Cerebellar Infarction Surgery

- Endovascular treatment of a craniocervical junction dural arteriovenous fistula associated with lateral medullary syndrome: A case report
- Wallerian degeneration of the middle cerebellar peduncles secondary to pontine infarction, case report, and review of literature
- A case of recurrent hemangioblastoma receiving blood supply from the mastoid and transosseous branches of the occipital artery
- Successful endovascular occlusion of multiple fusiform aneurysms on the persistent primitive lateral basilovertebral anastomosis
- Delayed Diagnosis of Myxedema Coma in a Patient With Concurrent Severe Intracranial Atherosclerotic Disease
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- Atrial myxoma and posterior circulation stroke

The choice can be made between ventriculostomy (external ventricular drainage; EVD), suboccipital decompressive craniectomy (SODC) [1].

Concerning the extent of the surgical procedure, some authors argue that implantation of an extraventricular drainage (EVD) is sufficient, whilst others fear the possibility of upwards herniation across the tentorium

Surgical indications

Contrary to supratentorial hemispheric strokes, deterioration is not necessarily dependent on a specific vascular territory, but on the initial infarct volume [2].

The surgical treatment is decided when there are threats of herniation or brainstem compression.

Surgical decompression should probably be done as soon as any of the following signs develop if there is no response to medical therapy.

It is important to recognize a lateral medullary syndrome (LMS) which may often accompany a cerebellar infarction. With LMS, the signs are usually present from the onset (dysphagia, dysarthria, Horner syndrome, ipsilateral facial numbness, crossed sensory loss…), and are not accompanied by a change in sensorium. There is no place for surgical decompression in LMS since it represents primary brainstem ischemia and not compression.

Findings proceed in the approximate following sequence if there is no intervention:

1. abducens (VI) nerve palsy

2. loss of ipsilateral gaze (compression of VI nucleus and lateral gaze center)

3. peripheral facial nerve paresis (compression of facial colliculus)

4. confusion and somnolence (may be partly due to developing hydrocephalus)

5. Babinski sign

6. hemiparesis

7. lethargy

8. small but reactive pupils

9. coma

10. posturing→flaccidity

11. ataxic respirations

In an institution experience for patients with worsening levels of consciousness and radiologically evident ventricular enlargement, they recommend external ventricular drainage, and reserve surgical resection of necrotic tissue for patients whose clinical status worsens despite ventriculostomy, those for whom worsening is accompanied by signs of brainstem compression, and those with tight posterior fossae [3].

Suboccipital Decompressive Craniectomy for cerebellar infarction

Suboccipital Decompressive Craniectomy for cerebellar infarction.

External ventricular drain for cerebellar infarction

External ventricular drain for cerebellar infarction.

Endoscopic third ventriculostomy

10 patients with a resulting hydrocephalus caused by a space-occupying cerebellar infarction were managed with ETV. Glasgow Coma Scale score on admission, cause of stroke, and computed tomographic signs, including the ischemic vascular territory involved and brain edema, were noted. Clinical outcome was evaluated using the Glasgow Outcome Scale.

Results: In all patients, there was a mean interval of 4 days from the onset of deterioration of consciousness to operation. Mean Glasgow Coma Scale score on admission was 11.2. In nine patients, ETV was the initial procedure of ventricular drainage. One patient was primarily treated with an external ventricular drainage, but the device dislocated and ETV was performed. In one patient, an external ventricular drainage became necessary 7 days after the initial ETV because of a malfunction of the stoma. One patient showed a progressive brain edema 2 days after ETV, and suboccipital

decompression was performed. Eight successfully treated patients demonstrated an improvement in the level of consciousness after ETV. Mean Glasgow Outcome Scale score on discharge of all patients was 3.4.

Conclusion: Occlusive hydrocephalus caused by cerebellar infarction is infrequent. When occlusive hydrocephalus is observed, ETV can be used successfully with minimal risks, especially with avoidance of a higher rate of infectious complications caused by external drainage systems [4].

References

1)

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2)

3)

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4)

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