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Post-traumatic epileptic seizures revealing hydrocephalus

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Keywords
cerebrospinal fluid,
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head trauma,
hydrocephalus,
ventriculo-peritoneal shunt

ABSTRACT

Background: Hydrocephalus is one of the late complications of head injury. More often, post-traumatic epileptic seizures are due to intracranial hematomas and brain contusion. Hydrocephalus is rarely reported in this clinical registry. The literature is not very rich in cases of epileptic seizures linked to posttraumatic hydrocephalus.

Objective: We report the results of our study, the aims of which were to establish the epidemiology of post-traumatic hydrocephalus in our practice, to determine the mechanisms of trauma onset, to describe the initial traumatic lesions and to highlight the timing and pathophysiological mechanism of epileptic seizures in post-traumatic hydrocephalus.

Materials and methods: We conducted a descriptive, retrospective study over a period of 4 years at the Neurosurgery Department of the Gabriel Touré University Hospital in Bamako. We included patients of both genders and all ages who had at least one seizure after head injury and in whom the CT scan performed during the investigation of the seizure showed hydrocephalus.

Results: Between 1 January 2020 and 31 December 2023, out of 1024 cases of traumatic brain injury (TBI), hydrocephalus was diagnosed in 37 patients (3.6%) during the investigation of epileptic seizures. All patients were male with a mean age of 27.4 years. The trauma was caused by road traffic accidents in 48.6%. The initial brain injuries were dominated by hemorrhagic contusions (29.7%), osteo-meningeal injuries caused by fracture of the frontal bone (19%) and traumatic subarachnoid haemorrhage (16.2%). The mean time to onset of the first attack was 28.3 days. Seizures were generalised in 86.4%. Sudden onset without prodrome occurred in 64.8% of patients. Tetraventricular hydrocephalus was diagnosed in 89.1% of cases. Ventriculoperitoneal shunting was performed in all patients. After 5 months of follow-up, seizures had completely resolved in 87.5% of patients despite complete cessation of antiepileptic treatment. No deaths were reported.

Conclusion: Epilepsy may be a clinical manifestation of post-traumatic hydrocephalus. Morphological imaging is essential for diagnosis. Management is based on cerebrospinal fluid drainage.

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Introduction

Head injury is a major public health problem in our developing countries. It is responsible for late complications including hydrocephalus. Most often in the torus, all post-traumatic hydrocephalus is attributed to subarachnoid hemorrhage. Hydrocephalus secondary to head trauma can occur outside of any subarachnoid hemorrhage. The etiology can be multiple with varied mechanisms. Post-traumatic hydrocephalus (PTH) occurs in a significant number of patients after head trauma and may affect the outcome of head injury (32). It is a complication of head injury and refers to excessive accumulation of cerebrospinal fluid (CSF) in the brain ventricles due to altered CSF dynamics (32). Post-traumatic hydrocephalus occurs in 0.7% to 29% of patients with head injury (8, 10, 21, 30, 38). In children, the incidence ranges from 0.07% to 1% (6, 16, 33). Posttraumatic epilepsy accounts for approximately 20% of the causes of epilepsy in the general population and 5% of all patients seen in specialized epilepsy centers (2, 26).

Objectives

The aims of this study were to establish the epidemiology of this condition in our practice, to determine the mechanisms of trauma onset, to describe the initial traumatic lesions and to highlight the delay and the physio-pathological mechanism of epileptic seizures in posttraumatic hydrocephalus.

Materials and methods

We conducted a retrospective descriptive study over 4 years, from 1 January 2020 to 31 December 2023. It took place in the Neurosurgery Department of the Gabriel Touré University Hospital Centre in Bamako. It focused on patients with head injuries. Patients of both sexes and all ages who had at least one epileptic seizure after a head injury and whose CT-scan confirmed the diagnosis of hydrocephalus were included in the study. Patients with a history of previous head injury, cranial surgery, stroke or epilepsy were excluded from the study. Data were collected from medical records and the operative report registry. SPSS version 2.0 software was used for data processing. Sociodemographic parameters, causes and mechanisms of head injury, time to onset and treatment modalities were examined.

