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Acute Spontaneous Posterior Fossa Subdural Hematoma

Akut Posterior Fossa Subdural Hematom

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ABSTRACT

Acute posterior fossa subdural hematomas are rare and most of them are trauma-related. Non-traumatic ones have been reported in patients who had idiopathic thrombocytopenic purpura or those who had been receiving anticoagulant therapy. We report on the case of 57-year-old Iranian man who developed sudden severe occipital headache, drowsiness, repeated vomiting, and instability of stance and gait. He was neither hypertensive nor diabetic. No history of head trauma was obtained and he denied illicit drug or alcohol ingestion. A preliminary diagnosis of acute intra-cerebellar hemorrhage was made. His CT brain scan revealed an acute right-sided, extra-axial, crescent-shaped hyperdense area at the posterior fossa. His routine blood tests, platelets count, bleeding time, and coagulation profile were unremarkable. The patient had spontaneous acute infratentorial subdural hematoma. He was treated conservatively and discharged home well after 5 days. Since then, we could not follow-up him, clinically and radiologically because he went back to Iran. Our patient's presentation, clinical course, and imaging study have called for conservative management, as the overall presentation was relatively benign. Unless the diagnosis is entertained and the CT brain scan is well-interpreted, the diagnosis may easily escape detection.

Key Words: acute subdural hematoma; posterior fossa; cerebellar compression; ataxia; spontaneous

ÖZET

Akut posterior fossa subdural hematom nadir olarak gözlenir ve çoğu travmaya bağlıdır. Travmatik olmayanlar ise anti-koagulan tedavi görmüş yada idiopatik trombositopenik purpura hastalarında rapor edilmiştir. Çalışmamızda ani ve ciddi oksipital baş ağrısı ile birlikte halsizlik, tekrarlayan kusmalar, dengesiz duruş ve yürüyüşü olan iranlı 57 yaşındaki bir erkek rapor edilmiştir. Bu kişi hipertansiyon veya diabet hastası değildir. Başında travma geçmişi bulunmamaktadır ve kişi uyuşturucu yada alkol kullanmadığını söylemektedir. Akut intra serebral hemoraji için ön bir tanı yapıldı. CT beyin taraması, posterior fossa bölgesinde sağ taraflı, ekstra aksiyal hilal şeklinde yoğunlaşmış bir alan olduğunu gösterdi. Rutin kan testleri, kanama zamanı, koagülasyon durumu ve trombosit sayımı normaldi. Hastada spontan gelişen akut infratentoryal subdural hematom vardı. Hasta tedavi edildi ve 5 gün sonra iyi bir şekilde taburcu edildi. O zamandan beri hasta klinik olarak veya radyolojik olarak takip edilemedi, çünkü hasta irana geri döndü. Hastanın durumu, klinik seyiri ve görüntüleri konservatif bir yönetim gerektirmiştir. Dolayısıyla genel anlamda hastanın durumu nispeten iyi olsada, CT beyin taramasını iyi yorumlanmadıkça ve olası teşhis konusunda önceden fikir sahibi olunmadıkca hastalık kolayca gözden kaçabilir.

Anahtar Kelimeler: Akut subdural hematom, posterior fossa, serebral basınç, spontan, ataksi

INTRODUCTION

Acute subdural hematomas (SDHs) of the posterior fossa are rare and constitute about 0.6 to 1% of all such intracranial hematomas[1,2]. Most cases are trauma-related and there is relatively a high frequency of occipital impacts and fractures[3]. In the absence of head trauma, rupture of posterior fossa arteriovenous malformation or Berry's aneurysm might be the cause in some patients. The pertinent literature has also reported several non-traumatic SDHs in patients who had idiopathic thrombocytopenic purpura or patients who had been receiving warfarin[2,4-7].

CASE PRESENTATION

A 57-year-old Iranian male was referred to our Acute and Emergency department one hour after developing sudden severe occipital headache and repeated vomiting. His blood pressure was 137/65 mmHg with a regular pulse rate of 110 beat per minute, a respiratory rate of 12 cycles per minute, and temperature of 36.8 ^{o}C. He was drowsy and demonstrated ataxia of stance and gait. There were no nystagmus, ocular palsies, facial weakness, or dysphagia. His fundal examination was unremarkable. Both planter reflexes were flexors. He was neither hypertensive nor diabetic. His family denied any history of head trauma or illicit drug abuse and there was no family history of note. A preliminary diagnosis of acute intracerebellar hemorrhage was made by the referring primary care hospital.

