CLINICAL CASE

Subgaleal hematoma leads to the diagnosis of aortic coarctation

Hematoma subgaleal orienta al diagnóstico de coartación de la aorta

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DOI: https://doi.org/10.61651/rped.2024v76n2p2c1-7

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Fuente de financiamiento: no se ha obtenido ninguna financiación para la realización de este estudio.

Declaración de Conflicto de Interés: los autores declaran no tener ningún conflicto de interés.

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Fechas:
Envío: 26-Jun-2024
Aceptación: 16-Ago-2024

RESUMEN

El hematoma subgaleal (HSG) consiste en una acumulación de sangre en el espacio entre el periostio y la aponeurosis del cuero cabelludo. Se presenta el caso de una niña de 5 años con antecedentes de un traumatismo banal, sin trastornos de coagulación y con un extenso hematoma subgaleal. Se realizó un drenaje quirúrgico de la colección hemorrágica. Durante la observación en el servicio de urgencias, se observó una presión arterial persistentemente alta, con una diferencia de presión entre las extremidades superiores e inferiores. Se realizaron pruebas adicionales y se diagnosticó una coartación de la aorta (CoA). Se han descrito casos de hemorragia subaracnoidea, epistaxis y hemorragia gastrointestinal asociados a hipertensión arterial por CoA. No está claro si la hipertensión podría haber jugado un papel relevante en el sangrado de esta paciente.

Palabras clave: Hematoma subgaleal, Coartación de aorta, Hipertensión.

ABSTRACT

Subgaleal hematoma (SGH) consists of a collection of blood in the space between the periosteum and the aponeurosis of the scalp. It is characterized by an epicranial extension beyond the sutures. It predominantly appears in newborns related to birth trauma, although it has also been described in older children related to trauma and other triggers. This clinical case presents a 5-year-old girl with a history of a minor trauma, without coagulation disorders, and with an extensive subgaleal hematoma. Surgical drainage of the hemorrhagic collection was performed, with subsequent re-bleeding. During observation in the emergency department, persistently high blood pressure was noted, with a pressure difference between the upper and lower extremities. Additional tests were conducted, and a diagnosis of aortic coarctation was made. Most congenital heart defects are diagnosed in the neonatal period or even in utero, although some are overlooked and diagnosed late in childhood. Aortic coarctation (CoA) represents 5-8% of all congenital heart defects. Delay in diagnosis can lead to serious complications. Intracranial hemorrhage secondary to intracranial aneurysms, left ventricular hypertrophy, and subsequent congestive heart failure can also be observed in adult patients with undiagnosed CoA. Less common cases of epistaxis and gastrointestinal hemorrhage have also been described. It is unclear whether hypertension could have played a relevant role in the bleeding in this case.

Key messages

- *Subgaleal hematoma is a rare condition that usually occurs in newborns as a result of childbirth and in older children secondary to traumatic causes.*
- *The management of subgaleal hematoma in children should be conservative. Drainage should be reserved for complicated cases, as it may be associated with re-bleeding.*
- *The presence of hypertension in children is a warning sign and should always be confirmed, even if it does not seem related to the main reason for consultation or the patient has confounding factors such as agitation or pain, as it may be a manifestation of a serious disease.*
- *Arterial hypertension secondary to coarctation of the aorta may be associated with bleeding, and therefore a high index of suspicion should be maintained for unusual bleeding.*

Introduction

Subgaleal hematoma (SGH) occurs due to the accumulation of blood in the space between the periosteum and the aponeurosis of the scalp. It predominantly appears in newborns related to childbirth, but several cases have been described in relation to other triggers, mainly of traumatic origin or in children with coagulation disorders. It usually resolves spontaneously within several days (1).

Coarctation of the aorta (AoCo) is a congenital malformation in which there is a narrowing of the descending portion of the aorta, usually after the origin of the left subclavian artery, leading to a significant decrease in systemic blood flow and, at the same time, an increase in pressure in the left heart chambers. The severity of the clinical manifestations is related to the severity of the obstruction to flow and by the association with other cardiac malformations, which appear in up to 75% of cases. When there is a critical obstruction to anterograde blood flow, the disease manifests in the neonatal period with severe heart failure, with systemic flow being exclusively dependent on the ductus arteriosus. In milder cases, the patient may remain asymptomatic for months or years. Over time, this pathology may lead to the development of hypertrophic cardiomyopathy and heart failure (2). An increase in collateral circulation in the thoracic region may also be seen as the disease progresses. Some children present with hypertension secondary to AoCo. No case of HSG associated with AoCo hypertension has been reported to date.

