Postpartum complications following neuraxial anaesthesia for obstetric physicians

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Abstract

Neuraxial analgesia and anaesthesia are widely accepted and well-tolerated modes of delivery analgesia, being employed in up to 76% of vaginal deliveries and 94% of caesarean deliveries in the United States.[1] A cause of considerable concern for postpartum women, their family and caring health professionals is the occurrence of unexplained postpartum complications, not only for management in the index pregnancy, but the uncertain risk of recurrence in future pregnancies. Complications of neuraxial blocks may impact significantly on the ability of mothers to care for and bond with their newborn. The reported incidence of temporary neurological deficit following obstetric neuraxial blocks is 1 in 3900 procedures, and the risk of permanent neurological harm estimated to be between 1 in 80,000 and 1 in 320,425 procedures.[2] Obstetric physicians may be asked to review women with postpartum complications following neuraxial blocks. This article reviews complications that may be seen following neuraxial blocks for delivery.

Introduction

Analgesia is a pain-free state, whereas anaesthesia is a state achieved when there is a loss of touch, pain, and temperature sensations with or without loss of consciousness. Epidural blockade involves insertion of a needle into the epidural space, located outside the spinal cord and spinal fluid. (Figure 1) Local anaesthetic, often combined with other medications, may be injected directly via the needle or as boluses or a continuous infusion via a catheter inserted through the needle. Epidural needles are typically 17–18 gauge. Spinal blocks almost always involve a single injection through a fine needle into the cerebrospinal fluid. (Figure 2) Spinal needles are typically 25–27 gauge with a pencil-point tip, much finer than epidural needles. Spinal blocks may be combined with an epidural by inserting a long fine needle through the epidural needle into the cerebrospinal fluid before insertion of the epidural catheter.

CSF pressure is unchanged during healthy pregnancy, with preeclampsia, chronic hypertension or with idiopathic seizures in pregnancy.[3] Uterine contractions alone do not cause increased CSF pressure on continuous monitoring during labour, however, Valsalva manoeuvres or pushing with contractions cause CSF pressure to increase to an average of 71cm H_2O.[4,5] Transient increases in intracranial pressure (ICP) with epidural injection depend on the basal ICP and the rate and volume injected.[6]

Leak of cerebrospinal fluid (CSF) following dural puncture leading to intracranial hypotension is the predominant mechanism by which postpartum complications may occur following neuraxial blocks. Inadvertent dural puncture occurs in 0%–2.6% of epidural blocks, with this being unrecognised at the time of the procedure in 16%–38% of cases.[7] Post-dural puncture headache (PDPH) complicates 51%–85% of episodes of dural puncture with epidural blocks. The risk of PDPH following spinal anaesthesia is lower at 1%–10% due to the much finer needle.[8–11] PDPH is associated with a statistically increased risk of subdural haematoma, cranial nerve palsy, meningitis, cerebral vein sinus thrombosis, depression, headache and persistent low back

pain compared with women without PDPH.[10] A challenge for the obstetric physician is in deciding when to investigate women with complications following neuraxial anaesthesia. Numerous rarer complications have also been reported following neuraxial blocks which physicians should be aware of, despite their reassuringly low incidence (Table 1).

Cranial

Post-dural puncture headache (PDPH)

The International Headache Society defines PDPH as headache onset within 5 days of dural puncture, usually associated with neck stiffness and subjective hearing symptoms.[12] PDPH is classically positional, appearing or becoming aggravated within 15 min after sitting or standing, and improving within 15 min of lying down, however, 5.6% of presentations with PDPH may be non-positional.[13] Hearing loss, photophobia and vertigo may occur.[8] The mechanism of PDPH is thought to be reduced ICP, compensatory meningeal venous dilation, downward

