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Case Report

Galen's Ampulla Aneurysm in Connection with a Neonatal Case

Nassima Jraifi[1]*, A Mejhed[1], F Tahiri[1], K Ettouini[1], M Zouine[1], A Oulmaati[1], K Kaddaoui[2], M Himmiche[2] and A Aggouri[2]

[1]Neonatology and Neonatal Intensive Care Department, CHU Mohmmed VI Tanger, Morocco

[2]Department of Neurosurgery, CHU Mohmmed VI Tanger, Morocco

***Corresponding Author:** Nassima Jraifi, Neonatology and Neonatal Intensive Care Department, CHU Mohmmed VI Tanger, Morocco.

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Abstract

Galen's ampulla aneurysm in newborns is often a potentially serious emergency, given the risk of significant morbidity and mortality.

We report the clinical observation of a female newborn admitted to the neonatal intensive care unit of the Mohammed VI University Hospital Tangier for a 35-week prematurity of amenorrhea associated with macrocrania.

Radiological investigations revealed a thrombosed aneurysm of the ampulla of Galen with a laminated cerebral parenchyma, lack of visualization of the cerebellum, corpus callosum and brainstem, and triventricular hydrocephalus. After a multidisciplinary consultation, we opted for a palliative treatment given the non-feasibility of surgical treatment. The evolution was marked by the death of the patient at the corrected age of 1 month and 2 weeks.

Keywords: *Galen's Ampulla Aneurysm; Neonatal Intensive Care Unit; Triventricular Hydrocephalus*

Introduction

Galen's ampulla aneurysm accounts for the majority of vascular brain malformations in newborns and infants [1-3]. About 20% of them are discovered antenatally, most often during the third trimester or in the neonatal period, in the presence of cardiac or neurological manifestations. The modes of revelation are heart failure from 0 to 6 months, hydrocephalus, macrocrania from 6 months to 2 years and delay in neurological development beyond 2 years [4]. In recent years, endovascular embolization has been the treatment of choice that has improved mortality rates and reduced morbidity in these children [5,6].

Through our observation, we report the case of a newborn admitted for a prematurity of 35 weeks of amenorrhea with macrocrania revealing a thrombosed aneurysm of the galenola of Galen.

Medical Observation

It is a female newborn, the first twin of a bichorionic, bi-amniotic twin pregnancy, the second twin is a male newborn in good health.

Our patient is from a non-consanguineous marriage, mother aged 25 with no particular pathological history and no notion of taking toxic substances, the pregnancy was carried to 35 weeks of amenorrhea with the discovery of hydrocephalus on antenatal ultrasound. The

delivery was carried out by caesarean section without any particular incident with an APGAR score at birth of 7 and 8 respectively at the first and fifth minutes.

Clinical examination at birth revealed a newborn with hypotonia, absence of ocular chasing movements, a bulging forehead, a wide anterior fontanelle, disjunction of the sutures and macrocrania with a cranial circumference of 40 cm [+97° percentile].

Figure

In view of the clinical symptoms of our patient, hospitalization in the neonatal intensive care unit and an etiological assessment were requested.

TORCH serologies: toxoplasmosis, rubella, cytomegalovirus, herpes simplex. It may also contain other neonatal diseases were negative.

The injected cerebral computed tomography (CT) showed a voluminous formation occupying the posterior cerebral fossa and extends into a tensor sup, first evoking a thrombosed aneurysm of the Galen vein, responsible for obstructive triventricular hydrocephalus, with laminated appearance of the cerebral parenchyma, absence of visualization of the cerebellum, corpus callosum and brainstem.

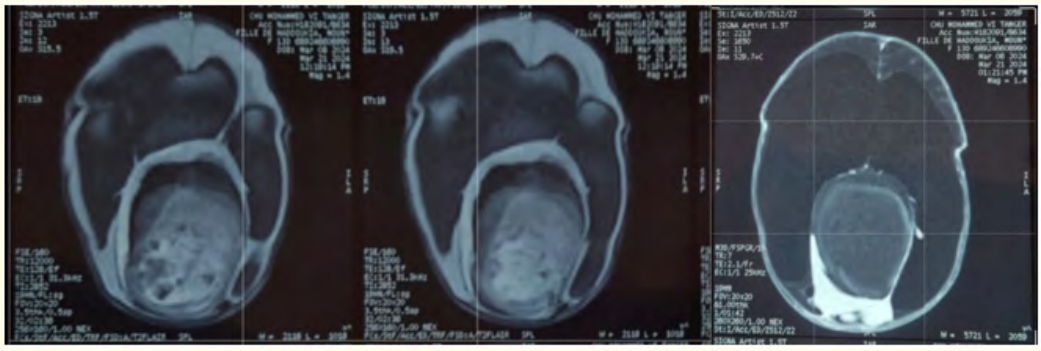

Figure 1: *Brain CT which shows a voluminous formation of the posterior cerebral fossa extending in addition to the tentorial tract, suggesting in 1st place a thrombosed aneurysm of the Galen vein.*

MRI angiography is in favor of a large thrombosed aneurysm of the Galen vein responsible for obstructive triventricular hydrocephalus, thrombosis of the right internal cerebral vein and minimal intraventricular hemorrhage with complete cerebellar agenesis.