Clínical Case

A 5-year-old girl was brought to the emergency department (ED) due to swelling in her forehead and eyelids. In the previous 24 hours, she had intermittent vomiting, with fluid tolerance between episodes. Her family reported that she had sustained a facial injury after falling while running two days before. The patient was born in China and was adopted at age of two. No known illnesses have been reported since adoption. Physical examination revealed diffuse swelling on the forehead and nasal root area, with a palpable soft, fluctuating, and diffuse mass extending from the nasal root to the back of the epicranial region. There were no skin lesions. The rest of the physical and neurological examination was normal. Blood pressure on arrival at the ED was 167/110 mmHg and heart rate 140 bpm. Laboratory tests showed haemoglobin 11.3 g/dL, leukocytes 11 200 cells/µL, neutrophils 8900 cells/µL, platelets

264 000 cells/µL and in the basic coagulation study INR 1.19, prothrombin time 14.4 seconds, cephalin time 39.2 seconds and fibrinogen 286 mg/dL.

Due to the described lesions, an emergency cranial MRI was performed with a rapid protocol without intravenous contrast. A diffuse, minimally asymmetric, circumferential extracranial collection was visualized, crossing cranial sutures and extending from the nape to the nasion. It had a heterogeneous signal, isointense to the cortex on T2, with hyperintense foci on T1 sequences and a very low-intensity signal on gradient echo related to bleeding at different stages of evolution (Figures 1 and 2). It had a maximum thickness of approximately 13 mm. These findings were compatible with diffuse SGH. No intracranial abnormalities were observed. Based on neurosurgery's indication, the collection was drained with a needle under sedation, evacuating approximately 50 ml of blood and applying a compressive bandage. After the procedure he was kept under observation in the ED, and elevated blood pressure figures were recorded, with peak systolic blood pressure 171 mmHg and a pressure gradient between arms and legs (right upper limb 121/59 mmHg, left upper limb 103/88 mmHg, left lower limb 107/80 mmHg and right lower limb 99/75 mmHg). No murmur was found on cardiac auscultation. Abdominal ultrasound showed a horseshoe kidney with no other abnormalities. Treatment was started with amlodipine at 0.1 mg/kg/day and later propranolol at 0.8 mg/kg/day was added. The chest X-ray showed an abnormal contour of the aorta with the "number 3" sign (Figure 3).

She was evaluated by cardiology, the echocardiogram showed a coarctation with a maximum systolic gradient of 70 mmHg with diastolic extension. Hemodynamic repercussions were associated with left ventricular dilation, with a slightly hypertrophied wall thickness. A CT scan of the aorta confirmed severe coarctation at the isthmus with a critical diameter of 2.5 mm. Dilatation of the supra-aortic trunks and marked collaterality at the expense of the intercostals and paravertebrals vessels were also observed.

In the following 36 hours, the patient developed progressive anemia with a decrease in hemoglobin from 11.3 to 6.4 g/dl and reappearance of the hematoma, so compressive bandage was maintained for 48 hours and was subsequently removed without incident. A coagulation study was completed, with determination of coagulation factors and platelet aggregation tests in which no alterations were found. She was discharged after controlling her blood pressure, and was readmitted after 3 weeks as scheduled for catheterisation with placement of an aortic stent. Subsequently, the pressure gradient improved and decreased to 18 mmHg. At discharge, antihypertensive treatment and low molecular weight heparin was maintained.

Discussion

We present the case of a girl who developed a subgaleal haematoma after a banal trauma. Following treatment of the haematoma, arterial hypertension and a AoCo were found to be the cause of the haematoma. We believe that the cranial bleeding may have been related to the arterial hypertension secondary to the vascular malformation, and we therefore consider this case to be of interest for discussion.

