

Case report

Cerebral venous congestion correlates to acute aneurysm rupture: An illustrative case with Doppler ultrasonography study

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ABSTRACT

Background: The objective of our description is to shed light on some new hemodynamic and clinical characteristics in the unstable cerebral aneurysm Case: We describe a 54 year old woman who presented a tension headache, that increasing for several days. A CT scan performed in ER suggests a possible arterial ectasia at the level of the circle of Willis. The patient is hospitalized. An angio CT shows an aneurysm of the anterior communicating artery, without signs of fixation and/or other instability. A subsequent TCCD examination with venous study shows clear congestion at the level of the sphenoparietal sinus. The Valsalva maneuver determines an increase in local congestion. In the light of the ultrasound picture, the patient was quickly received in Neurosurgery with success.

Conclusion: we describe a clinical case where the worsening tension headache was not secondary to the increase of volume of the aneurysm but was an epiphenomenon of venous congestion, explored with TCCD. The mass effect of the aneurysm determined venous sinus compression and changed the hemodynamic of the cerebral venous flow. We believe that venous outflow obstruction and a high intracranial venous pressure gradient may be a cerebral aneurysm rupture factor.

1. Introduction

Cerebral aneurysms are pathological dilatations of the arterial walls frequently located near arterial bifurcations in the circle of Willis. About 2–5% of the world population is suffering from aneurysmal disease with ratio male: female of 1:1.6 and a rupture rate about 0.7–1.9%. Estimated 30 days mortality rate following intracranial aneurysm rupture reaches 45% and one-third of the surviving patients suffer from moderate to severe disability. Rarely, intracranial aneurysms are diagnosed prior to rupture as an occasional pattern [1,2,4].

Size, shape, morphology and location are known risk factors for rupture of an aneurysm, but morphologic parameters alone may not be sufficient to perform proper rupture risk stratification [5,7]. For management of patients with un-ruptured aneurysms, prognostic criteria for the risk of rupture are needed. As a means of providing an inclusive risk prediction model, the PHASES (Population, Hypertension, Age, Size of the aneurysm, Earlier subarachnoid hemorrhage from another aneurysm, and Site of the aneurysm) score, based on a pooled analysis of six prospective natural history studies, was recently proposed [22]. There are few studies though examining the link between hemodynamic factors, so diurnal changes in blood pressure, brain venous drainage/ venous pressure gradient and cerebral aneurysm formation or rupture [3,6,8].

The central nervous system can be divided into three compartments: blood (arterial and venous), parenchyma (intracellular and extracellular) and cerebrospinal fluid (CSF) [9]. The sum of the intracranial volumes of blood, parenchyma and CSF must be constant and an increase in any one of these must be offset by an equal decrease in another. An increase in pressure, caused by an expanding intracranial volume, is distributed evenly throughout the intracranial cavity [10]. Intracranial hypertension occurs just when mutual inter-compartmental volume compensation fails [11]. Individual susceptibility to intracranial hypertension may be influenced by the anatomical balance of cerebral venous drainage and its compliance. As cerebral blood flow increases so too must venous drainage. Venous distension will occur up to a limit, after which intravenous pressure (and that upstream) will rise steeply [9]. The relationship between intracranial pressure (ICP) and cerebral venous pressure is tight, according to some authors, so Schoser et al. [12] About 70–80% of the intracranial blood volume is located in the venous system. Alteration of ICP results in changes of venous flow velocities. A space occupying process, like aneurysm, can result in changes of venous flow velocities up to cerebral venous congestion and venous thrombosis [5,12,13].

There are also some interesting data of cerebral aneurysm rupture likely relating to asymmetry of the dural sinus especially in female population [7,8]. Krahalios et al. reported that elevated intracranial

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venous pressure (ICP) may be due to a stenotic cerebral venous outlet for various causes [14]. With an asymmetric cerebral venous outflow pattern, for stenosis of a venous sinus (or hypoplasia/atresia), the venous outflow is faster and the ipsilateral venous pressure gradient is higher, that may be a factor in the rupture of a cerebral aneurysm. In Duman's study there was a statistically significant association between venous dominance and the aneurysmal side. This may mean that venous dominance can also be a predisposing factor for aneurysm formation. Venous dominance also had a statistically significant association with rupture in this study and a retrospective Y TSAI's study [23,24].

