UNEXPECTED COMPLICATION IN CEREBRAL HAEMORRHAGE. HOW DO WE PROCEED?

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Abstract: *Cerebral haemorrhage is the second most frequent cause of stroke, and the most important risk factor is hypertension, followed by aneurysms and arteriovenous malformations. The frequency of hemorrhagic strokes etiology is much lower than the ischemic stroke (15% hemorrhagic vs. 85% ischemic) but their gravity is higher, leading to a higher proportion to exitus (40% of those with hemorragic stroke die).*

INTRODUCTION

Spontaneous intracerebral haemorrhage represents the overflow of blood into the cerebral parenchyma, this overflow being produced from the intraparenchymal vessels in a non-traumatic manner.(1,2) Statistics show that intracerebral haemorrhage is more common in the male population.(3) The direct cause is the rupture of an intracranial vessel, most commonly due to high blood pressure.(4) Hematomas can be located: supratentorial (85%), infratentorial (15%). Of the supratentorial hematomas, the most common location is in basal ganglia (42%) and in lobes (40%).(2) They may also be located at ventricular or secondary levels with ventricular burglary.

High blood pressure is present in more than 50% of cases of high bleeding. Of particular importance in the production of intracranial haemorrhages are:

- the old age (the incidence increases significantly after age 55 years),
- smoking,
- excessive consumption of alcohol (one study suggests that consuming >3 drinks a day increases the risk of ICH by ≈ 7 times), liver dysfunction,
- aneurysms and arterio-venous malformations which are "a heterogeneous group of rare vascular lesions of the brain; some remain asymptomatic (static lesions), others enlarge and become hemorrhagic",(9)
- intracerebral neoplasia,
- drugs,
- clotting disorders,
- infections,
- cerebral amyloid angiopathy.(5,6,7)

Symptomatology is usually suddent onset with a focal neurological deficit in full apparent state of health that progresses over a few minutes, but may vary depending on the location and size of the hematoma. Often, there are: intense headache, nausea and vomiting, intracranial hypertension syndrome, altered consciousness depending on the location and size of the hematoma.

Other signs of onset may be:

- comitial crises,
- neck revitalization,
- subfebrile state,
- heart rhythm disorders.

Intracerebral hematomas are readily detectable on CT due to the hemoglobin content in the extravasated blood, so they can be highlighted even within the first 4 hours after haemorrhage.

After 72 hours of bleeding, the hematoma becomes hypodense at the periphery.

After 4-7 days of haemorrhage, the hematomas become difficult to highlight because the clot becomes parenchyma isodense. MRI examination is rarely used.

In case of suspicion of arteriovenous malformation, an Angio-CT investigation is required.

Prognosis depends on the location and volume of the hematoma, GCS on admission, intraventricular invasion, re-bleeding.

The hematoma volume can be calculated in different forms. One of the calculation methods is Broderick. Another calculation method is the one Vicenţiu Săceleanu studied in his PhD thesis and he says that the accuracy of the new hematoma volume calculation method is the area under the curve (AUROC). According to the data of the patients undergoing the comparative study of the two methods, there was observed the degree of higher specificity of the new method applied (0,46 compared to 0,38).(8)

Another factor is the age of the patient: in the acute stage, mortality is directly related to the severity of bleeding, regardless of age, but later it is related to the patient's general condition and complications that increase with age. The probability of survival is lower in the hematoma of the brainstem.

Treatment can be conservative when the patient is conscious, GCS 15 points, frustrating hemiparesis, without intracranial hypertension, GCS ≥ 13 and hematoma diameter <4 cm.

Treatment is surgical in emphasizing intracranial hypertension syndrome with all adopted conservative treatment, worsening of state of consciousness and motor deficit. Coagulopathy cases benefit from surgical treatment when intracranial hypertension is high after correction of clotting

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disorders.

It was found that no benefit was gained by operating patients with GCS <5, those with fixed bilateral midriasis, without brainstem reflexes.

The benefit of surgical intervention in deep hematomas (basal ganglia and thalamus) does not change from that to unoperated ones. Intervention in this case is only to the insistence of the family. In the infratentoral hematoma, tetraplegia is not a contraindication to an operation. Immediate postoperative surgery - rebleeding, infection. Late postoperative surgery - thrombophlebitis by prolonged immobilization of patients with motor deficits, pulmonary embolism, bronchopneumonia, urinary tract infections.