When there is an increase in scalp volume, it is important to consider the possibility of a caput succedaneum, a cephalohematoma, and an SGH. Caput succedaneum is a subcutaneous, extraperiosteal, serosanguinous collection that usually develops after birth and therefore appears in the immediate neonatal

period. Characteristically, it has poorly defined boundaries and in its distribution it crosses the cranial sutures. It is usually uncomplicated and resolves within a few days without treatment. In contrast, cephalohaematoma consists of subperiosteal bleeding, often associated with the presence of a skull fracture, both within and outside the neonatal period. It helps to distinguish it from the anterior one which does not cross the cranial sutures. It is usually unilateral, most frequently in the parietal region. In our patient, both entities were ruled out by the clinical presentation, as the age and history were not compatible with a caput succedaneum and the holocranial distribution meant that it could not be a cephalohaematoma.

SGH is a collection of blood located in the virtual space between the periosteum and the aponeurosis of the scalp (called galea). It is characterized by extending in the epicranial region, crossing sutures, and can extend posteriorly to the nape and anteriorly to the lower border of the orbits. It occurs due to the rupture and bleeding of emissary veins, which drain from the venous sinuses. SGH is predominantly seen in newborns related to birth trauma and should be differentiated from caput succedaneum in this context (3). However, many cases have also been described in children outside the neonatal period, related to mechanical factors (most commonly head trauma) and coagulation disorders. It is worth noting that several cases have appeared in relation to hairstyles that involve significant hair traction, such as braids (4-6).

The hematoma usually develops insidiously in the days following the trauma, as it is due to venous bleeding. When clinical suspicion of SGH arises, cranial imaging is indicated to confirm the diagnosis and rule out cranial fractures and associated brain injuries. It is also necessary to look for coagulation disorders and assess the need for transfusion support, as this space can accommodate large volumes of blood, potentially leading to complications such as anemia and even hemodynamic impact, especially in neonates. SGH typically resolves in a few weeks with conservative management, using a compressive cranial bandage to facilitate drainage. Complications such as proptosis and ophthalmoplegia due to anterior extension to the orbit, otorrhea, hyperbilirubinemia with jaundice due to hemolysis, or infection of the hematoma can occur (1,7). Prevention or treatment of complications, as well as failure of conservative management, are indications for hematoma drainage. However, drainage may be followed within hours or days by rebleeding, which can lead to the development of anaemia, as was the case in this patient.

Hypertension in children is a rare finding, although with an increasing incidence in adolescents in relation to increasing obesity. In the presence of high blood pressure outside the adolescent period, it is necessary to rule out disorders that may be the cause of secondary hypertension. The most common are renal disorders, although it may also be due to endocrinological or vascular diseases (8). Currently, most congenital heart defects are diagnosed in the neonatal period or even in utero, thanks to screening programs and prenatal ultrasounds. However, some are overlooked and diagnosed later in childhood, which is associated with more complications and a worse prognosis. In our patient's case, no health data from her early years in her birth country could be obtained. This frequently occurs with international adoptions, and for this reason, screenings are often performed to rule out certain pathologies. AoCo represents 5-8% of all congenital heart defects. When diagnosed in childhood, it generally presents with findings on physical examination, such as a pressure gradient between the upper and lower extremities >20 mmHg, weak or absent femoral pulse, or a murmur on auscultation.

Delayed diagnosis can lead to serious complications, as severe AoCo will cause aortic obstruction with lower body hypoperfusion, renal dysfunction, and metabolic acidosis. Intracranial hemorrhage secondary to intracranial aneurysms, left ventricular hypertrophy, and subsequent congestive heart failure can also be observed in adult patients with undiagnosed AoCo. Regarding the risk of bleeding in patients with hypertension secondary to undiagnosed AoCo, cases of subarachnoid hemorrhage, epistaxis, and gastrointestinal bleeding have been mainly described (10-12). These types of bleeding correspond to arteriolar hemorrhages. It is not clear whether hypertension could have played a relevant role in our patient's bleeding, since unlike the cases previously described, HSG is a low-pressure venous bleeding. In any case, it was the presence of hypertension that led us to suspect the existence of a serious underlying disorder, and blood pressure monitoring and differential pressure measurements allowed us to guide the diagnosis and definitive treatment

