ARTÍCULO ORGINAL

NEUROIMAGEN EN HEMATOMA EPIDURAL

NEUROIMAGING IN EPIDURAL HEMATOMA

NEUROIMAGEM EM HEMATOMA EPIDURAL

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Resumen

Objetivo: El hematoma epidural intracraneal (HE) es una acumulación de sangre localizada en el espacio entre la placa interna de la bóveda craneal y la capa externa de la duramadre. Como la EH es una de las complicaciones graves del traumatismo craneoencefálico, la tomografía computarizada es el examen de elección. El tratamiento ideal es la cirugía, con buen pronóstico.

Métodos: Se seleccionaron artículos de las bases de datos PubMed, Scielo y libros de neurorradiología, utilizando los siguientes términos: Neurorradiología, lesión cerebral traumática, hematoma epidural intracraneal. Se seleccionaron los trabajos desde 1977 hasta 2020.

Resultados: Se seleccionaron los artículos más citados sobre el tema de neurorradiología y EH intracraneal. A menudo se encuentra en hombres y jóvenes entre 10 y 30 años. Las causas más frecuentes de EH intracraneal son los accidentes de tráfico y las caídas. El cuadro clínico se caracteriza por dolor de cabeza, náuseas y vómitos. En la tomografía computarizada craneal, el HE suele aparecer como una colección extraaxial hiperdensa.

Conclusiones: La neuroimagen demostró ser útil en el diagnóstico, manejo y pronóstico de los pacientes con EH intracraneal.

Palabras clave: Trauma craneoencefálico. Hematoma epidural craneal. Neuroimagen.

Abstract

Background: Intracranial epidural hematoma (EH) refers to a collection of blood located between the inner table of the skull and the outer layer of the dura mater. As a potentially life-threatening complication of traumatic brain injury, EH is most accurately diagnosed using computed tomography, the imaging modality of choice. Surgical intervention is considered the ideal treatment and is generally associated with a favorable prognosis.

Methods: A literature review was conducted using articles from the PubMed and SciELO databases, as well as selected neuroradiology textbooks. The following search terms were used: neuroradiology, traumatic brain injury, and intracranial epidural hematoma. The review included works published between 1977 and 2020.

Results: The most frequently cited articles on neuroradiology and intracranial EH were selected for review. Intracranial EH occurs predominantly in males and in individuals between 10 and 30 years of age. The most common causes are traffic accidents and falls. Clinically, patients typically present with headache, nausea, and vomiting. On cranial computed tomography, EH appears as a hyperdense, extra-axial collection with well-defined margins.

Conclusions: The neuroimaging exam proved to be useful in the diagnosis, management and prognosis of patients with intracranial EH.

Keywords: Traumatic brain injury. Cranial epidural hematoma. Neuroimaging.

Resumo

Objetivo: O hematoma epidural (HE) intracraniano é um acúmulo de sangue localizado no espaço entre a placa interna da calota craniana e a camada externa da dura-máter. Como a HE é uma das complicações graves do traumatismo cranioencefálico, a tomografia computadorizada é o exame de escolha. O tratamento ideal é cirúrgico, com bom prognóstico.

Metodos: Foram selecionados artigos das bases de dados PubMed e Scielo e livros de neurorradiologia, utilizando os seguintes termos: Neuroradiology, traumatismo cranioencefálico, hematoma epidural intracraniano. Os trabalhos foram selecionados no período de 1977 a 2020.

Resultados: Foram selecionados os artigos mais citados sobre o tema neurorradiologia e HE intracraniana. Frequentemente encontrada em homens e jovens entre 10 e 30 anos. As causas mais comuns de EH intracraniana são acidentes de trânsito e quedas. O quadro clínico é caracterizado por cefaleia, náuseas e vômitos. Na tomografia computadorizada de crânio, a HE geralmente se apresenta como uma coleção extra-axial hiperdensa.

Conclusões: O exame de neuroimagem mostrou-se útil no diagnóstico, manejo e prognóstico de pacientes com HE intracraniana.

Palavras-chave: Trauma craniocerebral. Hematoma epidural craniano. Neuroimagem.

