Communicating hydrocephalus

Communicating hydrocephalus, also known as non obstructive hydrocephalus, is caused by impaired cerebrospinal fluid reabsorption in the absence of any CSF-flow obstruction between the ventricles and subarachnoid space. It has been theorized that this is due to functional impairment of the arachnoidal granulations (also called arachnoid granulations or Pacchioni's granulations), which are located along the superior sagittal sinus and is the site of cerebrospinal fluid reabsorption back into the venous system.

Etiology

Posthemorrhagic hydrocephalus see also Secondary normal pressure hydrocephalus

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Congenital absence of arachnoid villi. Scarring and fibrosis of the subarachnoid space following infectious, inflammatory, or hemorrhagic events can also prevent resorption of CSF, causing diffuse ventricular dilatation.

Normal pressure hydrocephalus (NPH) is a particular form of communicating hydrocephalus, characterized by enlarged cerebral ventricles, with only intermittently elevated cerebrospinal fluid pressure.

Idiopathic normal pressure hydrocephalus

Hydrocephalus ex vacuo.

It was suggested that communicating hydrocephalus is an almost universal finding after hemicraniectomy and that early cranioplasty may prevent the need for permanent cerebrospinal fluid diversion in these patients, but results suggest that, contrary to some beliefs, hydrocephalus does not frequently occur after decompressive craniectomy [1].

Communicating hydrocephalus associated with a vestibular schwannoma can occur in younger patients than was previously thought. An elevated CSF protein appears to be important, but other factors may be involved [2].

Communicating hydrocephalus is frequent in patients with malignant middle cerebral artery infarction after decompressive hemicraniectomy. A later time point of cranioplasty might lead to a lower incidence of required shunting procedures in general [3].

Pathogenesis

For the communicating hydrocephalus, without etiological treatment, pathogenesis has been considered as a research emphasis. Many factors can damage the CSF system and trigger communicating hydrocephalus, including tumor surgery and hydrocephalus neurological diseases, such as brain trauma, infection, ICH and SAH. But, a big proportion of patients do not develop hydrocephalus. That is because the absorbing ability of CSF can compensate within a certain range. If the damage exceeds that range, hydrocephalus will occur. Once it occurs, it is not likely to be reversed, so a shunt surgery is always needed. Therefore, Xu et al believe that there orientation could transform the treatment of patient who has already showed hydrocephalus symptoms to the prevention of the occurrence in the patient with high risk of hydrocephalus. Based on the hypothesis above, they first divide the process of hydrocephalus into three stages and believe that hydrocephalus are possible be reversed or halted in stage 1 and 2. The new concept of the pathogenesis in hydrocephalus will enrich the understanding and provide new insights to the therapeutic orientation. In conclusion, the future research direction should be the prevention of hydrocephalus, which should take a long period from the immediate occurrence of brain injury to several months or even years after the injury [4)].

Diagnosis

Measurement of intracranial pressure (ICP) is necessary in many neurological and neurosurgical diseases. To avoid lumbar puncture or intracranial ICP probes, non-invasive ICP techniques are becoming popular. A recently developed technology uses two-depth Doppler to compare arterial pulsations in the intra- and extra-cranial segments of the ophthalmic artery for non-invasive estimation of ICP. The aim of this study was to investigate how well non-invasively-measured ICP and invasively-measured cerebrospinal fluid (CSF) pressure correlate. We performed multiple measurements over a wide ICP span in eighteen elderly patients with communicating hydrocephalus. As a reference, an automatic CSF infusion apparatus was connected to the lumbar space. Ringer's solution was used to create elevation to pre-defined ICP levels. Bench tests of the infusion apparatus showed a random error (95 % CI) of less than ±0.9 mmHg and a systematic error of less than ±0.5 mmHg. Reliable Doppler signals were obtained in 13 (72 %) patients. An infusion test could not be performed in one patient. Thus, twelve patients and a total of 61 paired data points were studied. The correlation between invasive and non-invasive ICP measurements was good (R = 0.74), and the 95 % limits of agreements were -1.4 ± 8.8 mmHg. The within-patient correlation varied between 0.47 and 1.00. This non-invasive technique is promising, and these results encourage further development and evaluation before the method can be recommended for use in clinical practice [5)].

Treatment

Cerebrospinal fluid shunt

Endoscopic third ventriculostomy.

Outcome

Intracranial and intraspinal compliance are parameters of interest in the diagnosis and prediction of treatment outcome in patients with normal pressure hydrocephalus and other forms of communicating hydrocephalus.

Case series

Arachnoid villi from 50 adult patients were sampled at autopsy. All specimens were subjected to a variety of histochemical and immunohistochemical stains. The 23 cases of SAH consisted of patients in whom an autopsy was performed 12 hours to 34 years post-SAH. Fifteen cases were identified as moderate-to-severe SAH, with varying degrees of hydrocephalus. In comparison with 27 age-matched non-SAH controls, the authors observed blood and inflammation within the arachnoid villi during the 1st week after SAH. Greater mitotic activity was also noted among arachnoid cap cells. The patient with chronic SAH presented with ventriculomegaly 2 months post-SAH and exhibited remarkable arachnoid cap cell accumulation.

The authors postulate that proliferation of arachnoidal cells, triggered by the inflammatory reaction or blood clotting products, could result in obstruction of CSF flow through arachnoid villi into the venous sinuses. This does not exclude the possibility that SAH causes generalized fibrosis in the subarachnoid space [6].

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