Headache is the most common symptom in Intracranial Hemorrhage (ICH) and venous congestion up to Cerebral Sinus Venous Thrombosis (CSVT). Typically, it's described as diffuse and often progressive in severity over days to weeks. In a minority of patients may present as a thunderclap headache, suggesting a hemorrhagic process or migraine like headache [15–17].

2. Objective

to describe how a space occupying process, like aneurysm, correlates to an aggravating headache, to cerebral venous congestion, elevated intracranial venous pressure and a risk of aneurysm rupture.

3. Case report

We describe a 54 year old woman who has accessed to the Emergency Room (ER) for important headache (8-10VAS), for several days. She describes frontal and at the top headache, like a circle, that sometimes pushes, at the beginning present only during the day then continuous and unbearable. The remote anamnesis was silent. No vascular risk factors were known. Her sister was suffering from polycystic kidney.

Neurological objective examination revealed only mild edema of the optic papilla.

A CT scan performed in ER suggests a possible arterial ectasia of the anterior communicating artery (ACoA).

The patient is hospitalized.

Routine blood tests didn't highlights significant elements.

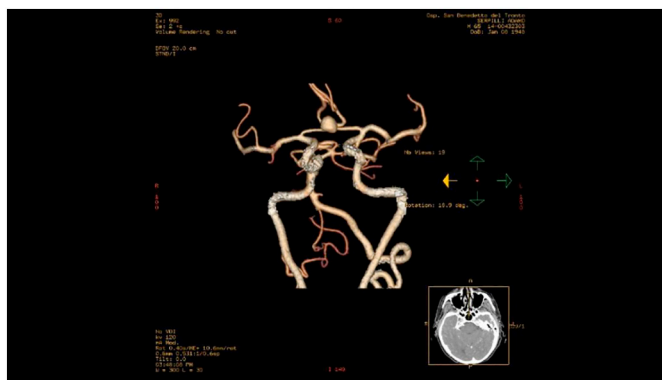
ECG: sinus rhythm.

The EEG was normal.

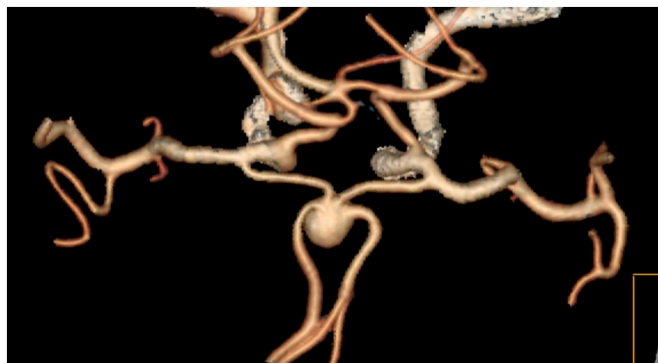
An angio CT in ER shows an aneurysm of the anterior communicating artery (ACoA), about 1.5 cm, with the fundus spherical and smooth. Radiologists report aneurysm without signs of contrast medium fixation and/or other signs of instability (Pictures 1–2). PHASES score was 10.

The neurosurgeon didn't therefore indicate urgent treatments.

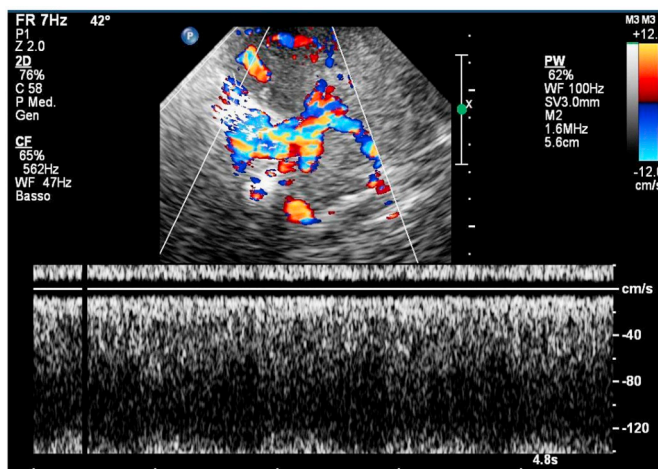
A subsequent TransCranial-echo-color-Doppler (TCCD) examination (phased array probe 2,5 MHz) with venous study showed clear bilateral



Picture 1. Angio-CT – Aneurysm of the anterior circle.