Introduction

Epidural hematoma (EH) is a blood collection located in the space between the inner plate of the skullcap and the outer layer of the dura mater. Being considered a serious complication of traumatic brain injury (TBI) [1]. Cranial computed tomography (CT) examination is useful for diagnosis and management [2]. Being neurosurgical intervention considered the definitive treatment [3].

Methods

A clinical case is presented involving a 6-year-old male patient with refractory focal epilepsy who developed super-refractory status epilepticus. In addition, a non-systematic review of the literature is conducted.

Study Design

This is a systematic review, based on the PRISMA method and a literature review with synthesis of evidence. A systematic review of the literature through electronic searches in the databases of Pubmed and Scielo, and neurosurgery books in january of 2021, using the following descriptors: “Neuroradiology”, “traumatic brain injury” and “intracranial epidural hematoma”. The search term was identified in titles, keywords or abstracts of the articles.

Population data

The population selection was based on articles that addressed adult individuals who were diagnosed with EH. No restriction on sex, race, color or socioeconomic status.

Inclusion criteria

Inclusion criteria were articles that presented evidence of EH in adults. Moreover, other inclusion criteria were to select studies published from 1977 to 2020, select qualitative and quantitative primary surveys (such as randomized clinical trial and cohort study) and secondary surveys were selected (with meta-analyzes); which were available online in full of article format in english.

Exclusion criteria

Exclusion criteria were adopted articles of narrative and integrative review, monographs and theses, and all works published before 1977 were also dismissed.

Results

Incidence and clinical history

It occurs in less than 2% of patients with TBI and between 5% to 15% of severe TBI [1,4,5]. It is more common in males and young people between 10 and 30 years old, and less frequent in children and the elderly [6,7,8].
Clinical history is characterized by headache, nausea, vomiting and focal neurological deficit. Other manifestations of EH are lethargy, anisocoria and contralateral hemiparesis. The lucid interval with late loss of consciousness occurs in only half of the patients with EH 9,10. The lucid interval can be characterized clinically by a lucid interval, in which the patient is totally asymptomatic or not severe. It corresponds to the time between the traumatic injury and the accumulation of blood in the epidural space, before exerting a mass effect on the adjacent brain area [11].

Etiology and location

Traffic accidents, falls and robberies are the most frequent causes of EH [5,6,7,12,13]. Lesion of the middle meningeal artery (MMA) is responsible for the majority of EHs (85% to 95% of cases) [14,15,16,17,18]. However, they can result from lacerations of the dural venous sinuses or diploic veins, meningeal vein or fracture line bleeding 19. The occurrence of EH has also been reported in cases of supratentorial decompressive craniectomy after drainage of subdural and intracerebral hematoma [20]. Causes of non-traumatic EHs are coagulopathies, thrombolysis, vascular malformations, neoplasms, epidural anesthesia, Paget's disease of the skull and ventriculo-peritoneal shunt [21].
Most lacerations are arterial (85%), in branches of the MMA, due to temporal or temporoparietal fracture [5,17,22]. Locations are unilateral (95%), supratentorial (90% to 95%) and in the temporoparietal regions (66%), with location in the posterior fossa occurring in 0.3% of all TBI and all 2.7% to 11% EHs [23,24,25].

EHs of venous origin are less common than arterial ones, occurring between 5% to 10% of all EH [26], being more frequent in children and occurring due to occipital, sphenoid or parietal fractures that damage the dural venous sinus. When located in the posterior fossa, it results from injury to the transverse or sigmoid sinuses, and in the middle cerebral fossa, due to injury to the sphenoparietal sinus. It rarely occurs in the vertex due to an injury to the superior sagittal sinus [27]. Therefore, an EH of venous origin should be suspected, when a fracture trace occurs through the mentioned dural sinuses [26].

Neuroimaging

A simple skull radiography, when performed, may reveal a cranial fracture, which is important for the indication of cranial CT [28]. EH is often associated with cranial fracture (91% adults, and 75% in children) [29,30]. The absence of a fracture line on plain skull radiography does not exclude the presence of an EH.

CT is the exam of choice in urgency 2,31,32. Mortality was 29% to 31% before the advent of CT, decreasing to 12% to 19% after the routine use of CT for diagnostic purposes, especially in emergency rooms.

