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Pathophysiology of Chronic Subdural Hematoma

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Description

Pathogenesis of chronic subdural hematoma has been controversial for more than a century and still remains obscure. Two theories have been proposed: the osmotic gradient pressure and the recurrent hemorrhage from hematoma capsule associated with hyperfibrinolysis theory. The latter hypothesis has been more widely accepted. We would like to make a comment on the current state of the other theory, that of osmotic gradient pressure. Gardner first proposed that the membrane around the hematoma acts as an osmotic membrane, with cerebrospinal fluid diffusing into the hyperosmotic hematoma [1]. He believes that this is the way that chronic subdural hematomas increase in size.

We know today that changes in intracranial pressure and cerebral blood flow are not significant factors in the evolution of neurological dysfunction after chronic subdural hematoma. Mechanical distortion of central regions of the brain is probably the main cause of neurological aggravation [2]. In patients suffering from chronic subdural hematoma, normal intracranial pressure has been observed in the presence of a midline shift as large as 20 mm. Owing to the slow expansion of the hematoma, the period of spatial compensation is probably long enough to cause significant distortion of the brain before there is a significant intracranial pressure rise. But can this brain distortion and even brain herniation happen without any pressure gradients within the brain?

The osmotic gradient theory of Gardner, even if in complete accordance with the physical laws, was discredited after the work of Weir, who did not find any significant increase in the osmolality of the hematoma with increasing age, or any significant differences between the osmolalities of the blood and the hematoma [3].

On the other hand, the fragility of the neocapillaries in the outer membrane results in microhemorrhages and excessive fibrinolytic activity [4]. Finally, Sato's work suggests an abnormally high capillary endothelial permeability, contributing to hematoma's enlargement [5]. These last theories give us a reasonable explanation for the development of chronic subdural hematomas and the clinical deterioration observed in these patients. But we must also know that even very recent articles show that in cases of experimental hydrocephalus, were microhemorrhages or abnormally high vascular permeability cannot be correlated with clinical deterioration, significant pressure gradients cannot be measured. In these cases, no alternative hypotheses exist, so we must conclude that the current resolution of our sensors within the brain (0,5 mmHg) is not adequate [6]. Undetectable pressure gradients do not mean that their results are insignificant. We believe that pressure gradients within and around chronic subdural hematomas cannot be excluded with our current technology. The complete elucidation of the pathophysiology of chronic subdural hematoma should wait for the development of more sensitive pressure sensors. Other newer techniques [7] can also help to solve this problem.

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