RESULTS

From 1 January 2020 to 31 December 2023, 1024 patients with head injury were treated in the department. Of these, 37 patients had epileptic seizures, the investigation of which led to the diagnosis of hydrocephalus. This corresponds to a prevalence of 3.6% and an average of 9.3 patients per year. The mean age of the patients was 27.4 years (extremes 17 and 49 years). All the patients were men. Motorcycle mechanics were most affected (21.6%), followed by students (19%) and workers (13.5%). Table I shows the distribution of patients according to their occupation. Traffic accidents were responsible for 48.6% of head injuries, followed by falls from height (29.7%), assaults (18.9%) and accidents at work (2.7%). Traffic accidents were caused by loss of control of the motorcycle in 19% of cases (7 cases), a collision between a motorcyclist and a car in 10.8% of cases (4 patients), a pedestrian hit by a motorcyclist in 10.8% of cases (4 cases), and a car overturning with passengers in 8.1% of cases (3 patients).

Table 1: Distribution of patients according their occupations

Occupations	Number	Frequency (%)
Motorcycle Mechanic	8	21,6
Student	7	19
worker	5	13,5
Farmer	4	10,8
School pupil	4	10,8
Military	4	10,8
Male nurse	2	5,4
Trader	3	8,1
Total	37	100

Table 2. Distribution of patients by mechanism of trauma brain injury

Head injury mechanisms		Effective	Frequency (%)
Public road accidents	Motorcycle fall	7	19
	Motorcyclist-car collision	4	10,8
	Pedestrian hit by motorcyclist	4	10,8
	Overturned car with passengers	3	8,1

Fall from a great height	Fall from scaffolding	5	13,5
	Falling tree	3	8,1
	Fall down stairs	2	5,4
	Fall from ladder	1	2,7
Assaults and battery	with a stick	3	8,1
	with stones	1	2,7
	with an iron bar	1	2,7
	with a gas cylinder	1	2,7
	with a stepladder	1	2,7
Work accident	receiving a bag of coal on the head	1	2,7
Total		37	100

Falls from heights were due to falls from scaffolding in 5 patients (13.5%), from trees 8.1% (3 cases), stairs 5.4% (2 cases) and ladders in 2.7% (1 case). Assaults were perpetrated using blunt objects such as sticks in 3 cases (8.1%), stones, iron bars, gas canisters and stepladders in one case each (2.7%). The only accident at work was due to a head impact with a bag of coal. The mechanisms of head trauma are detailed in Table II. Clinically, 29 patients (78.4%) presented with an initial loss of consciousness of short duration. On admission to the emergency department, 19 patients had a Glasgow score of 15, six patients (16.2%) had a Glasgow score of 13, and two other patients had a Glasgow score of 12. There were two cases of non-febrile meningeal syndrome. None of the patients had pupillary abnormalities. None of the patients had focal motor deficits. Initial brain computed tomography (CT) showed edemato-hemorrhagic contusion in 11 patients (29.7%) (Figure 1) and osteomeningeal breach due to fracture of the anterior skull base associated with pneumocephalus in 7 cases (19%) (Figure 2).

Traumatic subarachnoid hemorrhage was found in 6 patients (16.2%) (Figure 3). CT scans were normal in 35.1% of cases (13 patients). Within ten days of the trauma, 3 patients (8.1%) had developed infectious meningitis, with hyperthermia, headache associated with stiff neck and vomiting in one patient. Analysis of the cerebrospinal fluid (CSF) after lumbar puncture failed to isolate any germs (bacteria). The mean time to onset of the first seizure was 28.3 days, with extremes of 17 and 41 days after head injury. The number of seizures prior to diagnosis was known in 15 patients. There was one seizure in 4 patients (10.8%), 3 seizures in 2 patients (5.4%) and 6 to 8 seizures in 9 patients (24.3%).

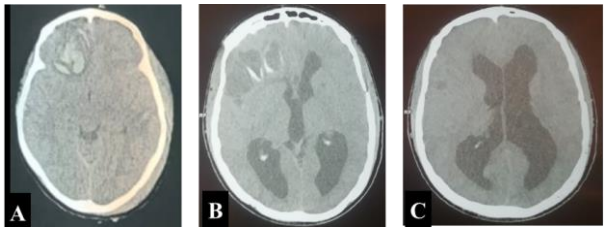

Figure 1. Evolution of brain contusion towards hydrocephalus on brain CT-Scan:

A. Post-traumatic right frontal edemato-hemorrhagic contusion.

B. Dilatation of the lateral ventricles and the 3rd ventricle and the stigmata of the right frontal contusion.

C. Periventricular hypodensity in the frontal horns (trans-ependymal suffusion).

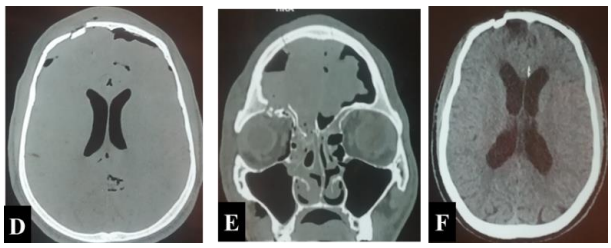

Figure 2. Occurrence of hydrocephalus on an osteomeningeal gap on brain CT-Scan:

D. Right frontal fracture with diffuse pneumocephalus extending to the lateral ventricles.

E. Coronal reconstruction, bony window, diffuse pneumocephalus reaching the base of the skull.

F. Ventricular dilatation associated with peri-ventricular hypodensity of the frontal horns (trans-ependymal suffusion).

Seizures were generalized tonic-clonic in 86.4% of cases (32 patients). They had a sudden onset without prodrome in 24 patients (64.8%). They were preceded by headaches in 8 patients (21.6%). Five patients (13.6%) had secondary partial generalized seizures. Clinical signs associated with seizures were progressive. They included intermittent headaches of moderate intensity in 13 cases (35.1%), vomiting in 8 patients (21.6%) and slowed thinking in 3 cases (8.1%). One patient (2.7%) presented gait disorders,

with small steps and widening of the weight-bearing polygon.

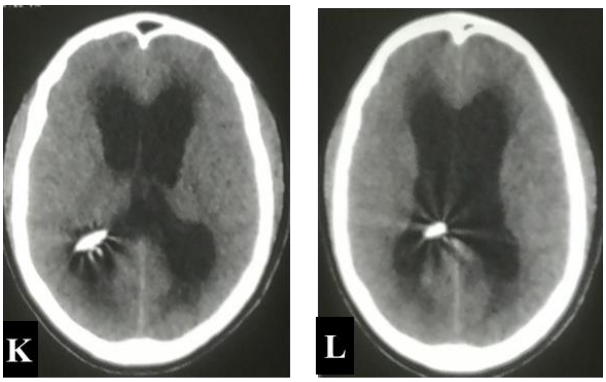

Figure 3. Early postoperative monitoring of hydrocephalus due to traumatic meningeal hemorrhage on cerebral CT-scan:
K. Presence of the catheter in the occipital horn of the right lateral ventricle.
L. Visualisation of the tip of the catheter at the level of the ventricular carrefour not far from the septum pellicidum.

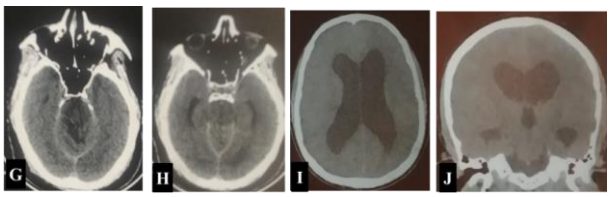

Figure 4.

Fundus examinations were performed in 7 patients (19%). Papilledema was observed in 6 patients (16.2%). An electroencephalogram (EEG) was performed in 11 patients (29.7%). It revealed generalized epileptiform abnormalities in 8 patients (21.6%) and temporal seizures in 2 patients (5.4%). It revealed active hydrocephalus in the form of significant ventricular dilatation associated with diffuse periventricular hypodensity reflecting transependymal suffusion in all patients (figures 1-C; 2-F). Hydrocephalus was tri-ventricular in 4 patients (10.8%) and tetra-ventricular in the remaining 33 (89.1%). Prior to neurosurgical treatment, 29 patients (78.4%) had received oral antiepileptic therapy. These included sodium valproate in 14 patients, phenobarbital in 9 and Levetiracetam in 6. Treatment was initiated on the advice of neurologists. Six patients (16.2%) were treated by traditional medicine.

