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Title: Aseptic subcutaneous inflammation presenting as late onset back pain after uneventful epidural anaesthesia.

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To the Editor,

We encountered a case of aseptic subcutaneous inflammation following epidural anesthesia (EA), which we want to share here.

A healthy female (26 years, G1P0) was admitted in active labour following uneventful pregnancy. She sought EA for labour pain. Antiseptic cleaning was done using chlorhexidine stick, and the target area was draped aseptically. After allowing adequate time for drying, skin and underlying subcutaneous tissue in the target needle entry site paramedian to the L3-4 intervertebral space were infiltrated using 2% lidocaine. Eighteen gauge epidural set was used for inserting epidural and epidural space was located using loss of resistance to saline technique. Epidural space was identified at 5 cm depth in first attempt uneventfully. Epidural catheter was then threaded and 5 cm of epidural catheter was kept inside epidural space. After a negative test dose, a loading dose of levobupivacaine was administered. Then an epidural infusion was started based on our institutional protocol. Patient had instant pain relief and remained comfortable throughout.

Ultimately a caesarean section (CS) was required due to failure to progress, and it was successfully performed using an epidural top-up. At the end of the surgery the epidural catheter was removed and a sterile dressing was applied. Total duration of epidural catheter was about 4.5 hours. She received standard antibiotic prophylaxis and postoperative care. Her recovery was uneventful and she was discharged after two days.

She returned on the seventh postoperative day with new onset atypical back pain without a clear dermatomal pattern. She complained of exquisite pain at the needle entry site with radiating to the
mid thoracic region contrary to downwards radiation without having any radicular involvement. She did not have any fever, headache, or signs of meningism apart from slight erythema at the needle entry site. Her C-reactive protein (CRP) and white blood cells (WBC) count remained normal. Her neurological examination was unremarkable.

She was treated conservatively with analgesic, antibiotic and physiotherapy. As the severe pain persisted interfering her childcare, a neurology consult was arranged, which did not reveal anything additional. A magnetic resonance imaging (MRI) scan revealed inflammation of the subcutaneous tissues overlying L1-5 vertebrae (Fig. 1). As there was no evidence of any drainable fluid collection or any hematoma, no further intervention was advocated other than continuing ongoing conservative management. Her pain reduced steadily, and she recovered fully after five days.

Infective complications after EA are reported due to the introduction of infection during the procedure, entry of microorganism through needle puncture site, or seeding of microorganisms from the blood after traumatic procedure [1]. Non-infectious complications neuraxis and outside neuraxis after EA are also reported [2,3]. Chlorhexidine used for skin asepsis reported to be cytotoxic and neurotoxic [4,5]. But chlorhexidine induced aseptic inflammation of subcutaneous tissue outside neuraxis presenting as clinical diagnostic dilemma after uneventful EA is not yet reported.

Atypical clinical presentation and the MRI report baffled us. Lack of signs of infection (fever, leukocytosis, raised CRP) or any drainable fluid collection went against diagnosis of any infection. Only positive finding was erythema in needle entry site and MRI picture suggestive of possible inflammation. After multidisciplinary discussions we suspected aseptic inflammation caused by inadvertent entry of cytotoxic chlorhexidine as most probable differential diagnosis. Possibly enough time was not given for complete drying of chlorhexidine before performing EA. This
probably has led entry of cytotoxic chlorhexidine leading to tissue inflammation. Our assumption was also proved by uneventful recovery of this complication using conservative management.

Non-infective subcutaneous inflammation outside neuraxis presenting as delayed onset atypical back pain is not yet reported. We wish therefore to make our colleagues aware of this atypical clinical presentation, as a potential differential diagnosis, and to reassure them of its uneventful resolution.
References


**Legend for figure**

**Fig. 1.** MRI Slice showing tissue inflammation in deep subcutaneous tissue superficial to the paraspinal musculature in the midline overlying L1-5 vertebrae. Arrow pointing to the area of inflammation.