CASE REPORT

We present the case of a 63-year old patient, male, known for high blood pressure untreated, obesity, mixed dyslipidemia and a history of ischemic stroke under platelet antiaggregation treatment, who is presented in the Neurosurgery Department via the Emergency Room for visual impairment, temporo-spatial disorientation with spontaneous onset and motor aphasia.

From a neurological point of view, the patient was conscious, slightly uncooperative on the background of motor aphasia, temporo-spatial disorientation, no signs of meningeal irritation, cranial nerve examination within normal limits, without sensitivity or motility disorders.

The cranio-cerebral CT scan highlights intraparenchymatous left parieto-occipital hematoma (figure no. 1, figure no. 2).

The patient was cardiologically assessed, biological samples were collected, and he was prepared for surgery.

Figure no. 1. Preoperative, sagital CT scan

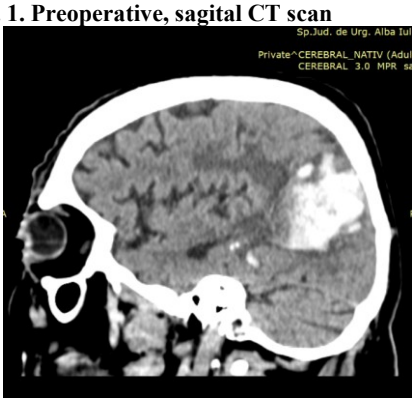
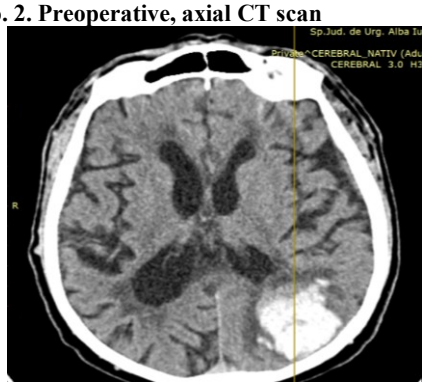


Figure no. 2. Preoperative, axial CT scan



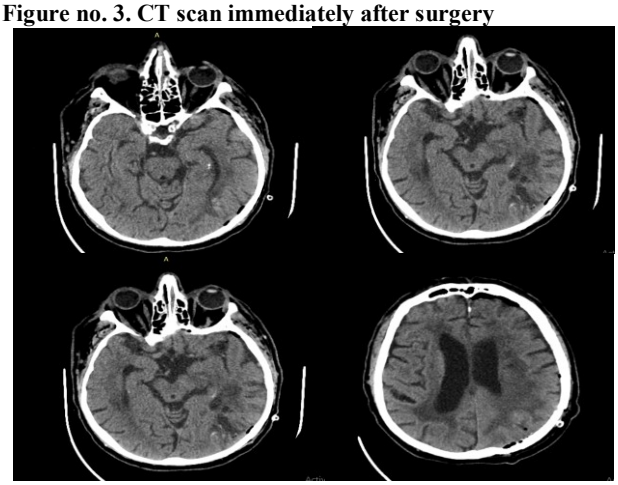
Treatment

Neurosurgical hematoma evacuation

- Left P-O Flap,
- Evacuation of the P-O left intraparenchymatous hematoma,
- Haemostasis, identifying and stopping the bleeding source.
- The bone flap was put in place,
- Suture of all the anatomical layers and placing a sub fascial drainage tube,

During surgery, the patient exhibits a large variation of blood pressure with decreases of up to 60 mmHG, followed by its increase after positive inotropic support. At the patient's entry into dorsal decubitus, the pupillary inequality with the midriasis in the right eye is observed. The patient is taken to the CT where was excluded the possibility of a cerebral haemorrhage (figure no. 3). He is transported to the Intensive Care Unit and supervised observing within a few hours the remittance of pupil inequality.

Figure no. 3. CT scan immediately after surgery



Postoperative

- Good neurological evolution.
- Postoperative CT scan highlighted evacuation of intraparenchymatous collection left P-O.

