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Case Report Open Access

A Bilateral Subdural Hematoma, Case Report

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Abstract

A 72 year-old male presented with deteriorating cognition and headache since 10 days. Two months earlier he fell on his head, after which an intracranial haemorrhage was excluded on CT. On physical examination, we found an impaired cognitive function and dysdiadochokinesia. A new CT-scan revealed an acute-on-chronic symmetrical bilateral subdural hematoma with active bleeding, compressing and balancing the brain in midline.

Keywords: Subdural hematoma; Brain injury trauma; Neurological disorders; Central nervous system injury; Morbidity and mortality of trauma; Trauma imaging

Case

A 72 year-old male was brought to our Emergency Department by ambulance services because of a progressive deterioration of cognitive functions of 10 days duration and a headache since that morning. Moreover, a tendency to fall to the left was present. There were no complaints of nausea or vomiting. His medical history only included glaucoma for which he used Latanoprost eye drops (Xalatan).

Two months earlier our patient fell on the back of his head after tripping over a lose tile. There were no neurologic symptoms at that time. A CT-scan of his skull and brain excluded an intracranial bleed or skull fracture and the patient was safely discharged home. His recovery was uneventfull until 10 days ago.

Neurologic examination in our ED revealed a wide based gait, a tendency to fall to the left, an inadequate coordination response and a dysdiadochokinesia of the right hand. His Glascow Coma Scale was maximal and further neurologic and general examination were unremarkable.

The CT-scan of skull and brain (Figure 1) revealed a relatively large bilateral subdural hematoma with possibly an small active haemorrhage in the rightsided frontolateral area. There was no midline shift because of the symmetry and there was no reduced patency of the basal cisterns.

Because of the slow onset of symptoms and the overall clinical appearence, the neurosurgeon opted for semi-elective surgery and admitted the patient for close observation with Dexamethasone 4 mg twice daily. During surgery the next day a bilateral burr hole was made, the dura mater was incised and access to both hematomas was achieved. Two partially liquified, dark redcoloured hematomas were evacuated. Information on the findings during surgery were not available to us. The neurosurgeon irrigated untill clear liquids returned and thereby excluded an active bleed on the right side. The wounds were closed with passive drains in situ, which did not return serosanguinolent liquids up till their removal the next morning. The patient recovered uneventfully without any persisting neurologic deficit and was discharged home after two days.

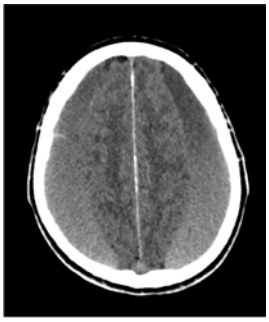

Figure 1: Subdural hematoma without midline shift and with active bleed in right frontoparietal area.

Discussion

Frequently a subdural hematoma (SDH) develops after traumatic brain injury [1]. Also, it can occur spontaneously in patients with significant cerebral atrophy, such as in the elderly, those with chronic alcohol abuse or patients with previous traumatic brain injury [1]. A SDH forms between the dura and the arachnoid membranes, when the bridging veins draining blood from the surface of the brain to the dural sinuses rupture spontaneously or by shearing forces in head trauma [2]. A SDH is usually venous in origin and is often self-limiting by the rising intracranial pressure. A SDH of significant size can disrupt the physiologic flow of cerebrospinal fluid and consequently raise the intracranial pressure [1,2].

If a SDH persist more than 3 weeks, it becomes a chronic SDH [1,2]. Until recently, a chronic SDH was thought to arise secondary to a high protein count and a subsequent osmotic fluid shift or expand secondary to an spontaneous recurrent bleeding. However, new evidence suggests that a chronic SDH enlarges because of recurrent spontaneous bleeding from a richly vascularized membrane encapsulating the hematoma. These bleedings are caused by a continuous process of angiogenesis, inflammation, coagulation and fibrinolysis. The precise mechanism is still unclear [2]. The expansion causes compression of functional cerebral tissue and thus causes neurologic deficiency [1,3].

As a consequence of chronic SDH, headache, light-headedness, somnolence, cognitive impairment and occasionally seizures may occur, even weeks after the initial (traumatic) event. A focal neurological deficit may or may not be apparent [1].

A chronic SDH can be unilateral or bilateral in nature. A bilateral chronic SDH can yield the same clinical picture as an unilateral SDH, depending on the interrelative size of the two lateral masses pushing and compressing the brain centrally [3].Our patient was diagnosed with a chronic SDH without midline shift due to the symmetrical bilateral

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involvement and therefore, we believe, the patiënt lacked significant focal neurologic deficiency.

The choice of treatment depends mostly on size of the hematoma, the Glascow Coma Scale, rate of deterioration, findings on neurologic exam and overall clinical appearance of the patiënt [3,4]. Treatment options are non-operative with admission for close observation and serial CT-imaging in small hematomas with little neurologic deficit, or, operative with burr hole trephination, craniotomy or decompressive craniectomy when elevated intracranial pressure, cerebral midline shift and/or signs of brain herniation are present [4]. In case of an active bleed, the bleeding vessel should be surgically identified and ligated or clipped [4].

Conclusion

Unilateral and bilateral chronic subdural hematomas (SDH) are well known to occur after traumatic brain injury or can originate spontaneously in patients with cerebral atrophy. Chronic SDH mostly presents with either slow cognitive or neurologic deterioration or with acute global or focal neurologic deficits.

Our patiënt, being diagnosed with a symmetrical bilateral SDH, compressing while balancing the brain in the midline was as such extraordinary to see for a professional working in emergency medicine care.

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