9:563-564.
28. Hanigan WC, Asner NG, Elwood PW. Magnetic resonance imaging and the nonoperative treatment of spinal epidural abscess. Surg Neurol 1990; 34:408-413.
29. Sadato N, Numaguchi Y, Rigamonti D, et al. Spinal epidural abscess with gadolinium-enhanced MRI: serial follow-up studies and clinical correlations. Neuroradiology 1994; 36:44-48.
30. Nordström O, Sandin R. Delayed presentation of an extradural abscess in a patient with alcohol abuse. Br J Anaesth 1993; 70:368-369.
31. Strong WE. Epidural abscess associated with epidural catheterization: a rare event? Report of two cases with markedly delayed presentation. Anesthesiology 1991; 74:943-946.
32. McDonogh AJ, Cranney BS. Delayed presentation of an epidural abscess. Anaesth Intens Care 1984; 12:364-365.
33. Bromage PR. Spinal extradural abscess: pursuit of vigilance. Br J Anaesth 1993; 70:471-473.
34. Reynolds PC, Hahn MB. Early diagnosis of a spinal epidural abscess. Reg Anesth 1991; 16:57-58.
35. Mampalam TJ, Rosegay H, Andrews BT, Rosenblum ML, Pitts LH. Nonoperative treatment of spinal epidural infections. J Neurosurg 1989; 71:208-210.
36. Leys D, Lesoin F, Viaud C, et al. Decreased morbidity from acute bacterial spinal epidural abscess using computed tomography and nonsurgical treatment in selected patients. Ann Neurol 1985; 17:350-355.
37. Wheeler D, Keiser P, Rigamonti D, Keay S. Medical management of spinal epidural abscesses: case report and review. Clin Infect Dis 1992; 15:22-27.