CASE REPORT

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Chronic subdural haematoma in a patient with arterial hypertension and Alzheimer's disease

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Abstract: An 82-year-old male patient was admitted to the Emergency Department 2.5 months after a minor head injury due to impaired consciousness, aphasia, and symptoms of left-sided paresis. The head CT scan showed a huge subdural haematoma by the right cerebral hemisphere. The patient was treated with right-sided craniotomy with evacuation of chronic subdural haematoma; his symptoms resolved and general condition improved.

Key words: subdural haematoma, arterial hypertension, Alzheimer's disease

INTRODUCTION

Chronic subdural haematoma (CSDH) is one of the most common clinical entities encountered in daily neurosurgical practice. The incidence of CSDH is 1-2 cases per 100,000 population per year, and more common in the old age group [1]. Chronic subdural haematoma is a well-defined clinical condition consisting in a slowly progressive accumulation of liquefied blood within the subdural space. Such a collection may, eventually, produce hemisphere compression and result in ultimate brain herniation.

Minor head trauma a few weeks before presentation of chronic haematoma is a common antecedent. Slow venous bleeding and the creation of neo-membranes around the subdural clot are some recognized pathogenic features in the development of CSDH. Head injury is the most common cause of this lesion, but several predisposing factors such as coagulopathy, alcoholism, cerebrospinal fluid shunt procedures, vascular malformations, seizure disorders, and metastatic tumours must also be considered [2].

The present paper describes the case of a patient who developed symptoms of chronic subdural haematoma following a minor head injury sustained two months earlier.

CASE REPORT

An 82-year-old male patient was transported by ambulance to the emergency department due to parietal pains, impaired consciousness, aphasia, and symptoms of left-sided flaccid paresis. The symptoms occurred on the day preceding admission to hospital. Two days before the onset of paresis symptoms, the patient played bridge. On admission, he was conscious, but logical contact was difficult. Arterial blood pressure was 140/90 mmHg. The physical examination revealed equal pupils reacting normally to light, muscle weakness of the left upper and lower limb, and positive Babiński`s sign on the left side. Heart action was rhythmic – 80/min. Vesicular sounds were heard above the lung fields. Abdominal integuments were soft, painful on deep palpation in the hypogastric region. Two and a half months before admission, the patients had sustained a minor head injury due to a fall following fainting without loss of consciousness. The CT carried out after that injury did not show features of heamatoma (Fig. 1).

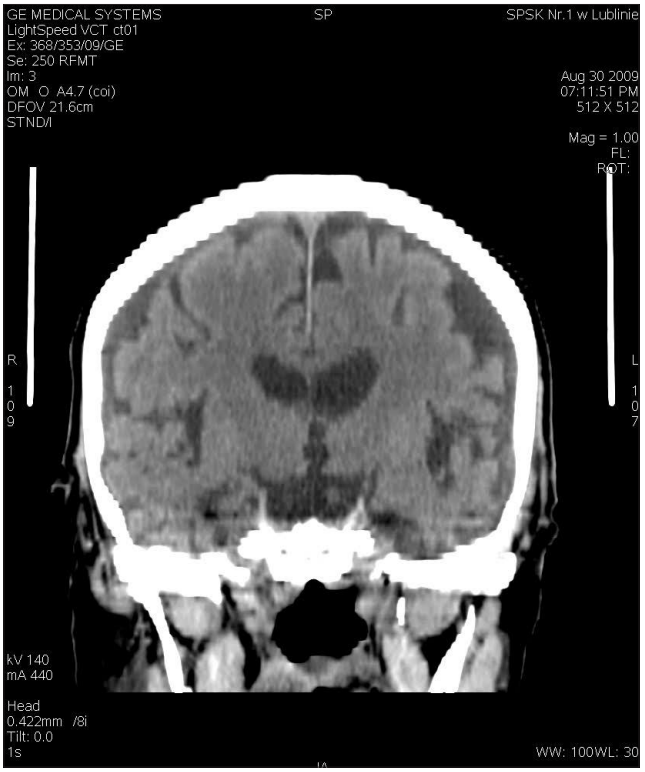

Figure 1 Coronal reconstructed image of the brain showing no signs of haematoma. Features of moderate cortical and subcortical brain atrophy are visible with slight enlargement of subdural liquid spaces.

