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## Meningitis associated with spinal anaesthesia: not always bacterial☆

### Meningitis asociada a anestesia espinal: no siempre bacteriana

*Sir,*

Laguna del Estal et al.[1] have recently reported a series of patients with bacterial meningitis associated with epidural analgesia and anaesthesia, and in their discussion they quite rightly pointed out that the differential diagnosis must include chemical meningitis.[1] It is important to stress that meningitis induced by the local administration of anaesthetics must also be suspected whenever the cultures are negative. The clinical picture produced is indistinguishable from that of bacterial meningitis, but what many clinicians are unaware of is that the cerebrospinal fluid (CSF) may also be negative, revealing intense pleocytosis and polymorphonuclear predominance. These situations are well documented with, for example, bupivacaine, which may trigger pleocytosis of several thousand leucocytes with a percentage of polymorphonuclear cells close to 100%.[2—4] Some important facts may help distinguish between bacterial and aseptic meningitis. First of all, the latency between epidural anaesthesia and the onset of symptoms as a time of less than 6 h suggests that it is chemical meningitis. Second, the presence of eosinophilia in CSF, which is ''never'' seen in bacterial meningitis but is in drug-induced meningitis, or else that the patient presents atopy. Third, the presence of hypoglycorrachia less than 30 mg/dL, typically occurring in bacterial forms (albeit also described in aseptic cases). And finally, a frank elevation of acute phase reactants, common only in bacterial meningitides.

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# Reply to Meningitis secondary to spinal anaesthesia: not always bacterial meningitis☆

## Respuesta a meningitis asociada a anestesia espinal: no siempre bacteriana

*Sir,*

We agree with Reus Bañuls et al. about the importance of defining, when evaluating patients with acute meningeal syndrome and negative results in Gram-staining and cerebrospinal fluid (CSF), whether the meningitis is bacterial (MB) or aseptic (MA).[1] This differentiation allows treatment to be adapted (need for antibiotic therapy), admission to hospital prescribed or its duration adjusted, and accurate prognostic information to be provided to the patient, etc.

The distinction between these two large groups of acute meningitis cases arises in those acquired in the community, where MB would basically tend to be differentiated from viral infections,[2] but also from other, less frequent aetiologies such as drugs (trimetroprim-sulphamethoxazole, non-steroidal anti-inflammatory drugs, immunoglobulins, etc.), intracranial tumours that may be a cause of chemical meningitis (dermoid cysts, craniopharyngioma, infarction of a pituitary adenoma) or systemic diseases occasionally coursing with meningeal involvement (lupus erythematosus, sarcoidosis, Behçet's disease, etc.).

It is no less important to distinguish both types of meningitis in an in-hospital setting as numerous medical procedures can lead to complications with either MB or chemical MA: neurosurgery,[3] intrathecal administration of medication[4] and spinal anaesthesia and analgesia.[5] In connection with this last technique, the subject of the report mentioned, it should be recalled that MA secondary to spinal anaesthesia was not so infrequent during the first half of the 20th century, with an estimated incidence of 0.26%.[6] Nonetheless, the improvement in material sterilization procedures and aseptic techniques, the use of disposable medical material and the administration of drugs with less allergy-producing effects have meant that chemical meningitis as a complication of spinal anaesthesia is currently exceptional and reflected in case reports when diagnosed.

Yet, however frequent and relevant it may be, the problem of differentiating MB from chemical MA secondary to medical procedures has not been resolved when the basic microbiological studies are negative. It is true that the presenting clinical symptoms and CSF analysis (cell count and formula, proteins and glucose) do not enable this distinction to be drawn,[7,8] although a short latency prior to the appearance of symptoms and the observation of eosinophils in the CSF (present in only 22% of the cases reported by Santos et al.[5]) suggest a chemical origin in cases secondary to the use of drugs delivered through the spine. It might be diagnostically more useful to determine a series of different inflammation/infection markers, which are considerably elevated in severe bacterial infections such as MB, but not in chemical meningitis: reactive C protein in serum,[8] procalcitonin in serum[8] and lactic acid in the CSF.[9] However, differentiating them quickly and for sure will not be possible until the techniques for detecting bacterial genoma in the CSF through polymerase chain reaction[10] are standardized.

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