

## EXTRADURAL HAEMATOMA AFTER CONTINUOUS EXTRADURAL ANAESTHESIA

I. H. TEKKOK, O. CATALTEPE, K. TAHTA AND V. BERTAN

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### SUMMARY

We report a case of extradural haematoma occurring after extradural anaesthesia in an anticoagulated patient. The diagnosis was confirmed by magnetic resonance imaging and the haematoma was evacuated surgically. A search of the literature revealed only five previous reports of extradural haematoma in association with extradural anaesthesia.

### KEY WORDS

Anaesthetic techniques: extradural. Complications: extradural haematoma.

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Extradural haematoma (EH) is an uncommon, but treatable cause of spinal cord compression. Since the original description by Jackson in 1869 [1], more than 200 cases of varying aetiology have been reported [2]. Trauma, anticoagulation, bleeding diatheses and intraspinal vascular malformations have been associated with EH, but occasionally it may occur without any apparent cause [2]. In recent years, more than 30% of reported cases have been associated with anticoagulant therapy [3]. Iatrogenic EH, although rare, may occur as a complication of lumbar puncture or extradural spinal anaesthesia [4]. We present a case of EH that occurred after continuous extradural anaesthesia while the patient was receiving anticoagulant therapy, and discuss the aetiology, diagnosis and treatment of this unusual complication.

### CASE REPORT

A 42-year-old man, with a history of diabetes mellitus and chronic pancreatitis of 4 years duration, developed left-sided intermittent claudication about 1 month before his admission to another hospital. Reduced pulsation of the left-sided dorsal pedal and posterior tibial arteries was

noted. A digital subtraction angiogram showed left external iliac artery obliteration. An ilio-femoral bypass graft was performed under extradural anaesthesia. The extradural space was entered with a single puncture at the L2-3 interspace with a 90-mm 17-gauge Tuohy needle. A 90-cm, 19-gauge flexible catheter with stilette introducer and open distal end (Vygon) was passed about 2-3 cm beyond the tip of the needle. Fifteen millilitre of 2% lignocaine was injected through the catheter. The procedure was uncomplicated and no haemorrhage was noted.

One hour after the start of surgery, the first dose of heparin 5000 u was given i.v. The extradural catheter was left *in situ* for the next 2 days for provision of pain relief and sympathetic block by the intermittent injection of 2% lignocaine. Meanwhile, heparin 5000 u twice daily and dextran 40 solution 1000 ml daily were given. During this period all pulses were present and full power and sensation were noted in the legs. The extradural catheter was removed approximately 48 h after insertion. Three hours later, the patient complained of paraesthesia and numbness of his feet which progressed to complete paraplegia within 12 h. An Iohexol-enhanced CT scan of the spine was performed, but surgery was not considered, under the impression that the scan was normal. No further heparin was administered.

Fourteen days after the onset of paraplegia the patient was brought to our centre by his relatives. On neurological examination, anaesthesia below T9 and total paraplegia were observed with no anal tone or reflex. A magnetic resonance (MR)

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ISMAIL H. TEKKOK\*, M.D., F.I.C.A.; OGUZ CATALTEPE, M.D.; KADIR TAHTA, M.D.; VURAL BERTAN, M.D.; Department of Neurosurgery, Hacettepe University School of Medicine, Ankara 06100, Turkey. Accepted for Publication: January 26, 1991.

\*Address for correspondence: 2 nci Cadde No 1-6, Kivanc Apt, Bahcelievler, Ankara 06500, Turkey.



FIG. 1. Sagittal MR view (GE 0.5 T, TR 600/TE 25). A fusiform hyperintense lesion is present behind the cord.



