

Pseudo-subarachnoid Hemorrhage. A Case Report

Pseudohemorragia subaracnoidea. Presentación de un caso

Elkin José Beltrán Carrascal¹

David Cianci Bonet²

Laura López Brokate³

Mauro Duván Mendoza Quevedo⁴

David Francisco Saavedra Segura⁵

Arturo José Viera Oliveros⁶



Key words (MeSH)

Subarachnoid
hemorrhage
Tomography, X-ray
computed
Hypoxia, brain
Cryptococcal meningitis

Palabras clave (DeCS)

Hemorragia
subaracnoidea
Tomografía computarizada
por rayos X
Hipoxia encefálica
Meningitis criptocócica

Summary

Pseudo-subarachnoid hemorrhage (PSAH) is an infrequent entity characterized by findings in non-contrast head computed tomography that mimic subarachnoid hemorrhage, but without evidence of blood products in further studies. It has been associated with multiple etiologies, with hypoxic ischemic encephalopathy following cardiac arrest and cardiopulmonary resuscitation as the leading cause in literature. Clinical context and attenuation levels measured in Hounsfield Units should be taken into consideration when establishing the differential diagnosis between these entities. The case of a patient with PSAH of multifactorial etiology is presented.

Resumen

La pseudohemorragia subaracnoidea es un fenómeno infrecuente que se caracteriza por hallazgos sugestivos de hemorragia subaracnoidea en la tomografía computarizada simple de cráneo, sin evidencia de la misma en estudios adicionales. Se ha asociado a múltiples causas, de las cuales la principal es la encefalopatía hipóxico-isquémica posparo cardíaco y reanimación cardiopulmonar. El contexto clínico y los niveles de atenuación medidos en Unidades Hounsfield (UH) se deben tener en cuenta al hacer el diagnóstico diferencial entre ambas entidades. Se presenta el caso de una paciente con pseudohemorragia subaracnoidea de etiología multifactorial.

Introduction

Subarachnoid pseudo-hemorrhage is an entity characterized by tomographic findings that mimic subarachnoid hemorrhage (SAH), without evidence of bleeding in additional studies, such as those performed on cerebrospinal fluid (CSF) or autopsy material. On cranial computed tomography (CT), symmetrical increased density is seen in the base cisterns, sylvian cisterns and subarachnoid space, and is therefore usually reported as SAH. It has been associated with multiple causes, including hypoxic-ischemic encephalopathy after cardiac arrest and cardiopulmonary resuscitation (CPR), diffuse cerebral edema, pyogenic leptomeningitis and intracranial hypotension, among others (1).

Presentation of the case

This is a 59-year-old female patient with a history of liver cirrhosis due to chronic hepatitis and suspected hepatocarcinoma. She was admitted for a 20-day clinical picture of headache associated with right hypoacusis, decreased visual acuity, dysarthria and difficulty in swallowing and walking. On admission she was alert, oriented, with vital signs within normal limits. There was evidence of bradypsychia, bradylalia, truncal ataxia and multiple cranial neuropathy due to involvement

of cranial nerves III, VI, VIII, IX, X and XII. Laboratory tests showed hyperbilirubinemia, elevated transaminases, thrombocytopenia and hyponatremia. Abdominal ultrasound showed changes due to chronic liver disease and liver masses highly suggestive of hepatocarcinoma (LI-RADS-US 3B), computed axial tomography (CT) of the skull with contrast medium whose result was within normal limits (Figure 1) and lumbar puncture. The results of the CSF study were: leukocytes 0, erythrocytes 180/mm³ (90% fresh and 10% crenated, possibly due to traumatic puncture), glucose 23 mg/dL, protein 126 mg/dL, Gram, Z-N and India ink negative and FilmArray positive for cryptococcus. Meningeal cryptococcosis was diagnosed and treatment for cryptococcosis was initiated. On the fifth day of hospital stay, he presented deterioration of consciousness and cardiorespiratory arrest. CPR was performed with return of spontaneous circulation after 8 minutes. She was transferred to the intensive care unit with vasopressor support. His neurological examination revealed Glasgow scale 3/15 and absence of stem reflexes. A simple cranial CT scan showed severe cerebral edema characterized by loss of differentiation of gray and white matter and loss of sulci, associated with a symmetrical increase in density in the base cisterns, with densitometric values between 33-46 HU, compatible with subarachnoid pseudo-hemorrhage

¹Neurologist, Pontificia Universidad Javeriana. Neurologist and professor, Hospital Universidad del Norte. Barranquilla, Colombia.

