

POST-DURAL PUNCTURE HEADACHE

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In the same report in which he described the first spinal anaesthetic, August Bier provided the first description of post-dural puncture headache (PDPH) associated with spinal anaesthesia (Bier, 1899).

PDPH is one of the most common complications of central nerve blocks, with an overall incidence that may be as high as 7 per cent (Halpern, 1994). Vandam (1956) reported an 11 per cent incidence after spinal anaesthesia; Bridenbaugh (1988) states that the incidence is up to 24 per cent and varying incidences are reported in different papers, with a level of up to 76.5 per cent after puncture with large diameter needles (Craft 1973, Di Giovanni 1970, Zoys 1996, Vandam 1956, Flaatten 1998, Lybecker 1990). Although usually self-limiting, it is a very unpleasant complication after spinal anaesthesia, diagnostic lumbar puncture, myelography and sometimes after diagnostic or therapeutic sympathetic blocks (Lehman 1996).

Mechanism

Two mechanisms have been postulated (Jankowic 2001). The first is a decrease in CSF pressure producing traction on structures within the cranium. The second suggests that the decreased CSF pressure leads to intrathecal hypotension and painful vasodilatation of the intracranial blood vessels (Camann 1990, Zoys 1996).

Diagnosis

This is based on clinical features. PDPH may occur shortly after puncture, but typically onset is from 7 hours to 48 hours afterwards (Jankowski 2002). There are reports of PDPH occurring up to 5 months after dural puncture (Vandam 1956). About 90 per cent of cases resolve within 10 days (Cass 1974, Zoys 1996). Symptoms are usually position dependent and consist of pain in the nape of the neck, stiffness of the neck, bifrontal and occipital headache and

spread into the shoulders. There can be associated nausea and vomiting, sensitivity to light, smell and noise, auditory disturbances and tinnitus, loss of appetite, depression and cranial nerve palsies (Day 1996). Seizures have been reported (Shearer 1995, Frison 1996, Paesch 1996). A subdural haematoma is a rare, but very serious, complication, said to have a mortality of 14 per cent (Vos 1991).

Differential Diagnosis

This includes migraine, tension headache, cervical myofascial pain, CNS infection (meningitis), sinus thrombosis, pneumoencephalus or allergy to dye or other drugs after epidurograms (Jankowic 2001).

Risk Factors

The range of reported incidences of PDPH is remarkably wide, from 0 to 85 per cent. Several patient-related factors have been shown to increase the risk of PDPH. These include age (Vandam 1956, Rasmussen 1989, Bolder 1986), pregnancy status (Vandam 1956, Meeks 1994), gender (Vandam 1956, Kortum 1982, Lybecker 1990) and a prior history (Lybecker 1990). There is a high incidence after inadvertent dural tap, complicating attempted epidural anaesthesia in labour. This may be due to lowered intra-abdominal pressure after delivery, pushing during the second stage or hormonal changes at the time of delivery. Numerous studies have demonstrated that age is a risk factor, the incidence increasing after the age of 10, peaking at 15 years and then decreasing with age, so that after 50 the risk is minimal.

Prevention

There are several technical factors that may lessen the likelihood of PDPH after intentional intrathecal tap. These include needle size and design (Halpern 1994, Cruikshank 1989, Ready 1989), bevel direction (Mihic 1985, Cruikshank 1989, Dittmann 1988) and mid-line

versus paramedian approach (Janik 1992). In healthy young patients, the risk of PDPH with a 27-gauge needle may be lower than 1.5 per cent (Flaatten 1998, Corbey 1997). A paramedian approach is said to decrease the risk, but this has not been verified in clinical trials.

Treatment

Non-invasive therapies include conservative management with bed rest, hydration, analgesics, sedatives and anti-emetics. There is no evidence that abdominal binders improve matters. The majority of patients settle within 5 to 7 days (Zoys 1996), and 80 per cent are improved within 2 weeks without any treatment (Jankowic 2001).

Active Treatment

This includes the use of caffeine sodium benzoate, ACTH or Sumatriptan.

Many trials have confirmed the value of caffeine, given intravenously in a dose of 500 mg of caffeine sodium benzoate in 1 litre over 1 hour (Camann 1990, Yucel 1994, Weber 1997). A further litre of the fluid is then administered and the treatment can be repeated after 4 hours. The belief is that cerebral vasoconstriction occurs with a reduction in cerebral blood flow (Sechzer 1978). This drug is not readily available in the UK, but personal experience has been somewhat disappointing. Sumatriptan does appear to be beneficial in some patients (Carp 1994, Jankowic 2001). ACTH or its analogues have been advocated, but this is yet to be tested (Kshatri 1997, Foster 1994).

Epidural blood patches have a long history of efficacy in treating PDPH, even though the mechanism by which they provide relief is unclear (Brownridge 1983). The blood may act as a plug, as it clots, or the increasing pressure within the space may stop the leakage (Beards 1993, Carrie 1993). Neither of these theories really explains the nearly immediate relief many patients experience. There may well be cephalad displacement of CSF, relieving the traction on the meninges. Crawford (1980) recommended the use of 20 mls, stating that this provided relief in 96 per cent

of cases, where 6 to 15 mls was only 70 per cent effective. More recently, Taivainen (1993) showed no difference between 10 mls versus 10 to 15 mls based on height.

Complications include back pain (35 per cent), fever and dizziness (Barash 1989, Bridenbaugh 1988). There is a risk of inadvertent sub-arachnoid injection, spinal cord compression, infection, persistent back pain, radicular pain, facial palsy (Perez 1993) and neural injury (Bolton 1989, Jarvis 1986). More recent studies, looking at large needle punctures, suggest that epidural blood patches are only 60 to 75 per cent effective in providing long-term relief of symptoms (Brownridge 1983, Taivainen 1993, Stride 1993).

Alternate methods include the use of an epidural Dextran patch (Stevens 1993, Zoys 1996), after a test dose, which should produce a longer period of compression but eventual complete disappearance of the injected substance and possibly less risk of infection. Infusion of isotonic saline via an epidural catheter only produces a temporary solution (Hawkins 1995).

Many clinicians argue in favour of conservative treatment initially; certainly ACTH and Sumatriptan could be tried and possibly caffeine sodium benzoate if available. Epidural blood patch is usually reserved for patients with severe symptoms, or those whose symptoms persist beyond an arbitrary limit. Prophylactic blood patching is of dubious side-effect benefit. Although some recommend that unintentional dural puncture with a large epidural needle should be treated by prophylactic blood patch (Duffy 1999), there is no data to support this.

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