He underwent a battery of tests, including complete blood count, blood film, erythrocyte sedimentation rate, prothrombin time, activated thromboplastin time, bleeding time, random blood sugar, lipid profile, urea and electrolyte, liver function, thyroid function, and general urine examination. All of these turned out to within their normal reference range. His CT brain scan showed a small crescent-shaped hyperdense area between the right occipital bone and the right cerebellar hemisphere. The midline vermis was displaced a little bit to the left side and the fourth ventricle was compressed (figures 1 and 2). The blood had also dissected upward between the tentorium layers. The patient had developed acute spontaneous non-traumatic subdural hematoma of the posterior fossa.

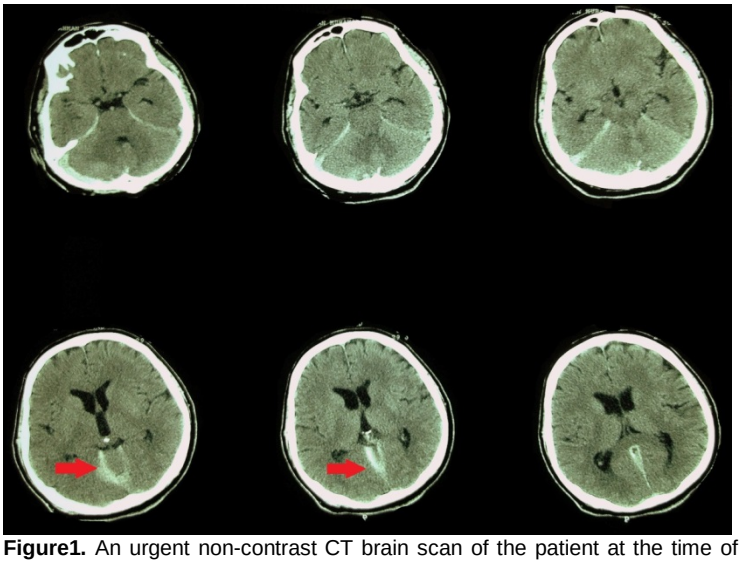

Figure1. An urgent non-contrast CT brain scan of the patient at the time of Acute and Emergency department admission. Note that the right acute posterior fossa subdural hematoma is not that obvious (left upper slice at the level of mid-pons). However, the hyperdesne blood at the tentorium cerebelli is remarkable (red arrows).

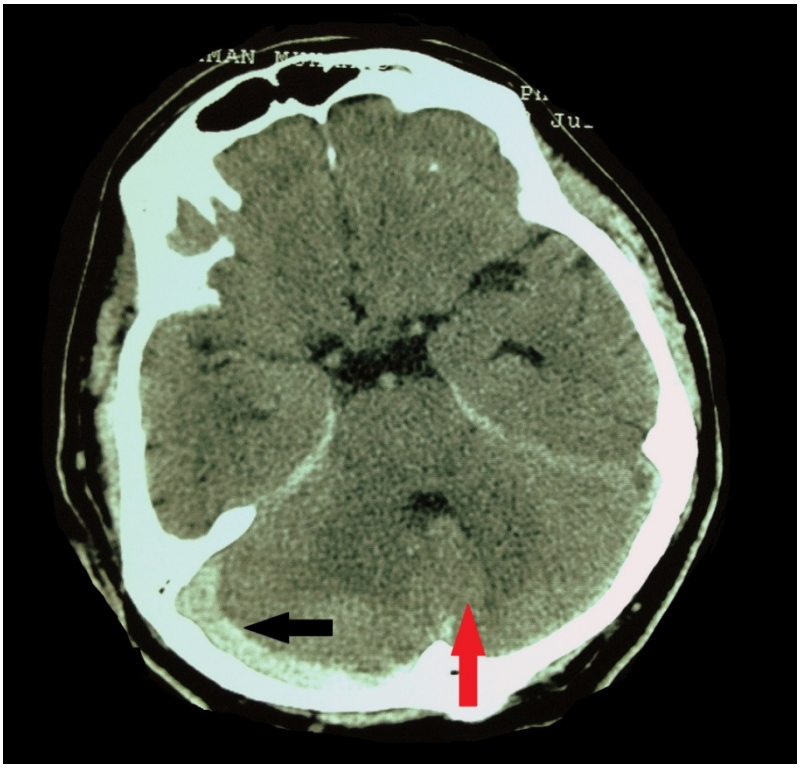

Figure 2. Non-contrast CT brain scan of the patient (level of mid-pons) at the time of Acute and Emergency department admission. Note the crescent-shaped, extra-axial, hyperdense area between the right occipital bone and the right cerebellar hemisphere (black arrow). This is posterior fossa acute subdural hematoma. The blood had also extended along the tentorium. The midline cerebellar vermis is displaced towards the left side (red arrow) and 4th ventricle is mildly compressed.