At CT, EH typically presents as a hyperdense, homogeneous extra-axial collection with a shape like a biconvex or lentiform lens that does not cross the suture line [31,32]. **(Figs. 1 and 2).**

They rarely have a crescent shape or irregular shape, more typical in subdural hematoma. It can also demonstrate additional findings, such as deviation from the midline, traumatic subarachnoid hemorrhage, obliteration of the cisterns at the base, volume and thickness of the hematoma, brain contusion and a trace of cranial fracture [2,33,34]. **(Figs. 3 and 4)**.

In the case of massive EH, cortical convexity bulges, with consequent ventricular compressions, cerebral herniations and adjacent cortical contusions or by counterstroke [33].

On a CT scan, an EH could be distinguished from an acute subdural hematoma (ASDH) based on its shape. EH tends to be biconvex when compared to ASDH which is generally lenticular in its configuration. Another way would be that EH is generally confined to the cranial suture lines, when compared to the ASDH that crosses the suture line because it occurs more deeply in relation to the dura mater [35,36,37].
Occasionally, EH can extend from the supratentorial compartment to the infratentorial compartment [35,38].

The hemorrhage volume can be estimated by examining the skull CT using the standard formula A x B x C divided by the denominator 2 [39]:

A: the maximum diameter of the hematoma in centimeters.
B: the maximum perpendicular diameter of A in centimeters.
C: the number of 10 mm slices in which the hematoma appears.

The values of A, B and C are multiplied and divided by 2, resulting in the total volume of the hematoma.

Petersen et al [40] use this formula to calculate the hematoma volume:
hematoma volume (ml): 0.5 transverse diameter (mm) x anteroposterior diameter (mm) x craniocaudal diameter (mm).
The CT neuroimage of EH depends on several factors, such as source of bleeding, the interval between the lesion and the examination, the severity of the hemorrhage and the degree of organization or decomposition of the clot [30].
Zimmermann and Bilaniuk [30], classified EH based on the bleeding period as:

Type I: acute EH
Type II: subacute EH
Type III: chronic EH

Acute EH typically presents as a homogeneously hyperdense collection in two thirds of cases. In one third it is presented as a heterogeneous collection, containing areas of hyperdense blood and isodense serum. This characteristic translates to active arterial bleeding and is called the "swirl sign" [41,42]. **(Fig. 5)**. The swirl sign has two components, one active and the other more chronic. The active component is usually a small, round, isodense lesion with cerebral parenchyma, with a density between 30-50 UH. The most chronic component is a hyperdense blood collection measuring between 50-80 UH and represents a blood clot. Its presence reflects the need for an emergency surgical procedure to avoid the progression of the hematoma and its subsequent complications [41]. There is no enhancement post-administration of intravenous contrast in acute EH. In the presence of air inside the hematoma, a fracture of the paranasal sinuses or mastoid should be investigated [43,44] **(Fig.6)**. According to Frankauser and Kramer [45], the CT scan may be negative in the first hours after the trauma.

Subacute EH is one that occurs between 24 to 48 hours after the trauma, up to 21 days [46,47]. At CT, it is demonstrated as a homogeneously hyperdense collection, representing a solid blood clot, and may also be isodense or hypodense 48. It presents a more favorable evolution in relation to the acute and its location is different from the temporal region often seen in the acute 28. The EH subacute usually occurs in 24% to 65% of cases. They may be associated with cerebral contusion, diffuse axonal injury, subdural and intracerebral hematoma [33,49].

Chronic EH is usually due to venous laceration [50], is characterized by reduced (hypodense) or heterogeneous attenuation, as well as enhancement of the dural membrane as a result of blood degradation products. Peripheral dural enhancement

can be seen with the use of intravenous contrast media due to neovascularization [51] **(Fig. 7)**. Contrast CT shows enhancement of displaced dura mater due to neovascularization and granulation tissue [30].
The venous origin can be suspected when the hematoma crosses both sides of the tentacle [38]. Rarely does EH cross a suture, however in children it occurs in about 11% of cases, mainly in cases of cranial fracture that crosses the suture, suture diastasis and Vertex EH due to venous laceration.

The most common location is in the posterior fossa, because of injury to the sigmoid or transverse sinus **(Fig.8)**. It can also occur in the paramedian location above the cerebral convexities or in the middle cranial fossa due to laceration of the superior sagittal sinus (SSS) or sphenoparietal sinus, respectively 52 (Fig.9). Venous EH is unlikely to expand rapidly due to venous pressure that is insufficient to cause detachment of the skull dura [53].