This was a non-medical treatment consisting of drinking herbal teas and decoctions of medicinal plant leaves or barks. Two patients (5.4%) received no regular treatment. The mean time from diagnosis of hydrocephalus to surgical treatment was 7.3 days, with extremes of 2 and 16 days. Surgery involved ventriculo-peritoneal diversion of cerebrospinal fluid using a medium-pressure valve in all patients (Figure 4-K; 4-L). Concurrently with surgery, patients received a short course of corticosteroids for 3 to 7 days immediately after surgery. Anti-epileptic treatment was gradually reduced over 10 days in 11 patients (37.9%). After 3 weeks, antiepileptic therapy was discontinued in 22 patients (75.8% of cases). Two months later, three patients (8.1%) reported a recurrence of seizures. In one of them, the seizures were partial, and in the other two, they were generalized. They were put back on sodium valproate. After 3 months' follow- up, one patient showed significant ideomotor slowing (2.7%). Radiological studies showed a recurrence of hydrocephalus due to valve dysfunction. Revision surgery was performed. After 5 months' follow-up, 13 patients were lost to follow-up (35.1%), one patient still had ideomotor slowing and 2 patients were still on antiepileptic monotherapy. Progression was favourable in 21 patients (87.5%), characterized by a complete absence of seizures despite complete cessation of antiepileptic therapy. No deaths were reported.

Discussion

General and epidemiological data

Post-traumatic hydrocephalus has been known since Dandy's report in 1914 (21). It is a sequela of head trauma (32). It occurs in a significant number of patients following head trauma and may influence the outcome of head trauma (32). Its incidence ranges from 0.7 to 29% (8, 10, 21, 30, 38). This complication is characterised by a progressive accumulation of cerebrospinal fluid (CSF) in the ventricular cavities of the brain, which compresses the neurological structures within the skull (21, 38).

Pathophysiology

Head trauma may be responsible for the disruption of the normal dynamic balance between CSF production and absorption (29, 34). Clot formation following intraventricular or subarachnoid hemorrhage leads to obstruction of the CSF pathway, causing acute hydrocephalus (9, 29). This subarachnoid hemorrhage may also be responsible for the formation of adhesions in the basal cisterns

and inflammation of the arachnoid villi, leading to impaired CSF absorption and the development of chronic hydrocephalus (34). Sixteen point two percent of patients in our series presented with traumatic subarachnoid hemorrhage. As with subarachnoid hemorrhage, meningitis causes inflammation of the arachnoid villi, which reduces CSF absorption and leads to fibrosis (1). Post-traumatic meningitis affected 8.1% of patients in our cohort. The pathophysiological mechanisms of epileptic seizures associated with hydrocephalus are complex and multifactorial. Intraventricular hyperpressure due to CSF accumulation leads to dilatation of the cerebral ventricles. This causes compression and stretching of the surrounding brain tissue.

This disturbance can lead to desynchronization of the brain's electrical activity, favouring the triggering of epileptic seizures. Increased intracranial pressure can also reduce cerebral blood flow, leading to local ischemia. Lack of oxygen and nutrients can damage neurons and disrupt ionic balance, increasing the risk of seizures. Transependymal suffusion, the diffusion of cerebrospinal fluid through the ependyma of the ventricles, is responsible for inflammation of the brain tissue and the formation of gliosis. These changes can alter neural networks and create an environment conducive to epileptic seizures. CSF imbalances can also affect the concentration and dynamics of neurotransmitters such as glutamate and GABA, which play a key role in neuronal excitability. Too much excitation or too little inhibition can trigger seizures (7, 24, 28, 40,). Neuro-inflammatory pathways and neurochemical imbalances play a crucial role in the development and progression of this pathology.

Following head trauma, activation of glial cells (microglia and astrocytes) leads to the release of pro-inflammatory cytokines such as TNF-α, IL-1β and IL-6. This inflammatory response can lead to disruption of normal brain function and contribute to CSF accumulation. The resulting inflammatory response could alter the permeability of the blood-brain barrier. This allows pro-inflammatory molecules and immune cells to enter brain tissue, exacerbating brain damage and influencing CSF production and circulation. Activation of inflammatory signalling pathways such as NF-κB (nuclear factor kappa B) and MAPK (mitogen-activated protein kinase) can influence cytokine production and the cellular response to trauma. After head trauma, neurotransmitter levels can be disrupted. Excess glutamate can lead to excitotoxicity. The presence of inflammatory proteins or changes in ependymal cells can disrupt CSF metabolism (5, 19, 25, 32).