Analgesics, anti-emetics, intravenous fluids, and intravenous mannitol were prescribed and the neurosurgical department was consulted. The neurosurgical trainee missed the CT brain findings and suggested a brainstem ischemic stroke as an etiology behind the patient's presentation. The patient made a favorable improvement in terms of headache, vomiting, and consciousness within 3 days. On day 5, he requested to be discharged, as he intended to travel to Iran for further management. Therefore, we were unable to follow-up him clinically and neuro-radiologically. His self-discharge had transected with our management plan; we intended to do brain MRI and angiography. On the day of discharge, he was conscious and his stance and gait were normal. His blood pressure was 135/65 mmHg with a pulse rate of 90 beats per minute. Paracetamol tablets were prescribed on needed basis.

DISCUSSION

Intracranial supratentorial subdural hematomas (SDHs) are one of the commonest diseases encountered in the neurosurgical practice. Takeuchi and colleagues[8] analyzed 4315 patients with head trauma from 1995 to 2005. They found that 41(1%) their patients had traumatic hematomas of the posterior fossa and of those, only 10 had SDHs. However, the "spontaneous" development of acute infratentorial (posterior fossa) SDHs is a much rarer event. According to Isla et al[1] and Miranda et al[2] such hematomas constitute 0.6 to 1% of all intracranial hemorrhages. A spontaneous SHD refers to a hemorrhage occurring in the absence of provoking

factors, such as diffuse cerebral atrophy, head trauma, or anticoagulation therapy/bleeding tendency. According to Komatsu et al[9] and Ishii et al,[10] such spontaneous hemorrhages tend to have an arterial origin. In otherwise healthy patients without head trauma, a spontaneous rupture of a small bridging vein (especially in elderly people with marked brain atrophy), Berry aneurysm, or an arteriovenous malformation may be the culprit[2,12-14].

Head trauma is the comments cause behind the formation of SDHs in the posterior fossa and in half of those patients, a fracture of the occipital skull bones is found[3] Patients with coagulopathy or bleeding tendency may develop acute SDHs in the posterior fossa in the absence of head trauma[4-7]. The hemorrhage collects itself beneath the subdural space, between the occipital bones and the cerebellar hemispheres. Compression of the underlying cerebellum/brainstem and 4th ventricle therefore ensues, and the clinical presentation depends on the rapidity and amount of the blood present.

Posterior fossa SDHs therefore behave like a rapidly growing space-occupying lesion. A multitude of symptoms may result in the form of raised intracranial pressure, headache, vomiting, anisocoria, dysphagia, cranial nerve palsies, stance and gait ataxia, and even neck rigidity[2].

Cerebral hypoperfusion due to raised intracranial pressure or the mass effect itself may terminate in cerebral infarction. This is especially true in the posterior fossa, where the posterior cerebral arteries are vulnerable to compression along the free edge of the tentorium cerebelli[15-17].

In the absence of head trauma, most patients are usually clinically diagnosed with intracerebellar or subarachnoid hemorrhage and the correct diagnosis is not entertained until a cranial CT scan (and/or MRI) is done. Non-contrast CT scan of the brain is the best modality to diagnose these SDHs owing to its speed, relative simplicity, and widespread availability[18]. However, it is still sometimes difficult to detect a subdural hematoma in the infratentorial space because of the presence of bone artifacts; occurrence of isodense clots (especially in patients with anemia or coagulopathy); and the possibility of atypical forms of presentation leading to the wrong diagnosis of intracerebellar hemorrhage[2]. Gentry and coworkers found that 91% of SDHs ≥5 mm in thickness were identified on initial head CT while SDHs ≤3 mm in thickness were often missed initially[19].

Cranial MRI is more sensitive than CT scan for the detection of SDHs, especially for hematomas that are small in size, or those located adjacent to the tentorium cerebelli or interhemispheric fissure[20,21].

The pertinent medical literature mentions no randomized trials which might have compared neurosurgical decompression with conservative medical management for patients with SDHs, but limited observational data suggest that neurologically-stable patients with acute SDH who have small hematomas can be managed non-surgically[22-24].

Non-operative conservative treatment has been recommended for acute SDHs. Patients with coma at presentation who are clinically and neurologically stable (or improving); have no signs of brain herniation; and have a clot thickness of less than 10 mm and midline shift of less than 5 mm on initial cranial CT scan can be treated conservatively[25].

Our patient's presentation, clinical course, and imaging study have called for conservative management, as the overall presentation was relatively benign. The patient showed a favorable improvement clinically, but he requested an early discharged; he stated that he will go to Iran for further evaluation and management. Therefore, we have no radiological follow-up. We planned to do brain MRI and cerebral angiography.

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