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The patient has been treated for Alzheimer's disease for 9 years, and complicated arterial hypertension and glaucoma for 20 years. He had a 2-year history of post-renal failure, which was associated with urine flow blockage caused by a tumour in the urinary bladder. The lesion in the bladder induced periodic haematuria. The tumour was diagnosed on the abdominal ultrasonography scan performed 3 years before admission. Cystoscopic findings disclosed the presence of a big solid tumour affecting 2/3 of the urinary bladder. The tumour infiltrated the left ureter and prostatic ostium. The consulting urologist recommended palliative treatment. Moreover, 3 years ago the patient was treated for hyperthyroidism, receiving thiamazole 40mg/day.

Blood test findings showed microcyte anaemia with haemoglobin 10.5 g/dl (norm 13-18 g/dl), haemotocrit 32.7% (norm 40-54%) and MCV 78.3 fl (norm 80-94 fl) without increased WBC (White *blood cells).* The CRP level was 152 mg/l (norm below 5 mg/l). The level of glucose was normal, 100 mg/dl. The urine analysis revealed haematuria with albuminuria and leucocyturia.

The coagulation system was normal. Elevated levels of creatinine 4.3 mg/dl, urea 126 mg/dl (norm 18-55 mg/dl) and potassium 5.48 mmol/l (norm 3.5-5.1 mmol/l) in serum evidenced renal failure. Arterial blood gasometry showed metabolic acidosis with pH 7.3 (norm 7.35-7.45), and BE –5.7 mEq/L (norm –3-3 mEq/L). Concentration of bilirubin was normal, 0.65 mg/dl. The chest X-ray picture disclosed normally airy lungs, vascular hili. Heart silhouette was normal. ECG recordings showed regular heart rhythm accelerated to 100/min.

Abdominal ultrasonography scan revealed a hyperechogenic liver without enlargement. The right kidney was not enlarged and did not show features of urine retention, although marked dilation of the pyelocalyceal system in the left kidney was visible. The urinary bladder contained an infiltrating tumour – 24 mm.

The head CT scan showed the presence of a cerebral haematoma adjacent to the right hemisphere, 33 mm in width, with fluid layers of various densities, which demonstrated the chronic nature of the lesion with recent bleeding; right hemisphere was compressed and shifted to the left; deep structures of the brain were shifted about 13mm to the left. The ventricular system was asymmetric; the right lateral ventricle of the brain was constricted and displaced to the left (Fig. 2, 3). A fluid space – 37 × 27 mm – was observed at the base of the left temporal lobe. The facial skeleton structures had no evident traumatic lesions. Due to his life-threatening condition, the patient was operated on under general anaesthesia with wide temporo-parieto-occipital right craniectomy. After incision of the dura, the haematoma was evacuated spontaneously, and the rest of the clot and the formed capsule of hematoma were removed. The dura was closed and the space between dura and bone drained.

After surgery, the patient developed respiratory and circulatory failure and had to be mechanically ventilated. A continuous infusion of vasoconstrictor amines was administered due to low values of arterial pressure. The neurological and general status of the patient gradually improved. The patient was conscious, drowsy, periodically in logical contact, and responded to simple commands. The pupils were equal and reacted properly to light. The follow-up head CT showed partially reduced subdural haematoma and no features of oedema. The right lateral ventricle was decompressed. The patient was discharged home in good general condition, conscious and with logical contact.