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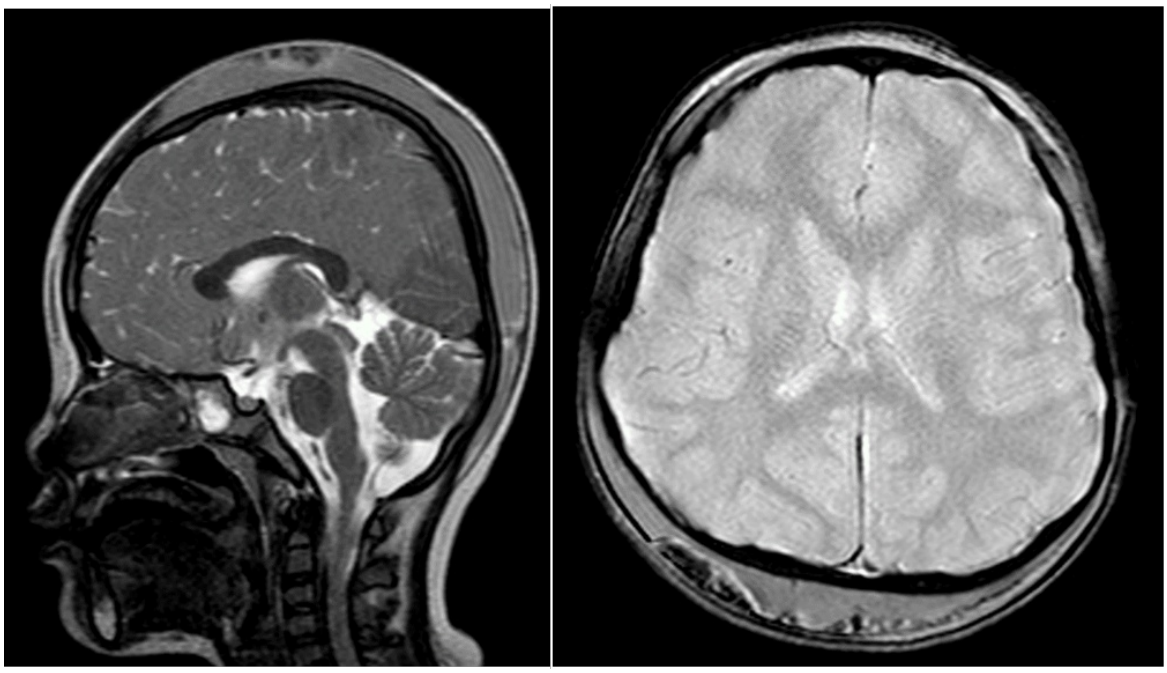

Figure 1: a) Magnetic resonance of the brain shows an extense and diffuse extracranial collection. It has a heterogeneous signal, because of the appearance of hemorrhage varies with the age of the hematoma. b) Echo gradient sequence with foci with very low signal intensity.