²Radiology and Diagnostic Imaging Resident, third year, Universidad del Norte, Hospital Universidad del Norte. Barranquilla, Colombia.

³General Practitioner, Universidad del Norte, Hospital Universidad del Norte. Barranquilla, Colombia.

⁴Internal Medicine Resident, second year, Universidad del Norte, Hospital Universidad del Norte. Barranquilla, Colombia.

⁵Internal Medicine Resident, first year, Universidad del Norte, Hospital Universidad del Norte. Barranquilla, Colombia.

⁶Internal Medicine Resident, second year, Universidad del Norte, Hospital Universidad del Norte. Barranquilla, Colombia.

Department of Radiology and Diagnostic Imaging, Hospital Universidad del Norte.

(Figures 2 and 3); 48 hours later he had further hemodynamic deterioration and cardiorespiratory arrest, after which he was declared dead.

Discussion

The sign of subarachnoid pseudo-hemorrhage was first described by Spiegel et al. in 1986 (2). They reported 10 cases of patients with tomographic findings suggestive of SAH and cerebral edema, with no evidence of SAH at autopsy. In 1998, Avrahami et al. reviewed 100 patients with cranial CT scans compatible with SAH, with no evidence of blood or xanthochromia in CSF (3).

Subarachnoid pseudo-hemorrhage is characterized by low density of the brain parenchyma on plain CT, which may be associated with high density of the vasculature, mimicking blood in the basal cisterns and subarachnoid space. It presents with high density in sulci and cisterns, but without clinical or pathologic evidence to support it, so it is usually interpreted as a false positive for SAH. In addition, there is loss of differentiation between gray and white matter with effacement of the basal cisterns, indicating diffuse cerebral edema (4). Unlike aneurysmal SAH, which is typically associated with “thunderclap” headache, in subarachnoid pseudo-hemorrhage the clinical manifestations are variable, as it can be associated with multiple etiologies (5).

Generally, the attenuation value measured in Hounsfield Units (HU) is useful to distinguish subarachnoid pseudo-hemorrhage from SAH. In subarachnoid pseudo-hemorrhage it varies between 30-45 HU, whereas in aneurysmal SAH, being blood products, it usually varies between 60-70 HU (6, 7).

The pathophysiology of subarachnoid pseudo-hemorrhage depends on its etiology. When infectious, toxin production may cause disruption of the blood-brain barrier and allow the passage of proteins into the subarachnoid space, which may increase the attenuation coefficient and mimic SAH in severe cases. When associated with diffuse brain edema, blood-brain barrier disruption and vasogenic edema are usually found. In the acute phase of a brain injury, CSF reabsorption partially resolves the edema and, although CSF protein content may be elevated, it does not significantly modify its attenuation value on CT (4). However, when edema is severe, intracranial pressure (ICP) increases and dural venous sinuses may be compressed, affecting venous and CSF drainage. As a consequence, congestion and dilatation of superficial veins occurs, which stand out against a low-density parenchyma, thus mimicking SAH. In addition, increased ICP and inflammation reduce the subarachnoid space, displacing CSF and increasing the proportion of meninges and blood vessels, contributing to its increased attenuation (4).

The patient manifested headache with warning signs of multiple cranial neuropathy associated with chronic liver disease and probable hepatocarcinoma. Meningeal cryptococcosis was diagnosed by CSF FilmArray test, which has a specificity of 99.7% for this diagnosis (8). The post-CPR control CT showed the previously described findings, compatible with subarachnoid pseudo-hemorrhage in a clinical context highly suggestive of it (post-CPR state, initial CT without pathological findings and CSF not suggestive of SAH). Among the possible causes of subarachnoid pseudo-hemorrhage in this patient is neuroinfection, which can alter the permeability of the blood-brain barrier and produce vasogenic edema. However, due to the chronology of the clinical and imaging manifestations, it is considered that the findings are mainly due to post-CPR hypoxic-ischemic encephalopathy

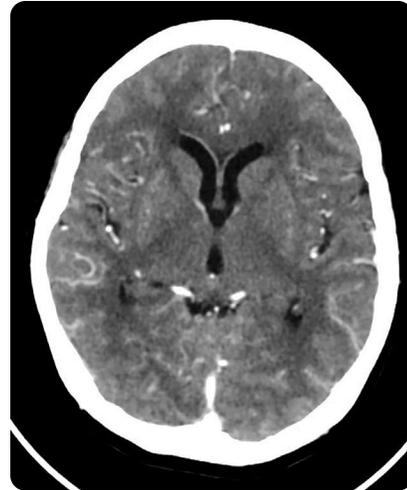


Figure 1. CT of the skull with contrast medium, axial view, performed at admission. No pathological findings are observed.