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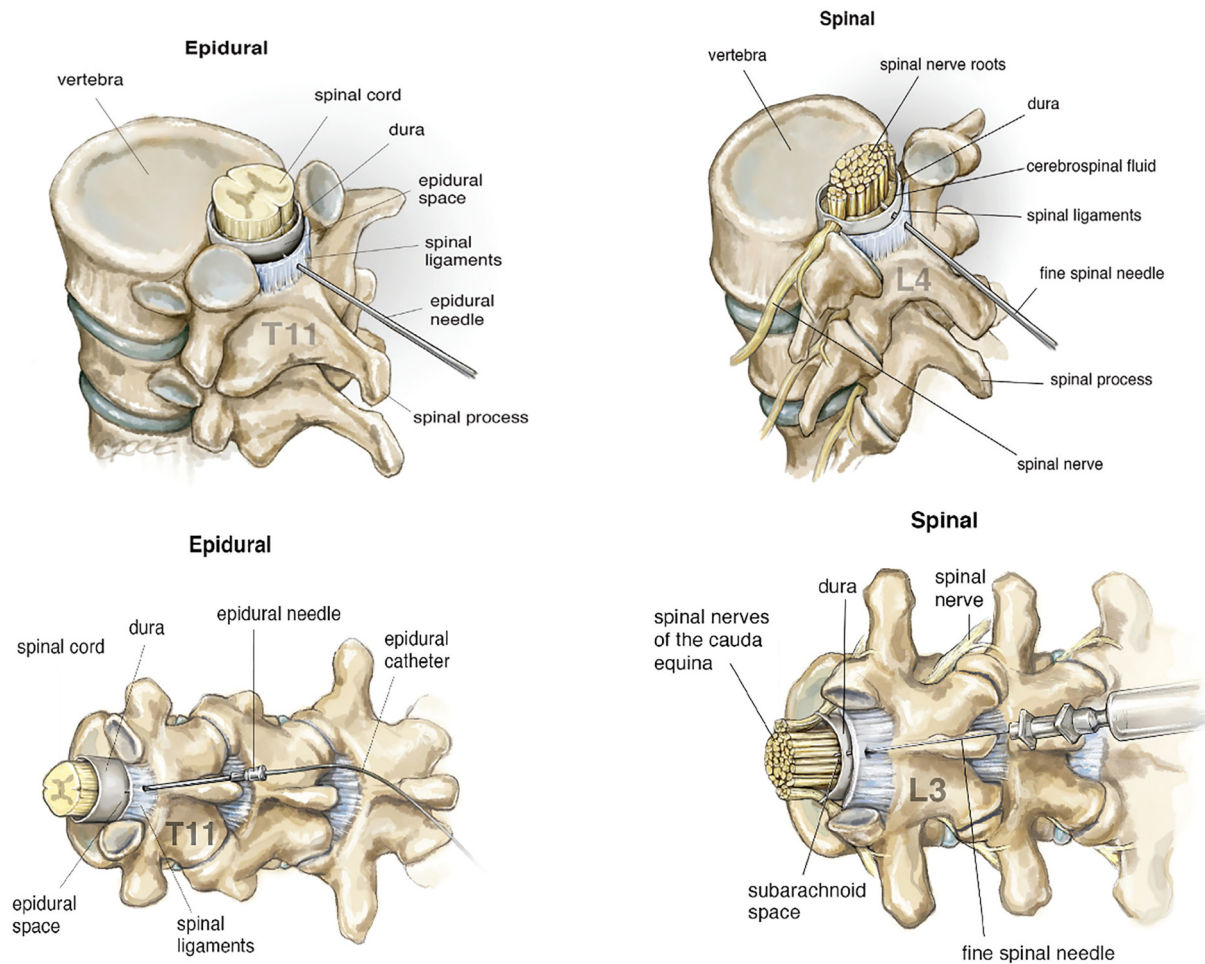Email: adam.morton@mater.org.au

Epidural

vertebra
spinal cord
dura
epidural space
spinal ligaments
epidural needle
T11
spinal process

Spinal

vertebra
spinal nerve roots
dura
cerebrospinal fluid
spinal ligaments
fine spinal needle
L4
spinal process
spinal nerve

Epidural

spinal cord
dura
epidural needle
epidural catheter
epidural space
spinal ligaments
T11

Spinal

dura
spinal nerve
spinal nerves of the cauda equina
subarachnoid space
L3
fine spinal needle

Figure 1. Anatomy of epidural and spinal blocks.

displacement of the brain and alterations in craniospinal compliance.[14] Accordingly, radiological features of intracranial hypotension following dural puncture include venous sinus distension, diffuse pachymeningeal enhancement, pituitary enlargement, transtentorial brain sagging and subdural fluid collection.[15] Cranial nerve abnormalities may occur due to mechanical traction on the cranial nerves when in an upright position.[15]

PDPH is uncommon, complicating 0.4%–1.6% of epidural and combined spinal-epidural blocks, and less than 0.5% of cases of spinal anaesthesia.[1,10,16]

