

Obstetrical and Pediatric Anesthesia

Case report: Epidural abscess in a parturient with pruritic urticarial papules and plaques of pregnancy (PUPPP)

[Présentation de cas : un abcès péridural chez une parturiente qui présente des papules et des plaques prurigineuses urticariennes de la grossesse (PPPUG)]

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Purpose: To describe the risk factors for epidural abscess (EA) formation following epidural analgesia in a parturient with pruritic urticarial papules and plaques of pregnancy (PUPPP).

Clinical Features: A 33 yr-old gravida 2 nulliparous patient at 36 weeks gestation presented with severe pre-eclampsia, and PUPPP (treated with prednisone). Magnesium prophylaxis was started and labour was induced. An epidural catheter was placed at the L₃₋₄ level using standard aseptic technique. Bupivacaine was incrementally injected to achieve a T₁₀ sensory level, and analgesia was maintained using a continuous infusion of 0.0625% bupivacaine with fentanyl. Nine days post-delivery, the patient developed back pain radiating to her right leg, but she was otherwise asymptomatic. She was afebrile; with a slightly tender, non-erythematous, non-draining, 1 cm nodule at the epidural catheter site. Motor and sensory examinations were normal at that time. However, the patient returned 24 hr later and further investigations revealed: WBC 17,800·mm⁻³, platelets 486,000·mm⁻³, erythrocyte sedimentation rate 50 mm·hr⁻¹, and C-reactive protein 8.8 mg·dL⁻¹. The magnetic resonance imaging demonstrated an EA at the L₃₋₄ level causing minimal cord compression. The patient underwent an emergency decompressive laminectomy. Cultures revealed methicillin-sensitive *Staphylococcus aureus*. Her pain improved, and she was discharged on the third postoperative day with a six-week course of iv ceftriaxone.

Conclusion: Causative organisms for EAs include coagulase-negative *Staphylococci*, *S. aureus*, and Gram-negative bacilli. Infection can occur either hematogenously or by direct contamination during catheter placement. Risk factors include immunocompromised states and PUPPP, as with the case of this patient.

Objectif : Décrire les facteurs de risque d'abcès péridural (AP) à la suite d'une analgésie péridurale chez une parturiente qui présente des papules et des plaques prurigineuses urticariennes de la grossesse (PPPUG).

Éléments cliniques : Une femme de 33 ans, nullipare, secondigeste, a consulté pour une sévère pré-éclampsie et des PPPUG (traitées avec de la prednisone) à 36 semaines de grossesse. L'administration préventive de magnésium a été amorcée et le travail induit. Un cathéter péridural a été placé à L3-4 selon la technique aseptique standard. De la bupivacaine a été progressivement injectée pour atteindre le niveau sensoriel de T10, et l'analgésie maintenue avec une perfusion continue de bupivacaine à 0,0625 % avec du fentanyl. Neuf jours après l'accouchement, en l'absence de tout autre symptôme, une douleur dorsale s'est développée, irradiant vers la jambe droite. La patiente afebrile présentait, au site du cathéter péridural, un nodule légèrement douloureux de 1 cm, non érythémateux et non exsudatif. Les examens moteur et sensitif étaient alors normaux. La patiente est pourtant revenue 24 h plus tard et d'autres examens ont révélé : des globules blancs à 17 800·mm⁻³, des plaquettes à 486 000·mm⁻³, un taux de sédimentation érythrocytaire de 50 mm·hr⁻¹ et des protéines C-réactives à 8,8 mg·dL⁻¹. L'imagerie par résonance magnétique a démontré un AP à L₃₋₄ causant une compression médullaire minimale. La patiente a subi une laminectomie décompressive d'urgence. Les cultures ont révélé des *Staphylococcus aureus* sensibles à la méthicilline. La douleur ayant diminué, la patiente a pu quitter l'hôpital au troisième jour postopératoire avec du céftriaxone iv pour une cure de six semaines.