The late EH develops after the first CT scan is negative 54, usually secondary to slow venous bleeding due to rupture of the dural sinus. The late appearance or subsequent increase in the volume of the hematoma is seen in 10% to 30% of the EH, and usually occurs in the first 24-48 hours [8,12].

The EH of the posterior fossa are rarer, they have high mortality due to fatal brain stem injury and are difficult to identify on CT, especially if small, due to the hardening of the simple radiography beam (beam hardening) derived from the adjacent bone structure. In these cases, the use of contrast medium facilitates its identification by demonstrating the displacement of the dural sinuses and the separation of the dura mater from calvary [55].

Sometimes it is difficult to differentiate by CT the extra or subdural location of a small hematoma, especially if it does not have the classic biconvex lens morphology. In these situations, magnetic resonance imaging (MRI) can bring greater diagnostic support, since the dura mater is very well identified as a curvilinear band with reduced signal intensity between the hematoma and the brain parenchyma. In addition, MRI suggests a venous nature when the hematoma is preferentially located in the planes of some venous sinus, which can move it away from the ordeal [56,57]. The relationship between the venous sinuses of the dura mater and the EH is well seen on MRI in the coronal and sagittal sections. The vertex EH due to SSS laceration is difficult to see on CT due to the partial volume effect of the hematoma with the internal face of the skull. In MRI, it is possible to demonstrate in coronal and sagittal

sections the blood interposed between the internal skull plate and the compromised venous sinus [58].

Factors that can lead to diagnostic difficulties of EH on CT:

1. Blood collection with low density can result from severe anemia.
2. Arterial leakage can reduce secondary to severe hypotension.
3. Positive CT finding requires a large accumulation of blood for your viewing: If the CT is performed early after the trauma, you may not have accumulated enough blood for proper interpretation.
4. If HE is secondary to venous bleeding, blood accumulation can be slow. This can be difficult, because of the difficulty with the interpretation of the CT.

The use of MRI in the acute phase is very limited. Being more sensitive than CT to reveal associated brain bruises. MRI has been important for its performance when the CT finding is inconclusive or for a better definition that the lesion requires [59]. The appearance and evolution of EH on MRI depend mainly on the age of the hematoma and the image sequence analyzed (T1, T2 and gradient-echo) [26]. Other factors influence its characterization: hemorrhage location, partial pressure of oxygen in the tissues, local pH, hematocrit of the patient, blood-brain barrier integrity and the patient's temperature.

In MRI, the signal intensity of the hematoma depends on the oxidative form of the iron (ferrous or ferric) in the hemoglobin molecule. Therefore, the phases of hemoglobin and its oxidation products (oxyhemoglobin, deoxyhemoglobin, methemoglobin, ferritin and hemosiderin) determine the signal characteristics of the hematoma on MRI. Therefore, five stages of bleeding are defined:

1. In the **hyperacute phase** (<12 hours), the newly extracted red blood cells from arterial blood contain completely oxygenated hemoglobin. Thus, oxygenated blood is diamagnetic and appears slightly hypointense or isointense in relation to the cerebral parenchyma at T1 and slightly hyperintense at T2. At this stage, the hematoma restricts the diffusion of water, appearing with hypersignal in the diffusion technique.
2. The **acute phase** begins after a few hours (12-48 hours) and is characterized by the formation of deoxyhemoglobin. The acute hematoma contains intracellular deoxyhemoglobin and appears markedly hypointense in T2. During this phase, the clot retracts, the hematocrit increases and forms a surrounding edema, which appears as a hyperintense perilesional halo in T2.

3. The **early subacute phase** begins after 2 to 7 days and is characterized by the oxidation of iron atoms to the ferric state, forming methaemoglobin. At this stage the blood-brain barrier is intact. Intracellular methaemoglobin is characterized by MRI as hypersignal in T1 and hyposignal in T2.
4. In the **late subacute phase** (8 days to 1 month), the blood-brain barrier breaks, conditioning the diffusion of the methaemoglobin, previously intracellular, into the hematoma (extracellular). It is characterized by hypersignal in T1 and T2.
5. After one month the hematoma enters the **chronic phase**. Methaemoglobin is degraded to ferritin and hemosiderin. This process causes the hematoma to appear with hypo-intensity in T1 and T2. The injection of paramagnetic contrast (gadolinium) is useful to confirm the venous origin of EH. The post-gadolinium T1 sequence demonstrates the displacement of the venous sinus by the hematoma.