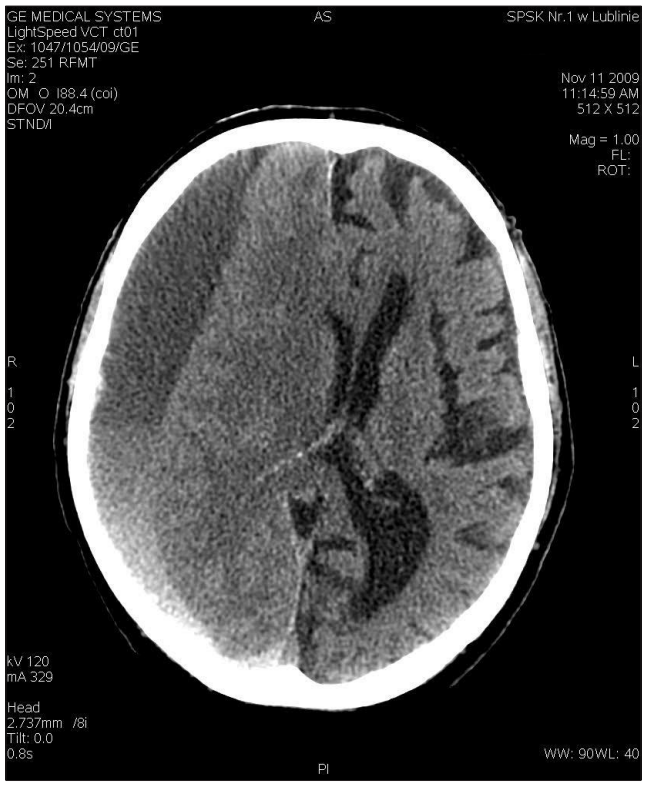

Figure 2 Axial CT image showing a large subdural heamatoma of the right hemisphere with signs of recent bleeding in its posterior part. Right hemisphere, right lateral ventricle, and deep structures of the brain are highly compressed and shifted to the left. The beginning of the brain herniation is observed.

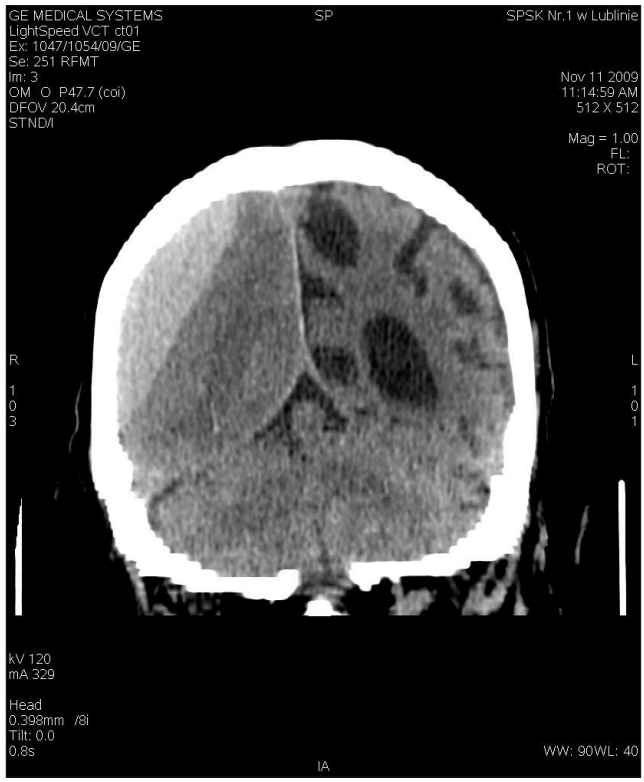

Figure 3 Coronal CT reconstruction image through the posterior cranial fossa showing a large area of bleeding within the right subdural haematoma. Brain parenchyma is edematous and compressed.