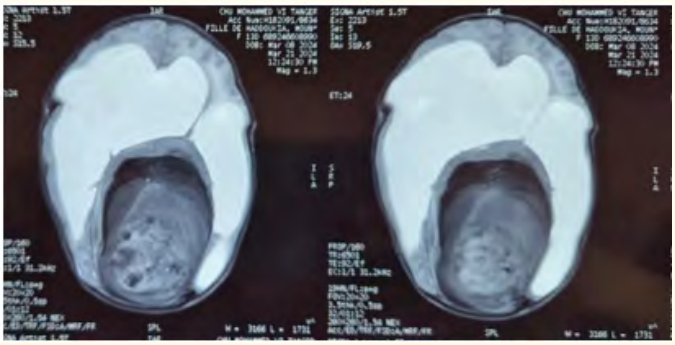

Figure 2: *Brain MRI showing a thrombosed aneurysm of the Galen ampulla with hydrocephalus, laminated appearance of the cerebral parenchyma with absence of visualization of the cerebellum, corpus callosum and brainstem.*

In order to rule out an associated malformation, a malformation assessment was carried out in parallel with cardiac ultrasound which proved to be without abnormalities, an abdominal ultrasound in favor of an uncomplicated multilithiasic gallbladder.

Therapeutic management was based on the initiation of anticoagulant treatment with LMWH at a dose of 150 iu/kg/day with anti-vitamin K and strict monitoring of the head circumference and INR.

Anticonvulsant treatment with phenobarbital was started in the event of convulsions at a dose of 3 mg/kg/day.

A multidisciplinary staff of our patient's case concluded that surgical treatment is not indicated, given the following poor prognostic factors: laminated appearance of the cerebral parenchyma, lack of visualization of the cerebellum, corpus callosum and brainstem with complete cerebellar agenesis.

During his hospitalization, we noted a progressive increase in head circumference up to 42 cm [+97° percentile] with recurrence of convulsions, an increase in the dose of anticonvulsant treatment was indicated with a dose of 5 mg/kg/day, with a follow-up brain computed tomography which objectified an increase in the size of the thrombosed aneurysm of the Galen vein measuring 76.5*92 mm VS 60*89 mm without visible bleeding.

A ventriculoperitoneal shunt was discussed with the neurosurgery team who judged that its contribution is unnecessary.

The evolution was marked by the death of our patient at the corrected age of 1 month and 2 weeks.

Discussion

Galen's ampulla aneurysm is a rare vascular malformation, associated with several arteriovenous fistulas that would explain the variability in clinical expression [7].

There have also been reported cases of spontaneous thrombosis of the Galen vein [8].

Treatment depends on the clinical manifestations. In the event of an antenatal diagnosis, it is important to look for serious factors such as heart failure or brain damage. Indeed, the presence of fetal brain damage has a very poor prognosis in terms of neurocognitive development, especially since the extent of the lesion is underestimated by imaging [9,10].

In newborns, a systemic analysis of the repercussions of LVAM is carried out by the Bicêtre neonatal score, which evaluates the cardiac, cerebral, respiratory, hepatic and renal repercussions over the first days of life.

In the case of heart failure, management includes medical treatment with ventilatory support if necessary, if it is refractory to medical treatment and in the absence of encephalomalacia lesions, embolization is indicated to reduce systemic and cerebral circulatory consequences [6,11]. In the absence of neonatal heart failure, the ideal age to start radiointerventional treatment is around 4 to 5 months, with clinical and radiological follow-up. If the cerebral parenchyma is normal or in the case of controlled hydrocephalus, there is no indication for emergency endovascular treatment. If there are encephalomalacia lesions, the neurological prognosis is very poor and palliative care with therapeutic abstention is then discussed with the parents [9].

As far as ventriculomegaly is concerned, its treatment, whether preventive or curative, is that of the arteriovenous shunt. Embolization will be performed as an emergency in the presence of: a rapid increase in head circumference, significant developmental delay, MRI signs of intraventricular hyperpressure. Ventricular shunt should not be considered at the outset, as it reverses the flow gradient along the spinal veins and leads to haemorrhage, neurological deficit or epilepsy in more than 80% of cases. It should only be considered after embolization if the clinical results of embolization are insufficient despite a reduction in the shunt.

Our observation revealed a neurological clinical manifestation of LAA which is: macrocrania, clinical convulsions with laminated brain parenchyma and cerebellar agenesis on brain imaging.

Paraclinical investigations of the newborn were normal, the laboratory work-up, the pulmonary parenchyma and the cardiac and abdominopelvic ultrasound did not reveal an associated malformation.

Faced with these poor prognosis data, palliative treatment was chosen.

Surgical treatment was not performed after consultation with the neurosurgery team.

Conclusion

Newborns with LVAM are at risk of having a delay in psychomotor development and behavioural disorders. A neonatal Bicêtre score of less than 12, symptomatic hydrocephalus, severe heart failure and encephalomalacia are factors of poor neurological prognosis.

Interventional treatment has improved the prognosis of these children, but the mortality rate remains high even if they are treated in a referral centre.

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