Although CT is more used than MRI to detect hyperacute hemorrhage, MRI is more sensitive after 12-24 hours. MRI is also more specific than CT in determining the age of the hemorrhage. The T1- and T2-weighted sequences, as seen previously, adequately characterize the hemorrhage stage **(Figs. 10 and 11)**.

The Table below summarizes the five phases of EH according to their time of appearance, their cellular components and their characterization in MRI sequences [60,61,62,63,64.65]. (**Table 1)**.

PHASE	PERIOD	COMPONENT	T1	T2
Hyperacute	<12 hours	Oxyhemoglobin	Hypo/Iso	Hyper
Acute	12 to 48 hours	Deoxyhemoglobin	Hypo/Iso	Hypo
Early subacute	>48 hours to 7 days	Methaemoglobin intracellular	Hyper	Hypo
Late subacute	8 days to 1 months	Methaemoglobin extracellular	Hyper	Hyper
Chronic	> 1 months	Ferritin / hemosiderin	Hypo	Hypo

Table 1. Hemorrhage phases, related to the period of the hemorrhage, its predominant component and its signal characteristic in the spin-echo T1 and T2 sequences.

Cerebral angiography

CT is better than cerebral angiography (CAG), as it allows a faster diagnosis and better defines the presence of associated brain lesions. CAG has been used in cases of need to detect vascular injury that may have occurred at the time of the trauma or in the post-trauma [59]. It has been indicated for non-traumatic causes of EH such as arteriovenous malformation, arteriovenous fistula and middle meningeal artery pseudoaneurysm. According to Andrade et al [66], in a series of 24 patients with small EH, a vascular lesion was demonstrated on digital angiography, 29% of which were pseudoaneurysms and 71% of contrast medium leakage.

Treatment and prognosis

Most cases require surgical treatment [7]. According to Mezue et al [67], surgical treatment remains the appropriate approach to reduce morbidity and mortality. Immediate surgical drainage of the EH results in significant clinical improvement. Conservative treatment is instituted in cases where the hematoma diameter is less than 30 cm^3 thickness less than 15 mm and the deviation from the midline is less than 5 mm, and the patient should not have any focal deficit and must be kept under strict neurological surveillance and serial CT scans [33]. Zwayed and Luke-Wold [68], treated a series of 62 patients with EH using the following criteria: ECG between 13-15. CT findings: hematoma volume less than 40 mm, midline deviation less than 6 mm and absence of associated intracranial lesions. There was no mortality, and the resolution of the hematoma apparently occurred within 21 days and definitely between 3 and 6 months.

The bleeding can progress for a few days after the trauma. New blood accumulation may occur after drainage, justifying the performance of a CT scan within 36 hours to assess whether there is an evolution of the condition. These should be suspected in the presence of worsening levels of consciousness, paralysis of the third cranial pair or increased intracranial pressure.

CT can evaluate postoperative complications, such as subdural empyema, cerebral edema, brain abscess, intracerebral or brain stem hemorrhage and hypertensive pneumo encephalon. Mortality between 0% and 17% has been reported [5,17,22]. According to Onodera et al [69], the patient's age and the score on the Glasgow coma scale (GSC) are predictors of prognostic factors in patients undergoing EH. For Yurt et al [16], GSC at the time of surgery and CT findings where there is a deviation from the

midline greater than 10 mm, hematoma volume greater than 90 ml and thickness greater than 30 mm, significantly increase mortality.

Conclusion

Imaging exams are essential for management and prognosis in cases of EH. In acute cases, neuroimaging determines the presence and extent of hematomas, allows planning surgical procedures and guiding minimally invasive interventions. Neuroimaging is also important in chronic cases, identifying sequelae, determining prognosis and guiding rehabilitation. Technological improvements make it possible to reduce the time for exam acquisition and improve resolution, providing faster and faster exams and better therapeutic planning.

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