CHRONIC KIDNEY DISEASE AND HYPERTENSION AS CAUSE OF INTRAVENTRICULAR HEMORRHAGE IN A YOUNG PATIENT. CLINICAL CASE

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Keywords: *intraventricular hemorrhage, hypertension, hydrocephalus, chronic kidney disease*

Abstract: *We present the case of a 32-year-old man, smoker, with medical history of chronic kidney disease on dialysis for 5 years, renovascular hypertension, lacunar ischemic stroke one year ago, left hemiparesis predominantly facially and brachially, hydrocephalus, with a suddenly onset of headache, nausea, vomiting, confusion, an hour previous presentation, while on dialysis. Neurological examination shows gait impairment, rotator nystagmus on right gaze, right hemiparesis (pronator sign), normal osteotendinous reflexes, bilateral Babinski sign, normal language, disoriented in time and space, confused. CT scan revealed third ventricle hemorrhage with normotensive hydrocephalus and no arteriovenous malformation was found on angio CT. After medication and neurosurgical treatment, the outcome was good.*

INTRODUCTION

Brain hemorrhage is the most fatal form of stroke and has the highest morbidity of any stroke subtype. Intraventricular extension of hemorrhage (IVH) is a particularly poor prognostic sign, with expected mortality between 50% and 80%.(1) IVH is a significant and independent contributor to morbidity and mortality, yet therapy directed at ameliorating intraventricular clot has been limited. Conventional therapy centres on managing hypertension and intracranial pressure while correcting coagulopathy and avoiding complications such as rebleeding and hydrocephalus. Surgical therapy alone has not changed the natural history of the disease significantly. However, fibrinolysis in combination with extraventricular drainage shows promise as a technique to reduce intraventricular clot volume and to manage the concomitant complications of IVH.

There are many studies suggesting that chronic kidney disease (CKD) has a high risk of stroke, especially ischemic stroke.(1,2) There are few studies that specifically examined the association of CKD with stroke, but demonstrated that association with primary intracerebral hemorrhage (ICH) is stronger than ischemic stroke.(3,4) Microangiopathy of small vessels is the most powerful of the many mechanisms underlying cerebrovascular disease associated with CKD and consists in inducing endothelial permeability due to high volume blood flow to these small arteries (brain and kidneys).(5) CKD has been linked to a higher prevalence of microangiopathy in MRI, including lacunar and cerebral infarctions, white matter hyperdensity, volume of white matter, than in patients without symptomatic cerebrovascular disease and those with ischemic stroke.(6)

CASE REPORT

Young man, 32 years old, smoker, with medical history of chronic kidney disease on dialysis for 5 years, renovascular hypertension, lacunar ischemic stroke one year ago, left hemiparesis predominantly facially and brachially, hydrocephalus, with a suddenly onset of headache, nausea, vomiting, confusion, an hour previous presentation, while on dialysis.

From family medical history, we learnt that his father died suddenly at the age of 64, from ischemic stroke.

Physical examination upon admission revealed:

- General altered, conscious, Glasgow Coma State (GCS) Score - 13 points, confused, disoriented in time and space;
- Rhythmic cardiac noises, atrioventricular (AV) = 65b', Blood pressure (BP) = 240/130mm Hg, without carotid bruises, peripheral arterial pulses present bilaterally;
- Spontaneous spasms without sphincter incontinence, oliguria

At the *neurological examination*:

- Headache, vomiting;
- Orthostation and gait impossible to accomplish;
- Rotating nystagmus looking to the right;
- Positive pronator drift;
- Muscle strength force (MSF) diminished at the upper 4/5 according to the Medical Research Council (MRC)
- Osteotendinous reflex present, symmetric plantar cutaneous reflexes: Babinski present bilateral
- Fluent language

Paraclinical examinations:

