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Unexplained episode of sensory-motor deficit following lumbar epidural analgesia

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LETTERS TO EDITOR

A 17-year-old male underwent abdominal surgery (proctocolectomy) under general anesthesia and lumbar epidural analgesia at the L1-L2 level. At the end of surgery, continuous epidural analgesia was administered for post-operative pain relief with 0.1% ropivacaine and 1 µg/ml fentanyl at 7 ml/h.

Two hours after surgery, the patient complained of weakness and numbness in both lower limbs during the acute pain service rounds. Neurological examination revealed sensory deficit and decreased muscle power in both lower limbs (2/5 in the hip and knee flexors, and 1/5 in ankle planter and dorsiflexors). The surgical team was informed about the episode, epidural infusion was stopped, and neurosurgery consultation was sought. MRI was advised from the neurosurgery team.

Blood investigations revealed high prothrombin time [INR (international normalized ratio) = 2.43] in the post-operative period (the coagulation profile was normal in the pre-operative period). In addition to this, there were no signs of infection (e.g., fever) and no abnormal findings according to the hematologic investigations, or liver function and urinalysis tests. An injection of vitamin K and a fresh frozen plasma transfusion was planned for the raised INR.

On the first post-operative day, the patient reported a mild improvement in motor weakness. Neurological examination revealed an improvement in lower limb motor power (3/5 in the hip and knee flexors and 3/5 in the ankle planter flexion and dorsiflexion); however, there was no improvement in the sensory deficits. On the second post-operative day, INR was normalised to 1.2 and the epidural catheter was removed. The neurological examination revealed normal motor power (5/5) in the lower extremities though the sensory complaints were still present. An MRI done on the 2nd post-operative day revealed a normal study; there wasn't any thecal sac compression visualised in the MRI. The sensory symptoms resolved completely on the fourth post-operative day.

We were unable to identify a specific cause responsible for this event. A likely hypothesis could be epidural blood collection due to a deranged coagulation profile in the post-operative period. This collection could have resulted in thecal sac compression giving rise to sensory motor deficits. The collection would have been resolved spontaneously giving rise to a full recovery of sensory motor deficits. Other possibilities could have been epidural drug collection related to continuous epidural analgesia, spinal anesthesia with dural puncture, and mechanical irritation

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of nerves or the spinal cord by the epidural catheter.

Spontaneous epidural hematomas with spontaneous resolution have been reported in the literature as a rare cause; we hypothesize it as a possibility in our case. We could not identify blood collection in the epidural space in the MRI, possibly because it was done on the second post-operative day when the INR had been normalized. The hematoma may have completely resolved before the imaging modality was undertaken, as it also correlated with the resolution of the symptoms. In this context, cases have been reported in which radiological findings of epidural hematoma have disappeared within 9 hours of the symptom onset [1].

Other possible causes of motor weakness and numbness as suggested by the literature are ropivacaine neurotoxicity [2], bilateral polyradiculitis [3], anterior spinal artery infarct [4], or arachnoiditis [5] associated with neurological deficits following epidural analgesia with a normal MRI study. But in all these cases the neurological symptoms persisted for a longer duration of time as compared to the spontaneous resolution of symptoms in this case.

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