Development of headache within 24 h of delivery and lack of side predominance differentiates PDPH from migraine.[17] Characteristics suggesting the need for investigation of secondary causes of headache include associated neurological symptoms or signs, headache peaking in severity in less than 5 min, fever, precipitation of headache by physical activity or Valsalva, headache waking the patient from sleep, change in the type or pattern of previous headache (e.g., postural to non-postural), and failure of headache to resolve within 2 weeks.[18,19] In a study of 95 women with postpartum headache, cerebral imaging in 22 women whose headache did not resolve or who had additional neurological symptoms or signs revealed abnormal findings in 15 cases (68%).[18]

Conservative treatment may be considered where the headache is not severe, with the natural course being resolution in 2 weeks. Regular simple oral analgesia should be offered, with the short-term use of opioid analgesia considered if simple oral analgesia is ineffective. Prolonged bed rest is not recommended because of the risk of thromboembolism. There is no evidence for excessive fluid administration and abdominal binders, and insufficient evidence to recommend the use of theophylline, ACTH, glucocorticoids, triptans, gabapentinoids, acupuncture, greater occipital nerve, or sphenopalatine ganglion blocks. There is limited evidence to support the use of caffeine in conservative management, and this should be limited to 24 h, with a maximal dose of 900 mg during this time. The mechanism of action is thought to be cerebral vasoconstriction and increased cerebrospinal fluid production.[20] Epidural blood patch is the "gold standard" treatment, with greater resolution when this is delayed for 24–48 h after dural puncture.[21]

Intracranial subdural haemorrhage

Intracranial subdural haematoma (ISDH) following neuraxial anaesthesia is thought to occur due to intracranial hypotension following dural puncture as previously described, with subsequent tearing of bridging veins. Accordingly, the incidence of ISDH is higher with recognised accidental dural puncture (110 compared with 26 per 100,000 epidural blocks), especially if PDPH occurs (83–147 per 100,000).[22,23] The adjusted odds ratio of subdural haemorrhage following neuraxial block with PDPH is 76.7 compared with all neuraxial blocks.[10] The incidence of ISDH with spinal anaesthesia has been estimated to be 1 in 500,000 procedures.[24] However,
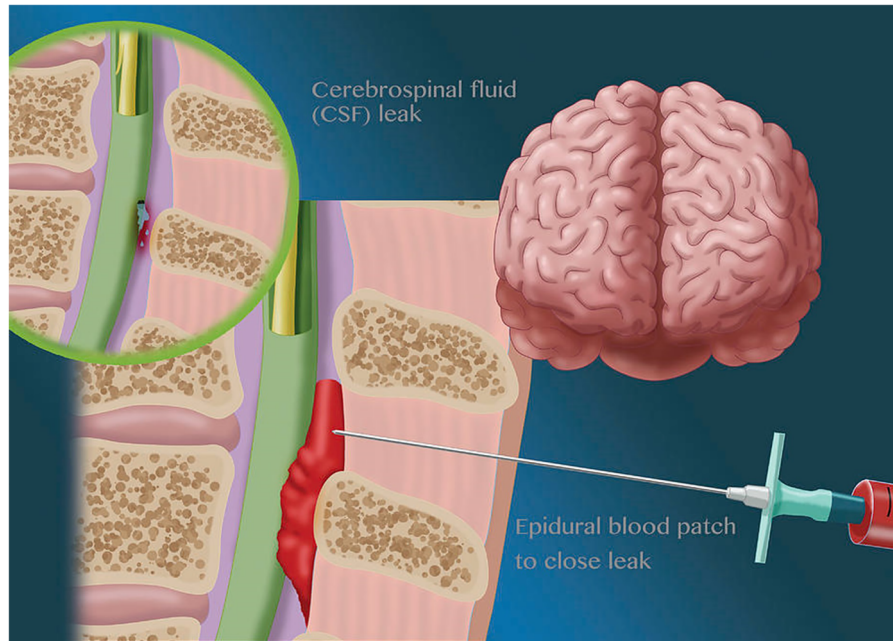
Figure 2. Epidural blood patch.

Table 1. Complications following neuraxial analgesia and anaesthesia.

