Known triggers include acute exacerbation of asthma and situations requiring the Valsava maneuver[4].

The combination of spontaneous pneumomediastinum and pneumorrhachis is extremely rare[5,6]. Possible causes of pneumorrhachis include use of the drug ecstasy, abscesses, asthma attacks, coughing fits, violent vomiting, epidural anesthesia, lumbar puncture, and thoracic or vertebral surgery or trauma[7,8]. In extremely rare cases, meningitis or pneumocephalus can occur[7]. Pneumorrhachis typically occurs directly, when atmospheric air reaches the epidural space by means of a needle or a penetrating wound from the spine, although it can occur indirectly, as in bronchial asthma. In the case of bronchial asthma, air from the rupture of a peripheral pulmonary alveolus leaks into the pulmonary perivascular interstitium and follows the path of least resistance of the mediastinum to the fascia of the neck. Due to the absence of fascial barriers, air crosses the neural foramen and deposits in the epidural space. In either situation, pneumorrhachis is usually asymptomatic and disappears spontaneously within a few weeks.

Whereas CT allows direct visualization of the presence of air in the affected compartment(s), X-rays can reveal signs typical of pneumomediastinum, produced when the air leaving the mediastinum delineates the normal anatomical structures. Such signs include subcutaneous emphysema, the "sail sign" of the thymus, pneumopericardium, the "ring-around-the-artery" sign, the "continuous diaphragm" sign, and the "double bronchial wall" sign[3].

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http://dx.doi.org/10.1590/0100-3984.2015.0031

Terson's syndrome: an important differential diagnosis of subarachnoid hemorrhage

Dear Editor,

A 42-year-old female patient presented to the emergency room with severe headache and hypertensive urgency (blood pressure, 220/110 mmHg), progressing to left hemiparesis, right anisocoria, and a decreased level of consciousness, with a Glasgow Coma Scale score of 4. Computed tomography (CT) of the brain showed acute subarachnoid hemorrhage (Fisher grade 4), due to rupture of an aneurysm in the anterior circulation, together with signs of bilateral intraocular hemorrhage (Figure 1). Those findings are consistent with a diagnosis of Terson's syndrome.

Terson's syndrome was initially described as vitreous hemorrhage secondary to acute subarachnoid hemorrhage, although recent studies have shown that it can also result from traumatic brain injury or even nontraumatic intracerebral hemorrhage[1]. Originally described in 1900 by Albert Terson, the syndrome has an incidence of 2.6–27.0% in the context of subarachnoid hemorrhage due to a ruptured aneurysm[2–4]. Although the etiology of the syndrome is controversial, it has been attributed to a

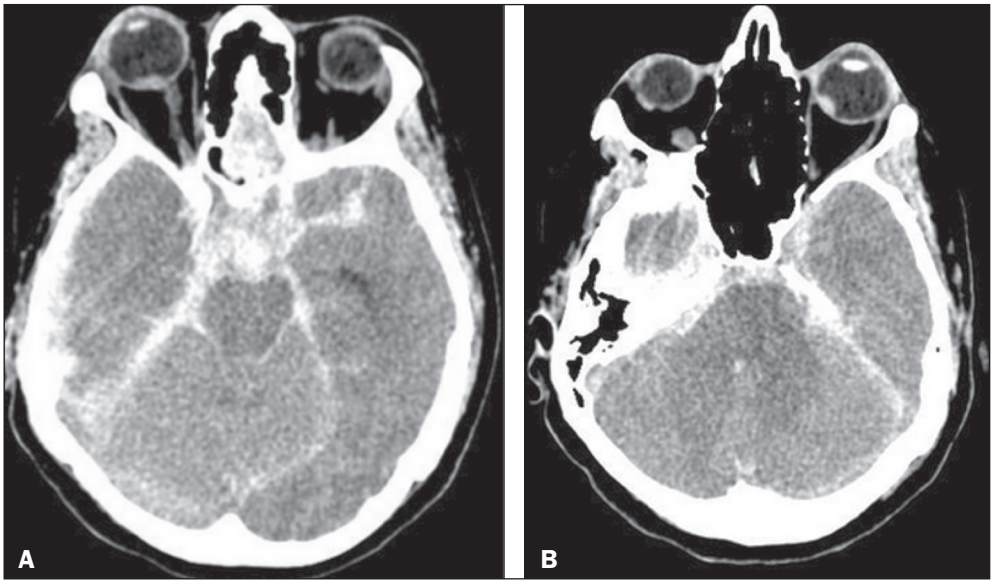

Figure 1. A: CT scan showing signs of subarachnoid hemorrhage and right intraocular hemorrhage, as a spontaneously hyperattenuating focus in the posterior portion of the right globe. **B:** CT scan showing left intraocular hemorrhage.

rapid increase in venous or intracranial pressure, which causes rupture of the peripapillary capillaries of the retina or results in compression of the central retinal vein, thus decreasing retinal venous drainage, promoting stasis, and provoking hemorrhage[5].