FIG. 2. Axial MR view (TR 740/TE 25). Hyperintense lesion 0.61 cm thick displaces the spinal cord anteriorly and to the left.

scan was performed and showed a posteriorly placed, "cigarette shaped" [5] haematoma extending from T12 to L1 on both T1- (fig. 1), proton density- and T2-weighted images. The lesion was displacing the spinal cord anteriorly and to the left (fig. 2). Although late, a decompressive laminectomy was considered to give a chance of recovery to the patient. Prothrombin, activated partial thromboplastin, clotting and bleeding times and platelet counts were found to be within normal limits.

A partial (inferior) T11 and total T12 and L1 laminectomy was performed. A 2×1×5-cm right-sided posterolateral extradural haematoma was found and removed. Histopathological examination revealed an organizing haematoma.

Upon discharge to a foreign rehabilitation centre, the patient demonstrated no major improvement in lower extremity function or sensation, but was mobile with the help of braces, facilitated by the spasticity that developed 1 month after operation.

## DISCUSSION

Although blood dyscrasias and anticoagulation therapy are recorded as contraindications to extradural or subarachnoid anaesthesia in most textbooks, there have been a few reports of extradural haematoma in patients given anticoagulants after catheter placement [6–10], mostly with poor outcome because of late diagnosis and late treatment. The incidence of EH after extradural anaesthesia may be greater, as such a complication is not always reported.

The extradural space contains loose areolar tissue and a dense network of large, thin-walled and valveless veins. When these veins are torn by a needle or catheter, a small amount of bleeding occurs into the extradural space. If haemostatic mechanisms are normal, the defect will be sealed, but if the patient's coagulation mechanisms are impaired, haemorrhage may continue or be re-started if the clot is dislodged by the withdrawal of the catheter.

The typical presentation of EH is acute back and radicular pain leading to paresis which may then progress to para- or tetraplegia over a variable time. We have found only one other report of "painless" EH as occurred in our patient [11].

Recovery from EH depends largely on the extent and duration of spinal cord compression and early recognition is mandatory. A high index of suspicion is necessary for early diagnosis, when detailed and frequent neurological examinations should be performed from the time of catheter insertion until 24 h after the catheter removal. Prolonged numbness or weakness, paraesthesia and severe back pain should prompt neurosurgical evaluation.

Conventional myelography may be useful in the diagnosis, but it requires insertion of a needle, which may be regarded as a relative contraindication to its use. It should be considered only after coagulation parameters return to normal and if alternative tests are not available. Myelography will show an extradural mass, but cannot differentiate between blood, pus or tumour. CT scanning may be useful but it, too, has disadvantages. First, it cannot survey satisfactorily all the spinal canal; second, resolution may be poor within the thoracic spinal canal compared with the lumbar or cervical regions. Typically, an EH would appear as a hyperdense, biconvex mass adjacent to the bony canal. Delineation of an EH may be improved by

CT-myelography, when characteristic extradural lenticular collections may be seen on either side of the midline [12].

Magnetic resonance imaging does not require either a lumbar puncture or ionizing radiation and is the preferred diagnostic technique for spinal imaging. On MR scans, an acute EH appears as iso- or minimally hyperintense on T1 and heterogeneously hyperintense on T2 images [5, 13]. Subacute EH show hyperintensity on both T1- and T2-weighted images which can be attributed to the paramagnetic effect of intracellular methaemoglobin with subsequent cell lysis and resultant free haemoglobin [14].

Although it has been emphasized that only emergency or early surgery of EH produces good results [15], there have been reports of occasional patients with spontaneous remission [3]. Notably, Shima, Mihara and Hachisuga reported a patient recovering completely after surgery 18 days after the onset of symptoms [16]. Percutaneous needle aspiration has been suggested as an alternative treatment [3]; nevertheless, EH almost always produces a permanent neurological deficit unless there is prompt surgical intervention.

We conclude that extradural anaesthesia remains contraindicated in the presence of impaired coagulation. If it is undertaken in these circumstances a high degree of clinical suspicion of EH should be maintained, with frequent neurological examinations from insertion of the catheter to 24 h after its removal. Early recourse to appropriate investigations and surgery is mandatory for successful management.

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