Cranial
  Post-dural puncture headache
  Haemorrhage
    Intracranial subdural haemorrhage
    Subarachnoid haemorrhage
    Pituitary haemorrhage
  Cerebral venous sinus thrombosis
  Posterior reversible encephalopathy syndrome
  Horner's syndrome
  Cranial nerve palsies
  Auditory loss
  Meningitis
  Aseptic meningitis
  Tonic-clonic seizures
  Pneumocephalus
Spinal
  Spinal or epidural haematoma
  Spinal epidural abscess
  Anterior spinal artery syndrome
  Epidural blood patch
  Spinal myoclonus
Other
  Fever
  Arrhythmias and cardiac arrest
  Transient neurologic symptoms
  Nerve irritation – direct needle or catheter trauma
  Necrotising fasciitis

incidence of ISDH is likely underestimated, with many patients treated as for PDPH alone without imaging and eventual resolution of symptoms.[25] Complication of PDPH by ISDH can manifest as persistent headache,

failure to respond to conservative therapies and change from postural to non-postural nature, with non-postural headache present in 74%–91%.[19,22,26] Other symptoms of ISDH include nausea/vomiting (31%–41%), altered mental status (31%–40%), focal motor deficits (23%–28%), visual changes (14%–20%) and aphasia/dysarthria (11%–13%).[22]

The duration from dural puncture to the time of diagnosis of ISDH ranges widely from 4 h to 29 weeks, with most readmissions for ISDH occurring within 5 days of discharge.[27] Post-dural puncture ISDH is associated with mortality of 7%–10% and a 10% risk of permanent neurological injury.[25,26] Diagnosis is with computerised axial tomography (CT) or magnetic resonance imaging (MRI). Management generally involves surgical drainage, which occurred in 16 of 24 ISDH following neuraxial block for delivery reported by Zeidan et al.,[19] or conservative management if the haematoma is small and without midline shift or neurological deficit. If conservative management has undertaken the risk of haematoma enlargement and neurologic deterioration is highest in the first 36 h after ISDH onset. Repeat CT/MRI should be performed within 6–8 h of the initial scan in patient managed non-operatively, and neurological examinations performed every 1–2 h for the first 24 h after presentation.

Other intracerebral haemorrhage

Four cases of subarachnoid haemorrhage (SAH) and two cases of intracerebral haemorrhage have been reported following neuraxial anaesthesia in the absence of an alternative precipitating cause.[28–33] The risk of SAH in the postpartum period has been estimated to be 5.8 cases per 100,000 deliveries.[34]

Three cases of pituitary haemorrhage have been described following spinal anaesthesia in the absence of an alternative precipitating cause, presenting between five minutes and one week following delivery.[35–37] The mechanism and significance of contribution by neuraxial anaesthesia are unclear, however, pituitary apoplexy after spinal anaesthesia has been described outside pregnancy, possibly precipitated by intraoperative

hypotension causing ischaemic necrosis in a region susceptible to changes in mean arterial pressure, especially where an a pituitary tumour is present.[38]

Cerebral vein sinus thrombosis

The estimated peripartum and postpartum incidence of cerebral vein sinus thrombosis (CVT) is 9–12 per 100,000 births.[10] With neuraxial anaesthesia the incidence is 15.2 per 100,000 births, and 166 per 100,000 births in the setting of PDPH (aOR 11.48).[10] In a review of 58 case reports of CVT following neuraxial anaesthesia, 46 women (79%) experienced a PDPH.[39] Twelve deaths from CVT were reported in the United Kingdom over the 15 years between 2006 and 2021.[39] Several mechanisms have been postulated for CVT following dural puncture. The reduction in pressure and volume of CSF result in compensatory cerebral venous and arterial dilation, reducing the velocity of blood flow in the straight sinus by 50%, increasing blood viscosity.[40] Traction on cerebral vessels by a negative spinal-cranial gradient increases the predisposition of the venous epithelium to damage. Combined with the physiological hypercoagulability of pregnancy these factors predispose to thrombosis.

