Non-aneurysmal perimesencephalic subarachnoid hemorrhage

- Perimesencephalic Subarachnoid Hemorrhage Bleeding Patterns Are Not Always Benign: Prognostic Impact of an Aneurysmal Pathology
- Comment on Roman-Filip et al. Non-Aneurysmal Perimesencephalic Subarachnoid Hemorrhage: A Literature Review. Diagnostics 2023, 13, 1195
- Frequency and Characteristics of Spinal Bleeding Sources in Nontraumatic Angiogram-Negative Subarachnoid Hemorrhage
- The clinical course and outcomes of non-aneurysmal subarachnoid hemorrhages in a single-center retrospective study
- Letter to the Editor Re; "Treatment and outcomes of non-aneurysmal perimesencephalic subarachnoid hemorrhage: A 5 year retrospective study in a tertiary care centre"
- Non-Aneurysmal Perimesencephalic Subarachnoid Hemorrhage: A Literature Review
- Hydrocephalus, cerebral vasospasm, and delayed cerebral ischemia following non-aneurysmatic spontaneous subarachnoid hemorrhages: an underestimated problem
- Treatment and outcomes of non-aneurysmal perimesencephalic subarachnoid haemorrhage: A 5 year retrospective study in a tertiary care centre

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In 1985 Van Gijn and co-workers were the first to describe a particular subgroup of patients who presented with an acute subarachnoid hemorrhage with the clot seen on computed tomography (CT) confined to the perimesencephalic cisterns and adjacent subarachnoid spaces [1].

Epidemiology

Nonaneurysmal perimesencephalic subarachnoid hemorrhage has become well recognized as a distinct type of subarachnoid hemorrhage and may account for up to two-thirds of all SAHs unknown cause [2) 3) 4)].

Etiology

The etiology is unknown, but many explanations have been proposed, including venous injury or rupture followed by thrombosis of a ruptured microaneurysm.

95% of cases of perimesencephalic subarachnoid hemorrhage have a normal cerebral angiography and the source of bleeding is not identified; the cause is thought to be a venous bleed. This is referred to as non-aneurysmal perimesencephalic subarachnoid hemorrhage.

Traumatic perimesencephalic subarachnoid hemorrhage

Diagnosis

Noncontrast CT has been shown to have very high sensitivity and negative predictive value for subarachnoid hemorrhages in the first 6 h of the onset of a thunderclap headache [5].

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https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10670416/

Although CTA is being increasingly used by emergency physicians due to the ease of ordering and the shorter associated ED stay, it is important to keep in mind that CTA is detecting aneurysms and not intracranial bleeds [6] [7]

Further investigations can also be used to highlight this diagnosis, such as interventional angiography or magnetic resonance imaging [8].

Non-aneurysmal perimesencephalic subarachnoid bleeding is characterized by an accumulation of blood in the perimesencephalic and prepontine cisterns identified on sectional imaging together with persistently negative cerebral angiography. Magnetic resonance imaging usually contributes no further information on the possible cause of the bleeding. Still, it may occasionally show additional features including associated parenchymal infarcts such as the pontine hemorrhagic infarct seen in a case described by Duncan et al. [9].

Outcome

All of the 13 patients in the series of van Gijn et al. had negative cerebral angiograms and followed a benign clinical course with excellent outcome [10]

Other authors have independently confirmed this as a distinct entity with consistent imaging findings and a universally benign clinical course and good outcome [11] [12].

Treatment

Given the rarity of this condition and its relatively better prognosis, treatment options usually remain conservative [13]

Literature Review

Further investigations can also be used to highlight this diagnosis, such as interventional angiography or magnetic resonance imaging. Given the rarity of this condition and its relatively better prognosis, treatment options usually remain conservative [14)]

Case reports

A 48-year-old man on apixaban for multiple venous thromboembolisms presented with the worst headache of his life associated with blurry vision, nausea, and neck stiffness. Computed tomography demonstrated a perimesencephalic pattern of blood (Hunt and Hess grade 2, Fisher grade 3). Computed tomography angiography and 6-vessel digital subtraction angiography demonstrated no precipitating cause. Systemic tissue plasminogen activator (tPA) was administered on postbleed day 8 owing to obstructive shock from saddle pulmonary embolism and pulseless electrical activity. He was safely discharged to rehabilitation with moderate neurological deficits attributed to ischemic effects of his cardiac arrest.

Conclusions: Symptomatic saddle pulmonary embolism in the setting of intracranial hemorrhage creates conflicting risks of medical intervention. There are no case reports or evidence of the use of systemic thrombolysis in the setting of SAH. Owing to the benign natural history of PNSH, tPA may be a safe intervention. Neurointensivists and neurosurgeons should be aware that intravenous tPA was used safely for life-threatening pulmonary embolism in the setting of PNSH. Additionally, the use of tPA without resultant rebleeding in this case opposes the theory of the presence of a thrombosed ruptured microaneurysm [15)].

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