Clinical presentation

Whether or not associated with head trauma, hydrocephalus in its acute form may manifest as intracranial hypertension (ICH). This results in the onset of headaches of increasing intensity, most commonly in the morning, accompanied by light and jerky vomiting which may (temporarily) relieve the patient, and visual disturbances ranging from a decrease in visual acuity to blindness due to optic atrophy (4, 21, 22 ,41). In the chronic form, the clinical manifestations are those of the Adams-Hakim triad, characterized by progressive cognitive decline, gait disturbances consisting of short steps, widening of the supporting polygon and urinary syndrome (3, 12, 18, 21). The occurrence of epileptic seizures in hydrocephalus is not common. The few articles reported on the subject in a traumatic context are clinical cases (3). Our series is one of the rarest with such a large cohort. In addition to seizures, 56.7% of patients in our study presented with signs of ICH. These were headache (35.1%) associated with vomiting (21.6%). The Adams-Hakim triad was observed in one patient. According to the authors, this clinical manifestation is most often secondary to subarachnoid hemorrhage (9, 29). On the other hand, this does not explain the presence of the same signs in patients presenting with post-meningitic hydrocephalus due to traumatic osteomeningeal rupture.

Treatment options

CSF diversion is the treatment of choice for hydrocephalus regardless of the cause. In our study, ventriculo-peritoneal shunt (VPS) was performed in all patients. It consisted of connecting the cerebral ventricular system to the peritoneal cavity by means of a medium pressure valve. Some authors had proposed external ventricular drainage of CSF for the treatment of acute hydrocephalus (36). This approach is indicated for acute hydrocephalus associated with ventricular hemorrhage or active infectious meningitis. Authors agree on the need for

CSF diversion in post-traumatic hydrocephalus (13, 14, 27, 31). Ventriculo-peritoneal shunt diversion remains the surgical technique of choice because of the reduced compliance due to the condition and the lack of CSF resorption (6, 20, 35). In our series, only fixed and medium pressure valves were used. The use of other valve types, namely programmable valves or the flow-regulated valve system, has been reported (17, 41). The success of surgical treatment is assessed by regression or complete resolution of signs.

A gradual normalization of intracranial pressure would eliminate the compression of neurological structures inside the skull. This improvement led to the total and definitive cessation of antiepileptic treatment in 75.8% of patients in our study after 3 weeks. The placement of a shunt brought a clear clinical improvement in patients with hydrocephalus who had a low Glasgow score after the trauma or post-traumatic meningitis (35, 39). The inflammation of the periventricular cerebral cortex by the transependymal suffusion of the CSF also involved in the genesis of the seizures had justified the short-term corticosteroid therapy administered to all patients in the immediate postoperative period thanks to its anti-inflammatory effect. Some complications may occur during treatment. These include corticosteroid-induced diabetes, of which we have not recorded any cases, and valve dysfunction. Valve disconnection is a mechanical failure of the bypass system. One case occurred in our cohort and required reoperation for valve revision.

The valve revision rate in the literature ranges from 17.9% to 24% (6, 35). Some authors had proposed endoscopic surgery for internal drainage of CSF in post-traumatic hydrocephalus (11, 15). The rationale for performing this endoscopic ventriculocisternostomy (ETV) was that it could restore intracranial compliance by reducing CSF stagnation in the ventricular system through an "internal shunt". A review of the literature suggested that ETV should be reserved for cases of obstruction of the midbrain aqueduct by hemorrhage of traumatic origin, or in cases of shunt dysfunction or repeated infection of the ventriculo-peritoneal drainage (15, 35, 36). This ETV may fail. A study from India reported a failure rate of 60% in the treatment of post-traumatic hydrocephalus (35). In the same series, only 37% of patients treated with VCS showed improvement compared to 73% after ventriculo-peritoneal shunt placement (35). In our cohort, all patients had undergone VPS placement with a favourable outcome. Medium-pressure valves had been the type of shunt used in all the patients in our series. According to Svedung Wettervik and al, patients with medium pressure hydrocephalus (chronic adult hydrocephalus) have a better response to ventriculo-peritoneal shunt (37).

Conclusion

Epilepsy may be the mode of expression of intracranial hypertension in patients after head trauma. Hydrocephalus may be the cause. In any case of post-traumatic seizures, morphological imaging becomes essential even if the first imaging was normal so as not to miss hydrocephalus. Effective and early treatment of this hydrocephalus can save patients from having to take antiepileptic drugs for a very long time.

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