Figure no. 4. CT scan postevacuation

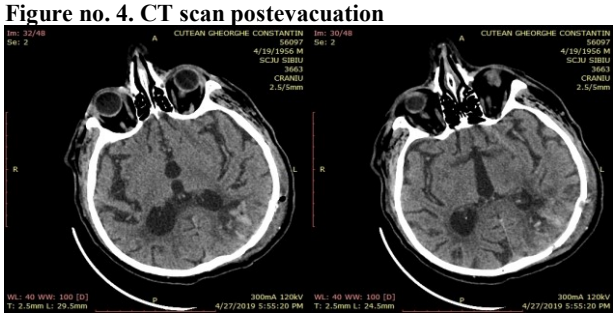
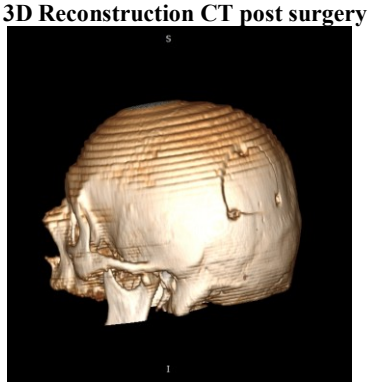


Figure no. 5. 3D Reconstruction CT post surgery



- The patient was discharged conscious, cooperative, without intracranial hypertension syndrome, without any complaints of painful nature, no motor or other deficits.
- He will be followed-up in clinical and imaging dynamics.
- With physical therapy, he will achieve the best socio-professional reinsertion.

DISCUSSIONS

This is a special case due to the unexpected complication immediately after surgery. We enumerate a few hypotheses that we thought about in relation to this contralateral mydriasis.

1. ischemia or haemorrhage in the common oculomotor core due to high blood pressure variations, excluded at CT scan postoperative and the favourable clinical evolution.
2. Claude-Bernard-Horner syndrome, excluded by the absence of other signs (enophthalmia, ptosis).
3. compression of the eye and implicitly a ciliary ganglion through positioning error that can lead to transient mydriasis.

The most important therapeutic resource in the treatment of patients with haemorrhagic stroke should be to stop the bleeding, to release compressed brain structures and minimize the secondary lesions, all applied in a shorter time sequence.

To gain time, a very important aspect is the urgent use of CT examination; the MRI is used less frequently, because by this examination the blood is hard to observe in the early hours, and the duration of the investigation is longer.

Due to our experience, focal cerebral lesions, after removal of the causal factor as fast as possible, often have a spectacular evolution, with rapid improvement in neurological status.

CONCLUSIONS

- High blood pressure is the most important risk factor in producing spontaneous intracerebral hemorrhage. In our case, the association of several factors such as high blood pressure, advanced age, obesity, dyslipidemia made the risk of bleeding even greater.
- The contralateral midriasis has been shown to be transient, most probably produced by compression on the eye after defectuous positioning.
- CT scan represents the most important investigation procedure that has the ability to highlight an intracranial blood collection.
- Neurosurgical treatment is not always necessary, but it can be a real success when the patient's age, location, hematoma size, and mass effect require this type of intervention. It has benefits if done within the first 12 hours of the onset of bleeding.

REFERENCES

1. Săceleanu V, Pereanu, M. Neurochirurgie clinică. Sibiu: Editura Universităţii „Lucian Blaga" din Sibiu; 2014.
2. Danil A. Neurochirurgie: curs pentru rezidenţi. Bucureşti: Editura Didactică şi Pedagogică; 2005.
3. Hinojosa AQ, et al. Schmidek & Sweet Operative Neurosurgical Tehniques. Indications, Methods and Results. Sixth Edition; 2012.
4. Florian I, Poeata I. Tratat de Neurochirurgie. Bucuresti: Editura Academiei Romane; 2014.
5. Greenberg MS. Handbook of Neurosurgery. Eighth Edition; 2016.
6. Arsene D. Neuropatologie. Bucureşti: Editura Didactică şi Pedagogică; 2002.
7. Feldman E. Intracerebral Hemorrhage. Armonk, NY; 1994.
8. Săceleanu V. Teză de doctorat. Opţiuni terapeutice şi strategice de reinserţie socială în hemoragia intracerebrală primară supratentorială; 2012.
9. Saceleanu V, et al. Cerebral Vascular Malformation - an incidental discovery after a traumatic brain injury. Acta Medica Transilvanica. 2017;22(3):31-33.
10. Ciurea A. Tratat de Neurochirurgie. Bucureşti: Editura Medicală; 2011.
11. Hua Y, Keep RF, Gu Y, Xi G. Thrombin and brain recovery after intracerebral hemorrhage. Stroke; 2009.
12. https://www.stroke.org/understand-stroke/what-is-stroke/hemorrhagic-stroke/. Accessed on 12.04.2019.
13. https://www.health.harvard.edu/a_to_z/hemorrhagic-stroke-a-to-z. Accessed on 12.04.2019.