The diagnosis of intraocular hemorrhage is more accurately confirmed by ophthalmoscopy, although CT can suggest it, with an estimated sensitivity of 66%. The changes seen most frequently are retinal thickening and hyperattenuating nodules overlying the optic disc[6].

Terson's syndrome most often occurs in patients with severe neurological disease, a Glasgow Coma Scale ≤ 8, and aneurysmal subarachnoid hemorrhage with a Fisher score ≥ 3 at presentation. It is also of note that the rates of morbidity and mortality are high among such patients. In the sample studied by Fountas et al.[7], the mortality was 28.6% among the patients with intraocular hemorrhage, compared with only 2.0% among those without.

Terson's syndrome is not an uncommon condition, perhaps being underdiagnosed. Given the prognostic implications of this diagnosis for morbidity and mortality, as well as the potential for secondary ocular lesion, it is of extreme relevance to radiologists and other medical professionals, especially in the context of acute subarachnoid hemorrhage[8] but also in other forms of intracranial hemorrhage.

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http://dx.doi.org/10.1590/0100-3984.2016.0053

Transient bilateral striatal lesion associated with varicella infection

Dear Editor,

A 5-year-old girl presented to our institution with an 8-day history of dermatological lesions typical of chickenpox, which had evolved to nausea, vomiting, and abdominal pain. During the observation period, she received symptomatic treatment (medication). Because she also experienced somnolence and apathy, she was hospitalized for further diagnostic investigation, evolving to a lack of fine motor coordination, difficulty in walking, tremor, dystonia, generalized tonic-clonic seizures, dysmetria, and decomposition of movement. Cerebrospinal fluid analysis revealed pleocytosis with a predominance of lymphocytes (12 leukocytes with 96% lymphocytes). A computed tomography scan of the head showed no abnormalities. Magnetic resonance imaging (MRI) showed hyperintense lesions in the caudate nuclei and putamen on T2-weighted and proton density-weighted sequences (Figure 1), without enhancement after contrast administration. The patient showed gradual improvement and was discharged after 6 days of hospitalization. She was referred to a pediatric neurology clinic. After three months of follow-up, her symptoms had completely disappeared and another MRI of the brain showed regression of the lesions (Figure 2).

Varicella-zoster virus causes chickenpox and is associated with a variety of complications. The most common noncutaneous site of involvement is the central nervous system. Complications

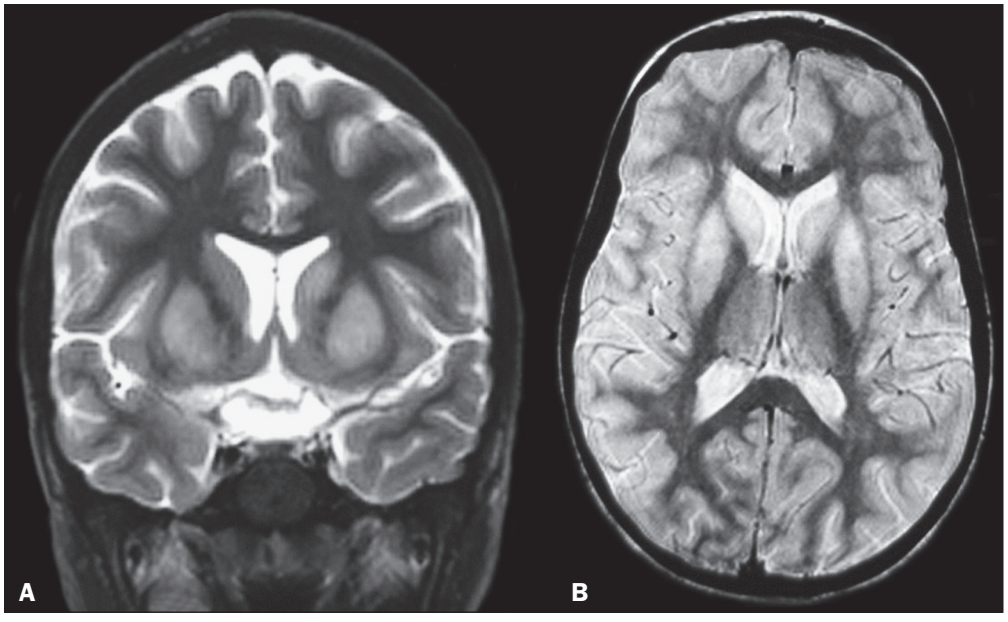

Figure 1. MRI of the brain showing hyperintense lesions in the caudate nuclei and putamen. **A:** Coronal T2-weighted sequence. **B:** Axial proton density-weighted sequence.