The median time from delivery to the diagnosis of CVT is 6.5 days, with 72% of women presenting within 2 weeks of delivery.[39] Headache occurs in 98% of women with CVT.[39] CVT-related headache is variable with no specific characteristics, and is difficult to differentiate from PDPH. Onset may be subacute or thunderclap, throbbing, migraine-like or constant. It may be localised or generalised. Headache with CVT has been described to lack a postural element; however, a review of cases reported a postural element in 72% of cases following neuraxial anaesthesia.[41] A change in the character of headache accompanied by the loss of a postural element, headache becoming constant, and failure of epidural blood patch to provide relief should prompt consideration of CVT. Other features of CVT may include seizures (45%) visual disturbance, papilloedema, fever, motor deficit, speech disturbance, cranial nerve signs, and coma.[39] Additional risk factors include thrombophilia (24%) and pre-eclampsia (17%).[39] Complications reported with CVT included intracranial venous infarction (26%), subarachnoid haemorrhage (10%), subdural haematoma (5%) and other intracranial haemorrhages (5%).[39] Diagnosis is by MRI venography, or CT venography if MRI is not available. Treatment is with low molecular weight heparin (LMWH), or intravenous unfractionated heparin if invasive procedures are planned or LMWH is contraindicated.

Posterior reversible encephalopathy and reversible cerebral vasoconstriction syndromes

Posterior reversible encephalopathy syndrome (PRES) is a clinical disorder which may present with headache, tonic-clonic seizures, visual disturbance (hemianopia, cortical blindness, visual neglect), confusion or decreased level of consciousness, and motor and/or sensory loss, accompanied by characteristic neuroimaging findings of subcortical white matter oedema typically affecting the parieto-occipital lobes. Eighteen cases have been described postpartum in the setting of PDPH, ten following epidural blocks, six following spinal anaesthesia, and two following CSE.[42] Initial presentation was with typical PDPH, with the subsequent development of seizures (83%), visual changes (50%), altered conscious state (28%) and focal weakness/sensory loss (17%) 2–7 days later. Diagnosis is by MRI. Management consists of control of hypertension, and magnesium sulphate if seizures occur. Three of the 14 cases of PRES also demonstrated features of diffuse arterial vasospasm on imaging suggestive of reversible cerebral vasoconstriction syndrome. All of the women made a full recovery with no residual neurological deficit.

Horner's syndrome

Eighty-three cases of Horner's syndrome have been reported following obstetric neuraxial blockade.[43] The mode of anaesthesia employed was epidural blockade in 68 cases (82%), caudal labour analgesia in 11 cases (13%), spinal anaesthesia in 3 cases (3.6%) and 1 case (1.2%) following combined spinal-epidural.[43] In the vast majority of cases Horner's syndrome was unilateral. Associated trigeminal nerve palsy occurred in 9 cases and hypoglossal nerve palsy in 1 case. Other associated features included hypotension, blurred vision, neck pain and sensorimotor upper limb involvement. Ptosis and miosis were noted in 94% of women and anhidrosis in 31%. Horner syndrome occurred within 1 h of the local anaesthetic bolus in 74%, and the median time for resolution of Horner's syndrome was 2 h. One case of permanent Horner's syndrome was reported thought secondary to neuraxial anaesthesia.[44] The mechanism is thought to be blockade of the stellate ganglion sympathetic fibres due to unexpected migration of local anaesthetic. An important aetiology to exclude in women with postpartum Horner's syndrome is spontaneous carotid artery dissection (SCAD). Horner's syndrome occurs in 25% of individuals with SCAD. Features suggestive of SCAD include severe headache (78%) ipsilateral to the Horner's syndrome, pulsatile tinnitus, symptoms or signs of anterior circulation stroke, and delayed presentation with onset a mean time of onset 11days (range 0–53 days) following delivery.[45] SCAD may be diagnosed using MR or CT angiography.

Cranial nerve palsies

A review of 43 cases of cranial nerve palsies (CNP) following obstetric neuraxial block showed that the most common nerves involved were abducens (17), followed by facial (12), vestibulocochlear (10) and trigeminal (7).[46] Twenty-seven cases (63%) were preceded by PDPH, whereas subdural haematoma and transverse sinus thrombosis were more rarely associated, with 6 and 1 cases respectively. Complete resolution of CNP occurred in 35 patients (81%), ranging from weeks to months after presentation. A case report described resolution of trigeminal nerve palsy associated with PDPH following epidural blood patch (EBP).[47] Not all PDPH CNP cases improve following EBP; some persist despite EBP or occur following EBP.[46]