Picture 2. AngioCT- Details of aneurysm of the anterior communicating artery (ACoA).



Picture 3. TCCS (Philips iU 22 and phased array probe 2.5 MHz) from temporal bone window. The picture shows the insonation of sphenoparietal sinus, identified as a venous signal along the hyperechogenic lesser wing of the sphenoid bone (arrow). The Doppler waveform of the SPaS show a flow direction that away from the probe and markedly increased flow velocity with dispersion of frequencies in the waveform, like a venous flow. Unavoidable aliasing on arterial branches as a result of venous presetting which low PRF and increased gain.

congestion at the level of the sphenoparietal sinus (Peak of Systolic Velocity (PSV) of about 1 m/s, with normal cut-off values of 0.18–0.20 m/s) (Picture 3). A clear asymmetry of the venous sinus wasn't evident. The Valsalva maneuver determined a further increase in local congestion. The lumbar puncture and scheduled brain MRI with angiographic phase were not performed. In the light of the clinic and ultrasound picture, the patient was quickly received in Neurosurgery Department with success. The aneurysm is un-ruptured.

4. Discussion

Our patient didn't previously report headache. It was tension type, frontal, subacute, initially present only during the day and than continuous, persistent and unbearable. It was associated with mild edema of the optic papilla and bilateral congestion at the level of the sphenoparietal sinus at the TCCD examination. It was clear the relation between the worsening headache, the aneurysm of ACoA highlighted at angio CT and the venous congestion. Not the shape, not the size but the location, the hemodynamic repercussion and the clinical manifestation were the warning signs.

Headache is the most common symptom in cerebral venous congestion/thrombosis and it's also the commonest initial alarm [15]. Typically it's described as diffuse or anterior/posterior bilateral and

often progressive in severity over days to weeks. In a minority of patients may present as a thunderclap headache (suggesting a hemorrhagic process) or as migraine like. There is no recognizable pattern of headache in CVC/T, but more commonly it's new onset, sub-acute to rapidly progressive over a few days, intermittent initially and constant later [16]. There was no association between localization of headache and site of sinus thrombosis/congestion except in sigmoid sinus, when the pain is usually in the occipital and neck region [16,17].

The pathogenesis of headache in CVC/T by compression is not well understood. The cerebral veins and sinuses neither have valves nor tunica muscularis; this permits veins to remain dilated and flow is possible in different directions. Compression of the cerebral veins can cause localized edema, venous congestion and thrombosis. Two different kinds of cerebral edema can develop, cytotoxic and vasogenic. Multiple mechanisms of headache include a combination of increased intracranial pressure, micro subarachnoid hemorrhage (SAH), stretching or irritation of nerves in sinus walls and inflammation of sinus walls. Stretching of the perivascular nerves of congested/dilated collaterals might explain the overlying headache and its throbbing/pulsatile quality. Most patients had diffuse headache without any significant association to the location of venous congestion/thrombosis [16–18]. To diagnose the CSVC/T associated with an aneurysm, CT is abnormal only in patients with neurological signs. It can be found dilatation, hyper-density of venous sinus, damage to the brain parenchyma, from edema to ischemia or hemorrhage.

Magnetic Resonance Imaging is more sensitive than CT in the diagnosis of CSV/T and instability of the aneurysm. We can detect the presence of sinus congestion or thrombosis with T1, T2 sequences, fluid inversion recovery analysis (FLAIR) in axial, coronal and sagittal planes. The sinus may appear isointense or hyper-intense, depending on the age of thrombus formation [19].