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Conclusion : Les pathogènes responsables des AP comprennent les *Staphylococci* négatifs quant à la coagulase, les *S. aureus* et les bacilles Gram négatif. Une infection peut se produire par voie hématogène ou par contamination directe pendant la mise en place du cathéter. Les facteurs de risque incluent les états d'immunodépression et les PPPUG, comme dans le cas de cette patiente.

EPIDURAL abscess (EA) is a rare but potentially devastating complication of neuraxial analgesia. The presentation may be indolent, and suspicion for the diagnosis must be maintained, especially in immunocompromised patients. We report a case of EA in a parturient treated with prednisone for pruritic urticarial papules and plaques of pregnancy (PUPPP), a common dermatosis of pregnancy. The patient whose case we discuss signed a standardized consent form allowing publication of information and images for research and educational purposes.

Case report

A 33-yr-old gravida 2 nulliparous patient presented at 36 weeks gestation with irregular contractions and severe pre-eclampsia. Pruritic urticarial papules and plaques of pregnancy had been diagnosed one week previously. She was started on magnesium prophylaxis, induced with misoprostol and oxytocin, and the anesthesia team was consulted for labour analgesia. She was also receiving a tapering dose of prednisone (60 mg daily at the time of admission). Past medical history included depression treated with sertraline.

Physical exam revealed an elevated blood pressure (160/90 mmHg), and a diffuse papular rash with several excoriated areas on the forearms. Her lower back, however, was free of lesions. Laboratory values revealed hemoconcentration and leukocytosis (Table). Uric acid was 6.6 mg·dL⁻¹; with transaminases and bilirubin values being normal. A 24-hr urine collection yielded 8 g protein.

An epidural catheter was easily placed at the L_{3,4} level following a standard aseptic technique including three sequential swabs impregnated with povidone-iodine. The iodine preparation was permitted to air-dry on the patient's back. Aspiration of the catheter and an epidural test dose (3 mL lidocaine 1.5% + epinephrine 1:200,000) were both negative for *in vivo* and intrathecal placement. Bupivacaine 0.25% (10 mL) with fentanyl 50 µg was injected incrementally, achieving good pain relief. An infusion of 0.0625%



FIGURE Sagittal T₂-weighted magnetic resonance imaging showing an epidural abscess at the L_{3,4} level.

bupivacaine with fentanyl 2 µg·mL⁻¹ and epinephrine 1.25 µg·mL⁻¹ was initiated. All epidural solutions (except for the test dose) were administered through an intact 0.2 micron bacterial filter. After an uneventful eight-hour labour, a healthy male infant was delivered. The epidural catheter was removed two hours postpartum.

One week after discharge, the patient experienced increasing back pain radiating to her right leg. She was afebrile, and otherwise asymptomatic. Physical examination revealed a slightly tender 1 cm nodule without erythema or drainage at the previous epidural catheter site. Motor and sensory examinations were normal, and the patient was advised to take analgesics and return if symptoms worsened. She returned within 24 hr. Examination at this time revealed decreased sensation to light touch over the right thigh in the L_{3,4} dermatome distribution. Relevant laboratory values included a white blood cell count of 17,800·mm⁻³, platelet count of 486,000·mm⁻³, C-reactive protein of 8.8 mg·dL⁻¹, and erythrocyte sedimentation rate = 50 mm·hr⁻¹ – all consistent with a pronounced inflammatory response. Blood cultures drawn at that time grew coagulase-negative *Staphylococcus* in one out of four bottles. The magnetic resonance imaging

TABLE Laboratory values

Laboratory variable (normal range)	First admission	Return to hospital (PPD#10)	Post-laminectomy (PPD#12)	6 Weeks postpartum
Hemoglobin (11.7–15.2 g·dL ⁻¹)	12.9	10.5	8.6	11.1
WBC (3.6–10.5 x1000/mm ³)	13.8	17.8	11.4	4.7
% Neutrophils (45–70%)		69	69	44
Platelets (140–400 x 1000·mm ⁻³)	281	486	460	304
ESR (0–20 mm·hr ⁻¹)		50	9	9
CRP (0–0.5 mg·dL ⁻¹)		8.8		0.12
BUN (7–18 mg·dL ⁻¹)	18	9		12
Creatinine (0.6–1.0 mg·dL ⁻¹)	1.1	0.8		0.8

WBC = white blood cell count; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein; BUN = blood urea nitrogen; PPD = post-partum day.

