Cerebrospinal fluid lactate

The predictive ability of CSF lactate for bacterial meningitis has been examined in two published metaanalysis [1) 2)].

It can differentiate bacterial meningitis (> 6 mmol/l), from partially treated meningitis (4 to 6 mmol/l) and aseptic meningitis (< 2 mmol/l) [3)] However, other researchers have suggested that CSF lactate offers no additional clinically useful information over conventional CSF markers [4) 5)].

In a febrile patient with a ventriculostomy, diagnosing or excluding bacterial or microbial ventriculitis is difficult, as conventional markers in analysis of cerebrospinal fluid (CSF) are not applicable due to presence of blood and inflammation.

Data from a large sample of CSF studies in patients with ventriculostomy indicate that no single value of CSF lactate provided both sensitivity and specificity high enough to be regarded as reliable test [6)].

Glutamate and lactate are significantly increased in nonsurvival relative to survival patients. We tested the accuracy of both biomarkers to discriminate patient outcome. Setting a cutoff of >57.75, glutamate provides 80.0% of sensitivity and 84.62% of specificity (AUC: 0.8214, 95% CL: 54.55-98.08%; and a cutoff of >4.65, lactate has 100% of sensitivity and 85.71% of specificity (AUC: 0.8810, 95% CL: 54.55-98.08%). BDNF and GDNF did not discriminate poor outcome [7)].

Cerebrospinal fluid (CSF) lactate concentration is known to increase during the acute phase after severe head injury. To determine the influence of glycemia or cerebral ischemia on this lactate increase, De Salles et al. studied 69 head-injured patients aged 28.7 +/- 15.4 (SD) years with a mean Glasgow coma scale of 5.7 +/- 1.7 (SD). They were intubated, paralyzed, and artificially respired. They measured lactate and glucose concentrations in ventricular CSF (VCSF), arterial blood, and jugular bulb blood for 5 days. Samples were obtained within 12 hours after injury and at regular 12-hour intervals. These patients were not treated for hypo- or hyperglycemia. Cerebral blood flow (CBF) was also measured within 12 hours and at 12- to 48-hour intervals. Hyperglycemia was found consistently within 12 hours after injury (224 +/- 98 mg/dl, P less than 0.001), and mild hyperglycemia persisted during the entire period of study. The VCSF glucose course was parallel to that in blood (the initial VCSF glucose value was 128 +/- 37 mg/dl, P less than 0.001). The blood lactate value was also elevated during the first 12 hours (4.2 +/- 2.0 mmol/litre, P less than 0.001), normalizing within 24 to 36 hours. The VCSF lactate course was independent from that of the blood lactate value. It was significantly elevated within 12 hours after injury (5.3 +/- 2.6 mmol/litre, P less than 0.001) and remained so during the 5 days of study. A high initial VCSF glucose value was associated with a high initial VCSF lactate value. However, a high VCSF lactate concentration was present even when the glucose value was close to the normal level [8)].

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