The acute phase of cerebral vein and sinus congestion/thrombosis near aneurysm may be diagnosed by transcranial color-coded duplex sonography (TCCS). One important anatomic characteristic of the intracranial venous system with relevance for ultrasound examinations is the lack of valves. This implies that the flow direction in cerebral veins and dural sinuses is governed solely by the current pressure gradient. Direct CSVC/T signs criteria comprise the lack of color signal and filling defect within a dural sinus after application of echo contrast agents. However, thrombotic occlusion or partial thrombosis cannot be differentiated from aplasia or hypoplasia by only ultrasound. Indirect criteria are based on the detection of venous collateral flow. Due to high venous flow velocities numerous prominent veins are visible even when an arterial presetting of the ultrasound system is used. Venous flow velocities are pathologically increased (defined as 50%) for the deep middle cerebral vein (dMCV) and the Basal Vein (BV). Due to the considerable rate of hypoplasia or caliber differences, side differences of flow velocities in paired sinuses have only a diagnostic relevance when venous flow velocities are pathologically increased [20].

There aren't studies that correlate aneurysm, venous flow congestion and recognizable patterns at TCCD examination. In our patient head CT shows only a possible ectasia of the ACoA. An Angio CT study shows an aneurysm of the ACoA without signs of contrast fixation and other instability. The neurosurgeon doesn't therefore indicate urgency. The patient reports that the headache was persistent and increased day by day and the pain had become like a helmet that pushes finally. There was something that did not fit.

A TCCD examination with venous study shows clear congestion at the level of the sphenoparietal sinus. In the [picture 3](#) we can clearly see the Medial Cerebral Artery (MCA) and the hyperechogenic small wing of the sphenoid over the sinus is located, which is detected by positioning the sample volume along the small wing. Unavoidable aliasing on arterial branches given the value of the Pulse Repetition Frequency (PRF) set and motivated by the search for venous slow flows. The use of ultrasound to study cerebral venous circulation is a recent application of TCCD. There are certain indispensable conditions to a correct

approach to cerebral venous study: correct setting of the equipment. A frequency of between 1.5 and 2.0 MHz; it's important a "low-flow setting": filters have to be switched off and the PRF must be reduced. Besides, in order to reduce the PRF (up to 0.5 kHz or lower) as much as possible, the gain needs to be gradually increased up to the limit of the noise [22]. Flow velocity analysis should only be performed if the vessel is clearly visible [21]. To detect PSV values a correct angle is indispensable, because can change the values enormously. Bidimensional imaging is irreplaceable in the search for reference points. Normally the flow appears "continuous" throughout the cardiac cycle, maintaining, however, a pulsatility which at times becomes more evident. We need for an activation maneuver (Valsalva maneuver) to verify the nature of the venous flow in case of strong pulsatility. Yet, the response to the Valsalva maneuver can vary in relation to the outflow activated, to the anatomical and functional variability, to the pressure gradient in a system with no valves, as well as to the influence of the continence of the Internal Jugular Vein (IJV) valve [22]. Spheno-parietal sinus can be insonated through the transtemporal bone window using the upper pontine axial insonation plane. Start to identifying the hyperechogenic lesser wing of the sphenoid bone in b-mode. Then a small color window with a low PRF setting is placed over this region. Or, alternatively, can be identified as a venous signal along the sphenoid bone. The flow is directed away from the transducer.

5. Conclusion

We describe a clinical case where the worsening tension headache was not secondary to aneurysm rupture but was an epiphenomenon of venous congestion near it. The mass effect of the aneurysm determined compression of venous sinus and changed the hemodynamic of cerebral venous flow. Venous outflow obstruction, impaired venous drainage and a high venous pressure gradient may be a factor of cerebral aneurysm rupture in our case.

Venous congestion determines wall distension and stretch or irritation of nerves in sinus walls and dural sensitive fibers, possible inflammation of sinus walls and increasing headache. Only after TCCD examination, with venous study, neurosurgeon suggests urgent treatment of un-ruptured aneurysm, with good outcome for the patient. We describe how some hemodynamic and clinical features are the red flags in cerebral aneurysm rupture. Transcranial ecoColor-Doppler sonography, may be a supplement diagnostic arm for detect cerebral aneurysms. It's a bedside real time neuro-imaging modality for the evaluation and follow up of a cerebral arterial stenosis/dilation, venous congestion and thrombosis. It's a non-invasive, cheap, fast, portable, reproducible procedure who can play an important role in differential diagnosis of cerebro-vascular disease and dynamic characteristics of a process occupying space.

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