Meningitis after spinal anaesthesia

Bacterial meningitis is a common and devastating illness that presents as a medical emergency requiring considerable diagnostic and therapeutic skills. Mortality is high and even those who recover may suffer a variety of permanent neurological sequelae. Because lumbar puncture (for whatever purpose) bypasses all the natural defence barriers of the central nervous system, it is clear that it carries at least the theoretical risk of introducing infection and causing meningitis. Two routes exist for such infection to reach the CSF. First, failure of aseptic technique could result in the introduction of exogenous organisms into the cerebrospinal fluid. Second, bacteria in the patient’s blood at the time of lumbar puncture may gain access to the sub-arachnoid space caused by microscopic bleeding caused by insertion of the needle.

Fortunately, there is good epidemiological evidence that the frequency of meningitis after lumbar puncture is no greater than in the ordinary population. Even in bacteraemic patients the incidence of meningitis after diagnostic lumbar puncture is not significantly different from the expected spontaneous rate. Furthermore, there were no cases of CSF infection in the classic prospective study of 8460 patients undergoing 10 098 spinal anaesthetics performed by Dripps and Vandam. Many of these patients underwent obstetric or urological procedures and were therefore at risk of bacteraemia, the incidence of which has been estimated at 3.6–7.2% after normal delivery. Higher rates might be expected in patients with obstetric complications such as chorioamnionitis or retained placenta, but two retrospective reviews of 27 000 and 505 000 patients who received extradural block in labour revealed only two with infective sequelae.

This safety record may be attributed in part to the practice of prescribing antibiotics in such patients to reduce both maternal and neonatal morbidity. Antibiotic therapy is usually introduced very early in any patient with meningeal symptoms, particularly if there are other signs of infection or a potential “aetiological factor” such as lumbar puncture for spinal anaesthesia. When purulent CSF is obtained from such patients, but no organisms are grown on culture, there is often an assumption that the antibiotic has inhibited bacterial growth. However, it is important to remember that bacteria are not the only cause of meningitis. The viral condition is generally much more benign, but may be caused by a wide range of organisms, and Coxackie B infection has been described after extradural block on at least one occasion.

It is a matter of mere epidemiological coincidence that some patients who have received spinal or extradural block could develop this infection. However, most cases of meningitis after central nerve block are probably aseptic in origin. Fifty years ago aseptic meningitis was not an uncommon complication of spinal anaesthesia and by 1947 more than 100 cases had been described in the literature and an incidence of 0.26% was quoted in a summary report of approximately 46 000 spinal anaesthetics. However, the incidence declined dramatically during the 1950s after a series of studies that had implicated chemical contamination of the CSF as the cause. It was shown in 1937 that this could result in meningeal irritation and animal studies showed that a whole range of detergents and chemicals could produce symptoms and histological changes consistent with aseptic meningitis. Recognition of these factors led to improvements in technique and equipment and these have reduced markedly the incidence of problems, although sporadic case reports still emphasize the need for attention to detail.

Another important, but much less widely appreciated, cause of aseptic meningitis is systemic drug administration. Drug-induced meningitis has been reported after non-steroidal anti-inflammatory agents, antimicrobials, ranitidine, carbamazepine and azothiaprine. In 1992, Marinac summarized 31 cases after ibuprofen alone in a literature review. The syndrome is more common in females with underlying autoimmune or collagen vascular disorders, but is occasionally reported in normal individuals, and is presumed to be an acute hypersensitivity reaction. There are some immunological data to support this hypothesis.

Signs and symptoms typically appear a few hours after drug ingestion, but may be delayed by weeks. In addition to the usual features of meningitis there may be peri-orbital oedema, conjunctivitis, hypotension, parotitis, pancreatitis, lethargy, diplopia, seizures
and confusion. Fever is common. The CSF picture varies greatly, but there is generally polymorphonuclear pleocytosis, increase in protein concentration and normal glucose. Symptoms worsen with continued drug administration, but most patients recover fully in a few days after the offending medication is stopped. Definitive diagnosis requires re-administration of the drug to the patient.

Irritation and inflammation of the meninges must be accepted as a potential complication of spinal anaesthesia. Perhaps the greatest advantage of the technique is that only a tiny dose of drug is required to produce a major degree of block, the main reason being that all the barriers surrounding the spinal cord and nerve roots are bypassed by the injection and the drug reaches its site of action easily. However, all the natural protection of the CSF is circumvented also. As long as equipment and drug solutions manufactured to modern standards are applied with a sound aseptic technique, the incidence of complications should be very low and the occasional case of meningitis is likely to be coincidental. However, every case should prompt a thorough consideration of all possible aetiological factors and this must include the equipment and technique used. When there is no obvious bacterial infection the possibilities of viral infection, chemical contamination and drug-induced meningitis must each be investigated. The latter is particularly important given that two groups of drugs used widely in anaesthesia (NSAID and H2 blocking agents) have been shown to cause the condition. We have traced five cases of meningitis after dural puncture in the recent medical literature; four of these were aseptic in nature but no intercurrent drug history was given and the possibility of drug-induced meningitis was not raised.

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References