(MRI) demonstrated a collection of fluid posterior to the spinal canal at the L₃₋₄ level causing minimal cord compression (Figure). The patient was re-admitted and, after consultation with the on-call neurosurgeon, underwent an emergency decompressive laminectomy. Wound cultures revealed methicillin-sensitive *Staphylococcus aureus*. The patient's pain improved and she was discharged on the third post-operative day with a six-week course of *iv* ceftriaxone. The patient remained well, and by six weeks post-partum, her laboratory values showed no further evidence of infection or inflammation (Table).

Discussion

Although rare, EAs can be devastating. Their incidence is approximately 1:2,000 epidural catheter placements.^{1,2} Patients can present with pain, fever, headache, or neck stiffness, typically four to ten days after catheter removal. Evaluation should focus on generalized signs of infection (fever, leukocytosis), a detailed neurological examination, and inspection of the catheter site for erythema, tenderness, swelling, or drainage. Blood cultures may or may not be helpful in diagnosis, as they only provide a "snapshot" of whether a patient is bacteremic at a specific point in time. Radicular pain or neurological deficits can indicate cord or nerve root compression. Early diagnosis (MRI is the gold standard), surgical decompression, and antibiotic therapy are the keys to management, although conservative therapy has been reported.^{3,4} In general, however, conservative therapy is the exception rather than the rule. The "gold standard" treatment of epidural abscesses remains surgical evacuation of infected tissue.

An abscess will usually extend three to five segments, but can be more extensive. The most common organisms are coagulase-negative *Staphylococci*, *S.*

aureus, and Gram-negative bacilli.² The mechanism of infection can be either hematogenous seeding or direct contamination from catheter placement. The risk is higher with longer (> three days) duration, diabetes or other immunocompromised states, and low molecular weight heparin prophylaxis.¹ Additionally, it has been suggested that skin abnormalities may also increase the risk of EA.⁵

Pruritic urticarial papules and plaques of pregnancy is an ill-defined cutaneous eruption of pruritic lesions, most commonly occurring in primigravidas in the third trimester.⁶ It is the most common dermatosis of pregnancy occurring in about 0.5% of pregnancies.⁷ The lesions are typically found on the lower abdomen and proximal extremities, and typically spare the face, palms, and soles of affected individuals.⁶ Treatment is symptomatic, with topical corticosteroids and diphenhydramine as first-line therapy.⁸ Systemic corticosteroids are typically used in refractory cases.⁷ In this case, the patient was in great distress and steroids were started immediately after diagnosis.

Clearly, this patient's EA most likely occurred as a result of labour epidural placement, since both the catheter placement and the EA occurred at the L₃₋₄ level. Whether her infection resulted from direct seeding or hematogenous spread is unknown. Of course, even with strict aseptic technique, some amount of contamination with skin flora may be introduced with the epidural needle.⁹ However, as she had a diffusely pruritic rash with several excoriated areas, it is also reasonable to hypothesize that scratching could have resulted in colonization of lesions and transient bacteremia. Microtrauma to epidural vessels occurring with epidural placement or removal during a bacteremic period could then result in seeding of the epidural space. Whether such patients should receive antibiotic prophylaxis or even be denied epidural analgesia is

unclear. Clinical judgment should be individualized to each circumstance. In either case, this patient's immunosuppression from steroids placed her at higher risk for development of an epidural abscess and likely masked the resulting inflammatory reaction (e.g., lack of fever or erythema). In the face of immunosuppressive drugs, it is especially critical that the clinician be suspicious of new back or radicular pain.

