PSEUDOTUMORAL ISCHEMIC STROKE. CASE REPORT

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Keywords: cerebral *Abstract:* Cerebral infarction (CI) is an irreversible necrosis of brain tissue due to a focal decrease or infarction, arterial occlusion, intracranial spasm. Arterial occlusion may be thrombotic or embolic in nature, and may be accompanied by associated massive cerebral edema, which will act as an intracranial expansive process.

INTRODUCTION

Ischemic and hemorrhagic strokes represent together the third cause of death after heart disease and cancers.(1) Annual mortality by this disease varies between 64-274 / 100.000 inhabitants in Europe.

CI represents the largest proportion (70-80%) of strokes. Approximately 50% of CIs are due to large vessels atherosclerotic disease. Another 20% are represented by lacunar infarctions produced as a result of the occlusion of the deep penetrating arteries with low diameter.(1) Embolic events are the source of 30% of CI, cardiac origin being the most common starting point.(1)

Ischemic stroke may be accompanied by marked cerebral edema, approximately in 10% of cases.(2)

Besides the classic symptoms occurring in cerebral ischemia, in the patients in whom cerebral edema is associated by accompaniment, brain stem damage signs appear, and in the absence of a local decompression intervention, the evolution is towards death.(3)

CASE REPORT

Male patient, G.P., 72 years old, retired, rural origin, was admitted to the Neurological unit for a motor deficit on the right hemibody, expressive aphasia, on 29/10/2014.

Patient's history revealed chronic ischemic heart disease, hypertension, insulin-dependent type 2 diabetes, obesity grade 2.

Clinical and neurological examinations performed on 10.29.2014 revealed right hemiplegia, aphasia, acute urinary retention.

Computed tomography (CT), initially performed, revealed the development of a left Sylvian stroke.

The patient undertakes treatment specific for ischemic stroke, but in evolution (on 31/10/2014), there occurs neurological worsening with the installation of a coma, spontaneous decerebration seizures, reactive bilateral mydriasis.

CT scan performed urgently on 31.10.2014 revealed left Sylvian stroke with small hemorrhagic areas with marked mass effect, pseudotumoral (figures no.1, 2).

The patient was on emergency transferred to the Neurosurgery ward of Sibiu Hospital on 31.10.2014, orotracheal intubation being practiced with hyperventilation, administration of cerebral depletives, and preoperative preparation.

Figure no. 1. Left Sylvian stroke



Figure no. 2. Pseudotumoral left Sylvian stroke with small hemorrhagic areas



Emergency Neurosurgery was practiced, with left fronto-temporal-parietal decompressive craniectomy, local decompression, epidural drainage, the patient being also thracheostomized.

Cranial check-up CT revealed the return of the ventricular system at midline level, sac with the presence of

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herniation in the brain substance in the surgery breach and small areas of bleeding (figures no. 3, 4).

Patient's neurological status was improved, being transferred to the Neurology department with the wound healed (figure no. 5), in order to continue the recovery therapy.

Figure no. 3. Post-operative appearance



Figure no. 4. Postoperative appearance



Figure no. 5. Postoperative appearance



DISCUSSIONS

Cerebral infarction is an irreversible necrosis of brain tissue due to the focal decrease or absence of blood flow and which may be thrombotic or embolic in nature.

Primary bleeding, where the initial event was a vascular rupture, should be differentiated from the hemorrhagic transformation, the latter occurring as a complication of the ischemic stroke, developing within the ischemic area and being produced by ischemic vascular occlusion that occurs

spontaneously or induced by the anticoagulation or thrombolytic therapy.(4) This differentiation is needed in order to establish the etiology and treatment, which are totally different.

This haemorrhagic transformation can be defined both clinically and radiologically, comprising a broad spectrum of secondary bleeding ranging from small areas of petechial hemorrhage up to space replacement hematoma.

Clinically, hemorrhagic transformation of ischemic stroke could be symptomatic and asymptomatic, manifesting through a rapid deterioration in the health of the patient, resulting in signs of intracranial hypertension, marked headache, nausea and vomiting and altered consciousness to coma.(5)

Surgical intervention should be performed before the installation of the brainstem lesions, as early as possible, taking advantage of the "window" provided by the administration of brain depletives.

Another decisive factor in taking the surgery decision is the biological status of the patient before the stroke onset. Patients under 60 years old are ideal candidates, as well as those with minimum associated defects.

Surgical intervention can be carried out even if tissue plasminogen activator has been administered at a dose of 0.9 mg / kg body.(6)

The operative technique consists of practicing decompressive hemicraniectomy, to get as much functional independence of the patient, associated or not with the secondary intraparenchymal hematoma evacuation, thereby reducing the hypertension syndrome. The patients who survive this will present major neurological deficits.(6,7) Cranioplasty can be made three months later, if there are no signs of local infection.

CONCLUSIONS

Surgery intervention is adapted to the patient's neurological status, being carried out on maximum emergency if the neurological status worsens.

Neurological monitoring is mandatory in patients with ischemic stroke or intracranial expansive process.

Plain CT scan is repeated whenever the patient's neurological condition deteriorates.

Interdisciplinary collaboration is required.

Surgery intervention is carried out with the informed consent of the family.

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AMT, vol. 20, no. 4, 2015, p. 95