Auditory loss

Although transient clinically significant hearing loss has been reported by 0.2%–0.8% of non-pregnant individuals following spinal anaesthesia, and audiometrically-measurable hearing loss has been found in 10%–50% of individuals after dural puncture, these events appear to be rare in obstetric populations.[48] In two studies totalling 81 women who received neuraxial anaesthesia for elective caesarean, none developed hearing loss on audiometry postpartum.[49,50] Sudden sensorineural hearing loss post-neuraxial anaesthesia is thought to occur during due to the transmission of decreased CSF pressure to the cochlear perilymph. A case report described complete resolution of spinal anaesthesia-associated hearing loss following EBP, congruent with the common mechanism of decreased CSF pressure.[51]

Meningitis

The incidence of meningitis following neuraxial anaesthesia in pregnancy has been reported to be 2.5 per 100,000 procedures.[10] Where PDPH occurs the rate of meningitis is 83 per 100,000 (aOR 39.7).[10] A retrospective study found the risk was lower after epidural blocks than after spinal anaesthesia.[52] *Streptococcus viridans* is the most common pathogen in bacterial meningitis post-dural puncture, accounting for 43%–60% of cases.[53]

Other organisms include gram negatives (*Pseudomonas*, *Escherichia coli*, *Neisseria meningitidis*) and *Staphylococci*. In pregnant women with meningitis unrelated to neuraxial anaesthesia the most common organisms are *Streptococcus pneumoniae* and *Listeria monocytogenes*.[54] Droplet contamination or needle contamination from incompletely sterilised skin are the major routes of infection. Headache is universally present, fever occurs in approximately 70% of cases, with other symptoms including vomiting, confusion and urinary retention.[55] The triad of severe headache, high fever and nuchal rigidity occurred in only 48% of cases.[55] Latency from neuraxial procedure to onset of symptoms ranged from 8 h to 8 days, with a median of 24 h. The exception is *Aspergillus* meningitis which may take weeks for symptoms to occur. Diagnosis is by assessment of CSF obtained by lumbar puncture. A CT should be performed prior to lumbar puncture in the setting of new-onset seizure, papilloedema, altered level of consciousness or focal neurological deficit. Antimicrobial therapy, with dexamethasone if indicated, should not be delayed if imaging is performed prior to lumbar puncture. Empiric antimicrobial therapy should consist of vancomycin combined with ceftazidime, cefipime or meropenem.

Aseptic meningitis

Aseptic meningitis is uncommon with neuraxial anaesthesia. Neurotoxicity from unintentional injection of an irritant or inflammatory response to medication in the intrathecal space may result in fever, headache, neck stiffness, photophobia and low back pain, typically onset within 24 h of spinal anaesthesia, with apyrexia, high CSF protein, normal CSF glucose, elevated CSF polymorphs, absent of organisms on CSF gram stain and full recovery within 48 h.[56,57]

Tonic-clonic seizures

Fourteen case reports have described otherwise unexplained tonic-clonic seizures in women with PDPH.[58,59] Shearer et al. described eight cases where the seizure was preceded by visual disturbance including cortical blindness in three women.[58] Seizures occurred between 2 and 7 days following dural puncture. Imaging revealed changes of intracranial hypotension with diffuse dural thickening and enhancement, venous sinus dilatation, brainstem slumping, reversible pituitary enlargement, and changes in cerebral vasospasm.

Pneumocephalus

Pneumocephalus may occur as the result of unintentional injection of air into the subarachnoid or subdural space. Symptoms include headache, seizures, focal neurologic deficits and neck stiffness.[60]

Spinal

Spinal or epidural haematoma

The estimated incidence of spinal epidural haematoma (SEH) is 0.55–5.5 per 100,000 epidural catheterisations, and 1 per 220,000 spinal anaesthetics.[14,61,62] Puncture of vessels in the venous plexus and resulting accumulation of blood may result in ischaemia/infarction of the spinal cord or cauda equina.[14] The epidural venous plexus lacks valves and is susceptible to changes in intra-abdominal and intrathoracic pressure. Due to the anatomical location of neuraxial block, 95% of haematomas involve the dorsal spinal canal.[63,64] Haematoma may be asymptomatic for some time. Symptoms suggestive of SEH include bladder or bowel dysfunction, progressive motor and/or sensory block and back pain, though the last may occur in only 25% of cases. Initial symptoms occur during puncture in 7%, during anaesthesia with an indwelling epidural catheter in 36%, and after catheter removal in 56%.[65] Symptom onset may post-date catheter removal by several days.[66,67] Early recognition of haematoma formation and neurological sequelae is critical as immediate surgical decompression is required, optimal neurologic outcomes occurring with surgery within 12 h of initial symptoms.[68] Emergency MRI (or CT if MRI is not available/contraindicated) should be performed if SEH is considered. Nonoperative management can be successful in the absence of significant neurology.[69] Additional risk factors for spinal haematoma include coagulopathy, thrombocytopenia and vascular and spinal malformations.[70] Interestingly, spontaneous spinal epidural haematoma unrelated to or distant from the site of neuraxial anaesthesia have been reported in second and third trimester, usually involving the cervicothoracic spine, highlighting that increased pressure in the venous plexus can cause spontaneous vascular rupture.[67,71]