Laboratory investigations revealed: leukocytes: 11990/mm^3, hemoglobin = 9,5 g%; Hematocrit = 28,7%; Platelets = 124000/mm^3, erythrocyte sedimentation rate (ESR)= 3mm/h; Fibrinogen =306,8 mg/dl; C reactive protein (CRP) =15, positive; glycemia =89 mg%; urea=104 mg%, creatinine =13,47 mg%, Aspartate Aminotransferase (ASAT) =12 ui; Alanine transaminase (ALAT) =11 mg%, Cholesterol total = 166mg%, High-density lipoprotein (HDL)=32 mg%, low-density lipoprotein (LDL)=107 mg % Triglycerides=135mg%, ionogram: Na =125 mmoli/l, K = 4,59mmoli /l; Amylasemia =97 ui; total bilirubin 0,60mg%; prothrombin time (PT) = 14 sec; activated partial thromboplastin time aPTT=34,3 sec; international normalized ratio (INR)=1,15

EKG = sinus rhythm, AV=65 per minute, normal.

CT scan = revealed third ventricle hemorrhage with

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Article received on 30.05.2017 and accepted for publication on 25.08.2017
ACTA MEDICA TRANSILVANICA September 2017;22(3):38-40

normotensive hydrocephalus; diffuse cerebral edema; periventricular lacunes and in basal nuclei (figure no.1).

Angio CT = excluded arterioid venous malformation (figure no.2.).

Figure no. 1. CT scan

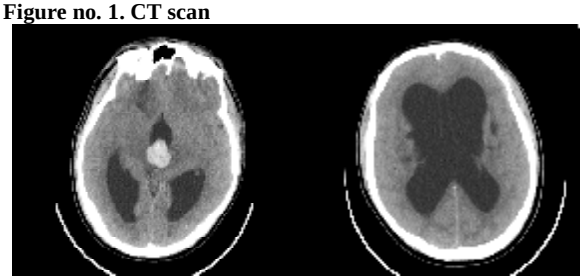
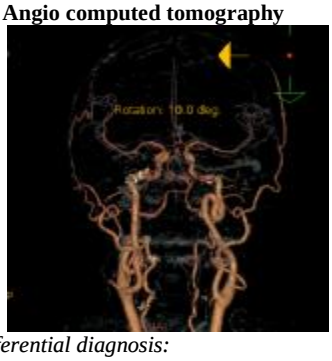


Figure no. 2. Angio computed tomography

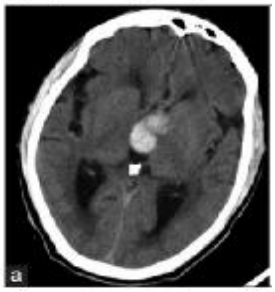


Differential diagnosis:

Secondary IVH is much more common than primary IVH.(12) The most common causes of primary intraventricular haemorrhage in adults include: hypertension, vascular malformations (aneurysm).(7)

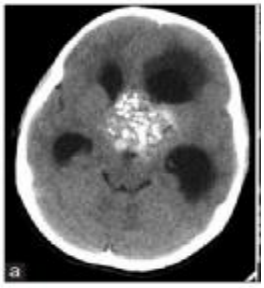
- Hypertension
- Vascular malformation:
  - anevrysm, developing within the distal lenticulostriate or choroidal, aneurysms of the anterior communicating artery, posterior inferior cerebellar artery, or basilar tip rupture into the ventricles without other obvious subarachnoid hemorrhage. 20 to 50% can remain cryptogenic.
  - arteriovenous malformation,
  - subeprndymal cavernom malformation. Third ventricular cavernomas are even rare, only 25 cases have been reported in the literature.(8) We report two more cases of third ventricular cavernomas (figure no. 3).

Figure no. 3. Cavernoma



- Anticoagulation therapy/coagulopathy
- Intraventricular tumours: ependymoma, choroid plexux/intraventraventricular metastases, adjacent parenchymal tumours (glioblastoma). Third ventricular cavernomas can present signs and symptoms of any third ventricular tumours, however they usually present symptoms of hydrocephalus.(8) Presentation as intraventriuclar hemorrhage is uncommon, and only three cases have been reported.(8) Our first patient presented with hemorrhage, and second with hydrocephalus (figure no. 4).

Figure no. 4. Intraventricular tumour



- Coagulopathies, pituitary apoplexy, sympathomimetic abuse, vasculitis, or fibromuscular dysplasia can also be attributed to IVH.