Another issue raised by this case is how to minimize the risk of contamination or colonization of the catheter, epidural skin site, or epidural space through either chemical or physical means. One method is to optimize the choice of skin preparation solution. Traditional practice dictates the use of an iodophor such as povidone-iodine. While it is an effective antimicrobial agent, proper use requires allowing sufficient time for the solution to dry. In recent years, chlorhexidine gluconate (CHG) emerged as the skin preparation of choice for central venous access, given a number of studies showing lower rates of catheter-associated bloodstream infection.¹⁰ Such a change has not occurred with epidural catheter insertion. The package insert for ChlorPrep® (2% CHG in 70% isopropyl alcohol, Medi-Flex, Inc., Leawood, KS, USA) specifically cautions against use of the product for lumbar puncture, due to a lack of supporting studies at the time of FDA approval (personal communication, Medi-Flex, Inc.). More recent studies have either shown a reduction¹¹ or no change¹² in the rate of epidural catheter colonization, although the studies used 0.5% chlorhexidine *vs* the 2% formulation which is marketed currently. This concentration difference may prove relevant to both concerns of efficacy and possible toxicity of CHG. There are only limited clinical data regarding the neurotoxicity of CHG. Two animal studies demonstrated both ototoxicity¹³ and ocular toxicity¹⁴ when applied directly to the target organs. No study, however, has addressed the potential toxicity of CHG used for skin preparation prior to neuraxial procedures. Another skin disinfectant studied for this purpose is DuraPrep® (3M Health Care, St. Paul, MN, USA), a solution of an iodophor in 74% isopropyl alcohol. A recent study¹⁵ demonstrated a lower number of positive skin cultures both immediately and at catheter removal when using DuraPrep, suggesting both increased initial bactericidal activity and prolonged action. Taken together, the above results indicate that plain povidone-iodine may not be the best antiseptic for neuraxial procedures.

In addition, the use of barriers for asepsis is equally important. In North America, the generally accepted practice includes wearing a surgical cap, mask, and sterile gloves. In the United Kingdom, however, it

is common to also wear a sterile surgical gown. This potentially would avoid the risk of the catheter brushing against the nonsterile arm of the anesthesiologist. Finally, use of bacterial filters for both bolus dosing and maintenance infusions may prevent seeding of the catheter and/or epidural space with pathogenic organisms arising from contaminated syringes or solutions.¹⁶

In conclusion, a parturient developed an EA despite strict adherence to a standard protocol involving appropriate skin preparation, accepted aseptic technique (mask, cap, sterile gloves), and use of a bacterial filter. The placement of the epidural was notably atraumatic. As this was the first EA experienced in a large volume practice (3,000–4,000 deliveries per year), it is our view that patient factors rather than performance factors best explain this occurrence. The predisposing patient factors included the relative immunosuppression induced by steroid therapy, and the extensive excoriations secondary to scratching of her pruritic urticarial papules and plaques of pregnancy. Microtrauma to epidural vessels during either catheter insertion or removal likely resulted in seeding of the epidural space by a common skin pathogen (*S. aureus*) previously introduced into the circulation through the excoriations. Consequently, donning a sterile gown would have not provided additional protection for this patient, although it certainly would not have been an unreasonable thing to do. Prophylactic antibiotics to cover skin flora (e.g., cefazolin) could have been considered, but would have exposed the fetus to antibiotics potentially complicating any neonatal sepsis evaluation. Steroid-induced immunosuppression was a likely contributing factor in the development of a full-blown infection and delayed its recognition. In immunocompromised patients, therefore, a meticulous sterile technique should be used and a high index of suspicion is warranted, as classic signs and symptoms of an epidural abscess may be masked.

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