Spinal epidural abscess

The incidence of spinal epidural abscess (SEA) has been reported to be 0–1.6 per 100,000 epidural catheterizations.[52,61,62] Women with SEA typically present with fever (74%), severe localised back pain (70–100%), with or without symptoms and signs of cord and nerve root involvement (50%). Spinal tenderness is present in 17%–98% of cases. The median time from procedure to onset of symptoms of epidural abscess is 12 days in the non-obstetric population.[72] The risk is higher with epidural catheter placement. C-reactive protein is typically elevated. MRI with gadolinium is the imaging modality of choice. Staphylococci are the most common causative organism, followed by gram-negative bacilli, streptococci, and more rarely, anaerobes and mycobacteria.[73] The causative organism may be obtained from the abscess or culture of blood and CSF. Management usually involves a combination of surgical drainage and systemic antibiotic therapy. Antibiotics should be commenced as soon as the diagnosis of SEA is suspected and blood cultures have been collected. Empiric therapy should consist of vancomycin with a third-generation cephalosporin or meropenem.

Anterior spinal artery syndrome

Five cases of paraplegia due to anterior spinal artery syndrome (ASAS) have been described following neuraxial anaesthesia.[74] ASAS following neuraxial block presents as failure to regain motor function and pain and temperature sensation below the level of the lesion, with sparing of proprioception and vibration. Autonomic neuropathy with sphincter dysfunction may be present. Possible factors related to delivery include peripartum hypotension, sympathectomy and vasospasm due to anaesthetic agents, the prothrombotic state of pregnancy, spinal vascular malformations, thoracoabdominal aortic disease, uterine-mediated compression of the spinal vasculature, increased CSF pressure and venous obstruction.[74] Thoracolumbar magnetic resonance imaging may show hyperintense lesions due to anterior spinal ischaemia, however, may not identify pathology.[74]

Epidural blood patch (EBP) complications

EBP remains the gold standard therapy for PDPH, with complete and permanent resolution of headache in up to third of cases of PDPH in with a single epidural blood patch, and partial relief in 50%–80%.[75] Smaller Australian prospective studies report similar success, with complete relief in 50%, partial in 38% and no relief in only 12%.[76] EBP can also prevent development of PDPH after an accidental dural puncture, although when used to treat PDPH it is best performed 48 h after headache onset.[21,77] There is insufficient evidence as to whether EBP reduces the risk of subdural haematoma or cerebral

vein sinus thrombosis, or improves CNP in women with obstetric PDPH. EBP should not be performed if the woman is febrile or there are any signs suggestive of systemic infection. EBP requires two anaesthetists both employing a fully aseptic technique. Twenty millilitres of blood should be drawn from an arm vein by the second anaesthetist and injected slowly into the epidural space at the site of the previous epidural or at one space higher or lower by the first anaesthetist Complications of EBP include further dural puncture (~1:100), failure, back pain, nerve injury, meningitis, abscess, seizures, ISDH, CNP, tinnitus and arachnoiditis.[78]

Spinal myoclonus

Six cases of spinal myoclonus have been reported following neuraxial anaesthesia in pregnancy.[79–82] The disorder is characterised by the onset of involuntary myoclonic movements of the upper or lower limbs or trunk within 4 h of the administration of anaesthetic. Symptoms typically resolved within 2 days, though one case persisted until 6 months postpartum.[79]

