Slit ventricle treatment

Logical management of slit ventricle requires an understanding of the specific pathogenesis of the problem in individual patients, whether based on monitoring of intracranial pressure monitoring or observation at the time of shunt failure or symptoms. Shunt overdrainage syndromes, whether intermittent proximal obstruction or low pressure states, are best managed with valve upgrades and the addition of devices that retard siphoning. Increased ICP without ventriculomegaly at the time of shunt failure is best managed by shunting devices that access the cortical subarachnoid space such as lumboperitoneal shunts or shunts involving the cisterna magna. Cranial expansion operations and subtemporal decompression should be limited to patients with craniofacial syndromes [1].

A significant number of patients may experience improvement in their symptoms and undergo shunt removal according to a protocol, improving their quality of life and simplifying their medical follow up [2].

Treatment of asymptomatic slit ventricles

Prophylactic upgrading to a higher pressure valve or insertion of an antisiphon device as initially advocated has largely been abandoned. However, this may be appropiate at the time of shunt revision when done for other reasons [3].

Treatment of intracranial hypotension

see Intracranial hypotension treatment.

Treatment of intermittent occlusion

1.- If symptoms occur early after shunt insertion or shunt revision, initial expectant management may be indicated since symptoms will spontaneously resolve in many patients as they equilibrate to the new intracranial pressure.

2.- Revision of the proximal shunt. This may be difficult due to the small size of the ventricles. One can attempt to follow the existing tract and insert a longer or shorter length of tubing based on the preoperative imaging studies. Some advocate the placement of a second ventricular catheter, leaving the first one in place.

3.- Patients may respond to either following interventions because the slight ventricular enlargement elevates the ependyma off of the inlet ports (this may not always be the therapy of choice):

a) Valve upgrade [4].

b) Antisiphon device insertion [5] [6] ; the procedure of choice in some opinions [7]. First described in 1973 [8].

4.- Subtemporal decompression [9) 10) 11)] sometimes with dural incisions [12)]. This results in dilatation of the temporal horns in most but not all cases [13)] cases.

5.- Endoscopic third ventriculostomy [14)].

Case series

2002

Seven patients with a diagnosis of slit ventricle syndrome (SVS) underwent lumboperitoneal shunting. The age at shunting ranged from 3 to 18 years. Most had undergone recent ventriculoperitoneal shunt (VPS) revisions for presentation of shunt malfunction-like symptoms. Despite this, all remained symptomatic and underwent a sedated lumbar puncture to measure opening pressure (OP). All had high OP in spite of a functional VPS and underwent LPS placement.

All 7 patients had a prolonged period of overdrainage symptoms after lumboperitoneal shunting that resolved completely over several weeks. The initial etiology of hydrocephalus was reported to include trauma, aqueductal stenosis and intraventricular hemorrhage of prematurity. Two patients required revision of their LPS, after which their symptoms again resolved.

In a certain subset of patients with SVS who are symptomatic from increased ICP, placement of an LPS is an effective treatment option. It appears that this subgroup of patients previously treated with ventriculoperitoneal shunting behave in a fashion similar to pseudotumor cerebri patients and respond well to lumboperitoneal shunting [15)].

1987

The slit-ventricle syndrome (SVS) has been the subject of diverse opinions and recommendations during the past 2 decades. In an effort to define the clinical features of SVS and to make recommendations concerning management we have reviewed 15 cases treated by a fairly uniform technique during the past 5 years. The syndrome consists of: (1) intermittent, but self-limiting episodes resembling shunt malfunction, usually lasting a few days, (2) nonfilling of the pumping device after compression, and (3) a slit-like ventricular system on CT scan. In all but 2 patients the initial shunt was performed in infancy. The mean interval from the initial shunt to treatment of SVS was 6 years. The age range at onset of SVS varied from 2 to 17 years with a mean of 7 years. All patients in this series were relieved of symptoms by placement of an antisiphon device and, in most patients, upgrading the valve resistance. Analysis of this series has led to the following conclusions: (1) SVS is a characteristic clinical entity, usually distinguishable from persistent shunt malfunction and from low-pressure headache, (2) the pathogenesis is intermittent obstruction of the ventricular catheter, (3) there is no good evidence that changes of brain compliance or La Place principles apply, and (4) placement of antisiphon device and upgrading valve resistance are effective treatments [16)].

1983

Pre- and post craniectomy ventricular areas were measured from computed tomography scans with a computer digitizing technique in three patients with this syndrome who had undergone four surgical

procedures. All patients improved symptomatically following craniectomy. A significant decrease in total ventricular area was noted in all instances. The results suggest that subtemporal craniectomy causes the ventricles to become smaller, not larger [17].

1979

Holness et al. performed subtemporal decompression to deal with recurrent shunt obstruction in 22 hydrocephalic patients with the slit-ventricle syndrome. 13 patients have been followed-up for more than 1 year and 2 others for more than 2 years. The frequency of hospitalization for shunt-revision has been greatly reduced. These results confirm that subtemporal craniectomy is useful in the treatment of patients in whom this syndrome develops [18].

1978

Acute obstruction of a shunt may occur when the ventricular space becomes slitlike. Four patients with collapsed ventricles were treated by adding a higher pressure valve to the system. The ventricles enlarged and the patients became asymptomatic. Twelve children who had been shunted in the neonatal period had a higher pressure valve added as an elective procedure to prevent ventricular collapse. There were no episodes of shunt obstruction in the subsequent seven to 38 months [19].

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