DISCUSSION

The development of subdural haematoma is favoured by atherosclerotic changes in the cerebral vessels. A slow and gradual increase in the volume of haematoma results in adjustment of the brain to the increasing pressure and may be asymptomatic for a long time. The unusual feature of chronic haematoma is hemiparesis on the affected side. Trauma occurs in 2/3 of haematoma cases. The trauma is very often mild and easily forgotten or overlooked by the aging patient so that its true place is difficult to estimate. Indeed, the distinction between the traumatic and the "spontaneous" haematoma (such as the one following a severe bout of coughing) is largely artificial: the more important factors are the low intracranial pressure, a mobile brain, and stretched fragile cortical veins traversing the subdural space. The most frequent presenting symptoms in the reported series have been intellectual deterioration and change in personality [3]. In the present case, symptoms suggestive of haematoma developed 2.5 months after a mild head injury. The first symptoms were disorientation and confusion, which were accounted for by Alzheimer's disease. The thorough physical examination and head CT demonstrated the presence of a chronic haematoma.

Another cause is the widespread use of anticoagulants in the elderly in cases of atrial fibrillation, valve replacements or coronary diseases. Traumatic subdural effusion (TSE) is also one of the main complications of blunt brain trauma and can be asymptomatic directly after the trauma. Ultimately, half of the asymptomatic TSEs evolve into chronic subdural haematomas. It is now hypothesised that TSE and CSDH are different stages, with different appearances of the same inflammatory reaction of the brain [4].

The typical location of chronic haematoma is the space above one of the hemispheres, frontal-temporal or parietal regions (bilateral in 15-20%).

During a period of 2-3 months, the haematoma liquid mass may increase, causing compression and displacement of the brain and consequently lead to various complaints: headaches (68%), dizziness, mental changes, drowsiness, altered consciousness (39%), hemiparesis (30%), aphasia, ataxia (30%), seizures – partial, or less commonly, generalized [5].

On a CT scan, a chronic subdural haematoma may have various imaging features ranging from an extensive concavo-convex, hypo-, iso- to a hyperdense area, depending on the age of the heamatoma and its degree of protein degradation. Acute SDHs appear highly hyperdense. Because the inner dural layer and arachnoid are not firmly attached, subdural haematomas are frequently seen layering along the entire hemispheric convexity, from the anterior falx to the posterior falx. The chronic SDH has a low-attenuation value similar to, but slightly higher than, CSF. It can be difficult to distinguish from the subarachnoid space in patients with cerebral atrophy. In such cases, intravenous contrast administration can be helpful by demonstrating an enhancing capsule or displaced cortical veins [6]. Rebleeding during evolution of a SDH appears as a heterogenous mixture of fresh blood and partially liquefied heamatoma, as observed in our patient. Such a sediment level may also be seen in patients with clotting disorders, and such a possibility should also be taken into consideration.

During the transition from the acute to the chronic SDH, usually 1-3 weeks after the trauma, an isodense phase appears. At this time, recognition of indirect imaging findings, such as effacement of cerebral sulci, displacement of gray matter with wavy white matter, and midline shift on a noncontrast CT scan, are important to establish a proper diagnosis [6].

Sometimes, in cases of isodense haematomas, CT with a contrast agent or MRI is recommended to better visualise the isodense area, which appears enhanced [7].

The treatment of subdural haematoma involves surgical decompression. For many years, surgery-treated CSDHs by craniotomy was the optimal technique, in spite of the high surgical mortality rate of up to 30%; it is now rarely indicated, except for the treatment of repeated recurrence of the haematoma or solid consistency of the clot.

The present indications for craniotomy involve the following situations:

a) when the subdural collection reaccumulates;
b) solid haematoma is present;
c) the brain fails to re-expand and obliterates the subdural space [8].

In our patient, the wide craniotomy turned out to be a successful method of treatment. Within a few days after surgery, the left hemiplegia retracted, the mental state of the patient improved, and 21 days after the surgery, the patient was discharged from hospital in a satisfactory condition.

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