Other

Fever

A meta-analysis of 39 studies found that pregnant women managed with epidural blocks had an odds ratio of 5.26 of developing intrapartum hyperthermia.[83] Subgroup analysis of the 13 randomised control trials found this association persisted with an odds ratio of 4.21.[83] Similarly, an earlier meta-analysis of 12 RCTs reported a risk ratio for fever after epidural anaesthesia of 3.54.[84] The observed maternal temperature increase during labour averages 0.1°C per hour of epidural analgesia, following a lag of 4 to 5 h.[85] Approximately 20% of women labouring with neuraxial anaesthesia will experience temperature above 38°C. A previous systematic review concluded it was unlikely that epidural anaesthesia increased the risk of intrapartum infection.[84] The most likely explanation for epidural hyperthermia is that it represents a distinct condition from immunomodulation and/or cholinergic sympathetic blockade.[83] A study of the Swedish Birth Registry suggested that fever associated with epidural analgesia was a benign rise in temperature not associated with adverse neonatal neurologic outcomes.[86] Assessment of peripartum fever is complicated by the physiological rise in white blood cell count and erythrocyte sedimentation rate during pregnancy. C-reactive protein levels are unchanged during pregnancy though may rise up to fourfold in the first 48 h postpartum.[87] It is, however, critical, that a fever in labour is not attributed to neuraxial anaesthesia without thorough history, examination and investigations to exclude an infective or other pathological cause.

Arrhythmias and cardiac arrest

Spinal anaesthesia may be associated with atrioventricular (AV) block in pregnant women during CS, as well as in nonpregnant individuals. A prospective study of 254 healthy women with neuraxial anaesthesia for CS found first-degree atrioventricular (AV) block in 3.5%, second-degree AV block in 3.5%, and severe bradycardia (less than 50 bpm) in 6.7% of women, with no reported adverse maternal outcome.[88] Two cases of third-degree AV block have been described soon after induction of spinal anaesthesia, both responding to atropine.[89,90] Ventricular arrhythmias are also rarely observed, with one case of ventricular fibrillation and cardiac arrest described post-CS confounded by hypomagnesaemia.[91] Severe sinus bradycardia followed by asystole has also been described during spinal anaesthesia for CS.[92,93] This may be related to overlapping

phenomena of a vasovagal response or the Bezold–Jarisch reflex, a cardioinhibitory reflex whereby poor ventricular filling causes reflex bradycardia, vasodilation and hypotension.[93]

Transient neurologic symptoms

Transient neurologic symptoms present as pain and/or dysesthesia which radiates from the gluteal region to the posterior thigh within 2–24 h of spinal anaesthesia.[94] Ambulation and the administration of nonsteroidal anti-inflammatory medications may provide relief. Symptoms usually resolve spontaneously within 2–3 days. The pathogenesis is not understood. The presence of sensory or motor signs on physical examination may be useful to guide whether electromyographic (EMG) testing and MRI should be performed.

Direct nerve injury

The reported incidence of postpartum lumbosacral plexus and peripheral nerve injury following obstetric neuraxial blocks ranges between 0.1% and 9.2% depending on studies and methodology.[95] One study of nerve injury following obstetric neuraxial blocks attributed 80% of nerve injuries to intrinsic obstetric palsy and 20% to anaesthetic causation.[95] Eighty-four percent of postpartum neuropathy occurs in the femoral nerve distribution.[96] Nerve injury to the spinal cord, conus medullaris and spinal nerves may occur due to direct needle or catheter trauma, and is typically associated with acute pain at the time of needle insertion. Pain and dysesthesia due to spinal nerve injury usually resolve spontaneously within 72 h, and persistent injury is rare.[97] Direct trauma to the spinal cord may present as cauda equina syndrome, radiculopathy or focal neurologic deficit including foot drop. Neurological examination should guide further testing with EMG and MRI. EMG may not disclose an abnormality in the first 72 h after injury, and may need to be repeated if symptoms or signs persist.[98]

Necrotising fasciitis

Necrotising fasciitis at the site of procedure has been described in five postpartum women following spinal anaesthesia in India, with two fatalities.[99–103] Organisms isolated included streptococci, gram negatives, anaerobes and fungi.

Conclusion

Postpartum complications of uncertain aetiology raise considerable concern for the patient, relatives and caring healthy professionals, both acutely and with regard to risk of recurrence with subsequent pregnancy. Postpartum complications should not be attributed to neuraxial anaesthesia until other causes have been excluded. Obstetric physicians' awareness of the array of complications potentially related to neuraxial anaesthesia is crucial for management of the postpartum woman.

Ethical approval

Not applicable.

Informed consent

Not applicable.

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