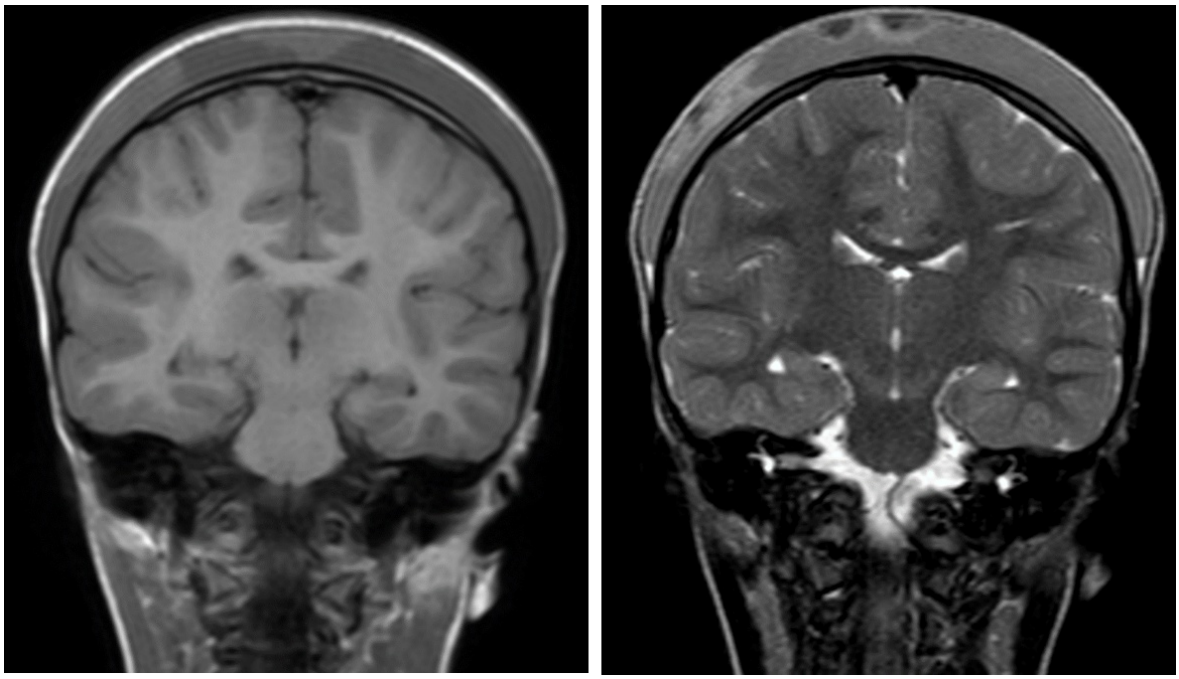

Figure 2: Magnetic resonance of the brain. Diffuse subgaleal hematoma in T1 and T2 sequences. It crosses the anterior fontanelle and the sagittal suture. It is the main characteristic that differentiates a subgaleal collection from a cephalohematoma. With this coronal projection it is impossible to confuse them.

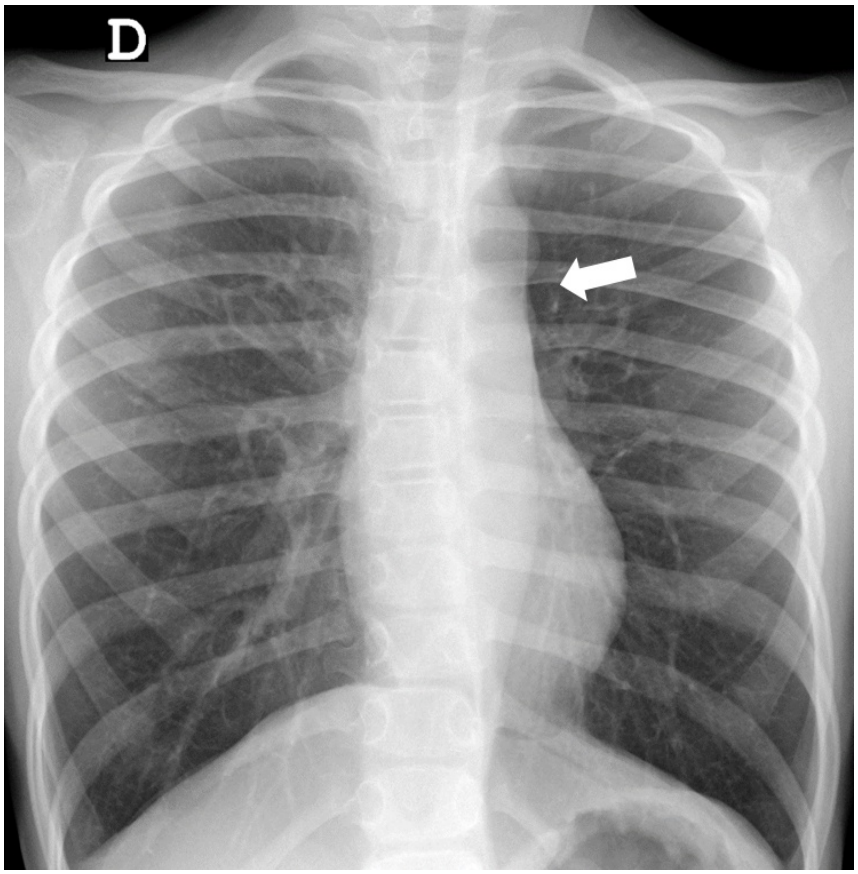

Figure 3: Plain chest radiograph. The figure 3 sign is formed by prestenotic dilatation of the aortic arch and left subclavian artery, indentation at the coarctation site and post-stenotic dilatation of the descending aorta.