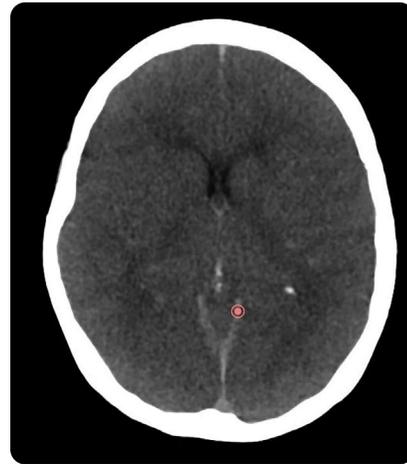


Figure 2. Simple skull CT, axial section. Loss of differentiation between gray and white matter is observed, with decrease in the depth of the sulci. The pointer indicates increased density in the subarachnoid space in the cerebellar tent.

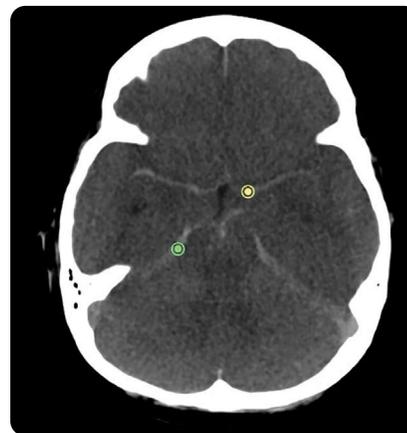


Figure 3. Simple skull CT, axial view. Loss of differentiation between gray and white matter is observed, with decreased depth of the sulci. Pointers point to increased density in the subarachnoid space in the cisterns of the base.

Conclusión

Subarachnoid pseudo-hemorrhage is of worse prognosis than SAH and has been associated with adverse neurological outcomes. Therefore, it is of vital importance to know its radiological pattern and to include it within the differential diagnosis when the clinical context suggests it, since its early identification may facilitate end-of-life decision making and invasive procedures.

References

1. Ferrante E, Regna-Gladin C, Arpino I, et al. Pseudo-subarachnoid hemorrhage: a potential imaging pitfall associated with spontaneous intracranial hypotension. *Clin Neurol Neurosurg.* 2013;115(11):2324-8.
2. Spiegel S, Fox A, Vinuela F, et al. Increased density of tentorium and falx: a false positive CT sign of subarachnoid hemorrhage. *Can Assoc Radiol J.* 1986;37(4):243-7.
3. Avrahami E, Katz R, Rabin A, et al. CT diagnosis of non-traumatic subarachnoid haemorrhage in patients with brain edema. *Eur J Radiol.* 1998;28(3):222-5.
4. Given C, Burdette J, Elster A, et al. Pseudo-subarachnoid hemorrhage: A potential imaging pitfall associated with diffuse cerebral edema. *Am J Neuroradiol.* 2003;24(2):254-6.
5. Hasan T, Duarte W, Akinduro O, et al. Nonaneurysmal "pseudo-subarachnoid hemorrhage" computed tomography patterns: challenges in an acute decision-making heuristics. *J Stroke Cerebrovasc Dis.* 2018;27(9):2319-26.
6. Yuzawa H, Higano S, Mugikura S, et al. Pseudo-subarachnoid hemorrhage found in patients with postresuscitation encephalopathy: characteristics of CT findings and clinical importance. *Am J Neuroradiol.* 2008;29(8):1544-9.
7. Senthilkumaran S, Balamurugan N, Menezes R, et al. Role of Hounsfield units to distinguish pseudo-subarachnoid hemorrhage. *Clin Toxicol.* 2011;49:948.
8. O'Halloran J, Franklin A, Lainhart W, et al. Pitfalls associated with the use of molecular diagnostic panels in the diagnosis of cryptococcal meningitis. *Open Forum Infect Dis.* 2017;4(4):ofx242.

Correspondence

Laura López Brokate
Carrera 56 # 79-342
Barranquilla, Colombia
lbrokate@uninorte.edu.co

Received for evaluation: October 26, 2019

Accepted for publication: August 19, 2020