Secondary causes of intraventricular haemorrhage include:(7)

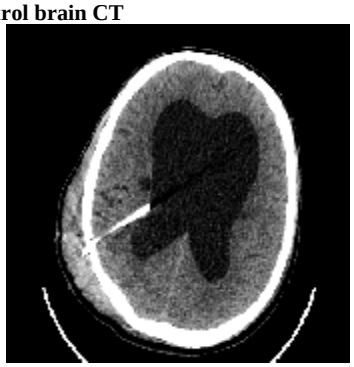
- Other intracerebral haemorrhages: hypertensive haemorrhage, especially basal ganglia haemorrhage, putamen, thalamus, cerebelar, lobar haemorrhage
- Subarahnoid haemorrahage
- Trauma

Evolution:

During admission, the general condition was maintained, altered with confusion and drowsiness. The treatment with cerebral, antihypertensive, neurotrophic, haemostatic, vitamins hydroelectrolytic rebalancing has been established since admission. Repeated brain CT and angio CT, excluded a arteriovenous malformation (AVM) and transfer to the Neurosurgery ward of the Sibiu County Clinical Hospital for surgical intervention. Ventriculo-peritoneal drainage was practiced. The evolution was favourable evolution, conscious patient, GCS score 15 points, cooperative, no neurological focal signs, temporo-spatial orientation, plague in course of healing.

Craniocerebral CT 10 days later, post-ventricular haemorrhage was in remission, ventricular catheter with right ventricular end (figure no. 3).

Figure no. 5. Control brain CT



Treatment and prognosis

The main methods of treatment of intraventricular haemorrhage can be divided into two: (9) treatment of the cause of haemorrhage (e.g. aneurysm, AVM); treatment of obstructive hydrocephalus; intraventricular thrombolysis.(10)

DISCUSSIONS

IVH usually contributes to morbidity in one of the following ways. Firstly, the clot itself can block the reduced flow of cerebrospinal fluid, leading to hydrocephalus and subsequent death, if not treated properly. Secondly, the degradation of blood products initiates an inflammatory response that can obstruct arachnoid granules, resulting in delayed hydrocephalus. After confirming the presence of IVH with computerized computed tomography of the head, other neuroimaging tools are essential to define the secondary cause of IVH.(11)

Although ventriculostomy appears to be effective in controlling intracranial pressure (ICP), this technique does little to reduce morbidity and does not address the inflammatory process. The severity of communicating hydrocephalus appears to be related to IVH volume.(6) To address the mechanical and biochemical factors that likely contribute to brain tissue injury, thrombolytic-mediated removal of clot emerged as a pragmatic solution to facilitate blood clot removal and thereby prevent hydrocephalus and inflammation.

The later may merely require careful monitoring of clinical state and serial CT brain to assess for ventricular size, or may require ventricular drain placement. A number of patients will go on to require permanent cerebrospinal fluid (CSF) diversion (ventricular pressure shunt).(11)

The 2007 American Heart Association/American Stroke Association guidelines recommend treating systolic blood pressure (SBP) greater than 180 mm Hg or diastolic blood pressure greater than 105 mm Hg.(12) Our formal meta-analysis found a significant association between eGFR <60ml/min/1,73m2 and increased incident stroke across various populations.(13) Primary IVH is rare, with secondary IVH being much more common.

Spontaneous intraventricular haemorrhage in a young patient is extremely rare and with severe prognosis. We do not know the etiology of kidney disease in our patient.

ICH is associated with substantial morbidity and mortality, with predilection for the black race, requiring a better understanding of its physiopathological mechanism. In CKD, the change in glomerular filtration rate (GFR) is significantly related to the higher intracerebral haemorrhage (ICH), especially among black patients. As such, low GFR may be a marker or potential risk potential for intracerebral hemorrhage or cerebral microembolies. Therefore, further investigation of GFR is needed.

Short-term prognosis depends on the severity of stroke and on long term, on the hemodynamic status.

CONCLUSIONS

The particularity of the case lies is that the intraventricular localization is atypical for hypertensive cerebral hemorrhage, and in a young patient. Intracerebral hemorrhage is more likely due to arteriovenous malformations than to hypertension. Baseline CKD on the basis of easily obtainable serum creatinine–based eGFR formula is significantly related to higher presence ICH.

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