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Case report

A patient with acute aortic dissection presenting with bilateral stroke – A rare experience

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ABSTRACT

Acute aortic dissection is a rare, life-threatening condition requiring early recognition and proper treatment. Although chest pain remains the most frequent initial symptom, clinical manifestation of aortic dissection varies. Rarely aortic dissection starts with neurological symptoms such as ischemic stroke, which is usually right-sided. A danger of performing thrombolytic therapy in these patients exists if aortic dissection is overlooked. Herein, we present a case of a patient with acute aortic dissection without typical chest pain whose initial manifestation was bilateral stroke. The uncommon presentation which masked the underlying condition delayed implementation of appropriate management. Moreover, the late admission to hospital prevented the patient from administration of recombined tissue plasminogen activator that would certainly decrease chances of survival. Presented case highlights the need for thorough physical examination at admission to hospital in all patients with acute stroke and points out the necessity of proper clinical work-up including adequate aorta imaging modalities of patients with acute stroke and suggestive findings of aortic dissection.

1. Introduction

Acute aortic dissection is a challenging clinical emergency. The risk of death is high but chances of survival can improve when it is diagnosed early and treated promptly. Usually the symptoms of aortic dissection are characteristic and include sudden severe chest or upper back pain, often described as a sharp, knife-like or tearing sensation that radiates to the neck or down the back, weak pulse in one arm compared to the other, shortness of breath or syncope. It becomes more difficult when aortic dissection starts with neurological symptoms, which often are dramatic and mask the underlying condition [1,2]. We present an unusual case of a patient with acute aortic dissection whose initial manifestation was bilateral stroke.

2. Case report

A 58-year-old man felt numbness followed by muscular weakness of his left limbs at 01:00 am, after quarrel with his son. He called emergency medical services, but because his

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symptoms gradually decreased he was not taken to hospital. Later, in the early morning the patient had seizure-like episode: convulsion of the left upper limb, he was somnolent, passed urine involuntary and his wife told that his speech was mumbling. He was than admitted to the Department of Neurology.

Anamnesis revealed untreated hypertension. On admission his blood pressure was 130/80 mmHg. In the initial neurological examination the patient was conscious, little somnolent, in logical contact, complied the commands, limping on left lower limb, had problems with balance control, eyeballs were in opposite position, pupils were equal, reactive, his face was symmetrical, his speech was intelligible, there was small degree monoparesis of upper left limb, loss of pain sensation on the left side, hyporeflexia of left plantar reflex, hyperreflexia of left tendon reflexes, left corneal reflex slightly decreased.

Laboratory tests were within normal limits apart from leucocytosis, small degree anemia, and elevated creatinine level (Table 1).

A CT scan of the head performed on admission revealed no hypodensity or hemorrhage. The initial diagnosis was stroke and intravenous tissue plasminogen activator was considered at this point; however, the patient was disqualified from thrombolytic therapy because of low National Institutes of Health Stroke Scale (NIHSS) score (5 points), long time from onset of symptoms and seizures at the beginning of the disease.

Due to seizure-like episode electroencephalogram (EEG) was scheduled and carotid ultrasound was planned as a routine evaluation.

On the second day of hospitalization, new symptoms occurred – aphasia and right hemiparesis. Second CT scan of the head with contrast revealed ischemic lesions in both hemispheres – ischemic lesion in frontal and parieto-occipital right region and left upper frontal and cingulate gyri (Fig. 1).

Laboratory test revealed elevated C-reactive protein – 82.1 mg/L. A chest X-ray showed intensified lung markings and parenchymal density in right lower lung field and shading 4 cm × 6 cm in the upper pole of right pulmonary hilus.

Additionally, blood pressure dropped, requiring fluid resuscitation and therefore the patient was consulted by a cardiologist. On the basis of transthoracic echocardiography a suspicion of aortic dissection was raised. A CT scan of the chest confirmed dilation of the ascending aorta – 50 mm, the descending aorta – 41 mm, the abdominal aorta – 28 mm and the aortic wall dissection of type I DeBakey (Fig. 2). Diameter of false duct in dissected aortic was 12 mm in the arch and exceeded true lumen in further parts of the aorta. The dissection reached common iliac arteries, internal and external iliac arteries. Right renal artery and coeliac trunk arose from aortic lumen, but left renal artery arose from false duct lumen. Arteries branching off from aortic arch were not visualized.

Table 1 – Laboratory test at admission to Emergency Room.

Parameter	Level
WBC	13.3×10^9/L (3.8–10)
RBC	4.27×10^{12}/L (4.20–6.0)
HGB	13.1 g/dL (14.0–18.0)
HCT	37.4% (40.0–54.0)
PLT	172×10^9/L (140–440)
INR	1.1 (0.7–1.2)
APTT	31.1 s (23.6–34.8)
Na	140 mmol/L (136–145)
K	4.0 mmol/L (3.5–5.1)
Creatinine	136 μmol/L (62–106)

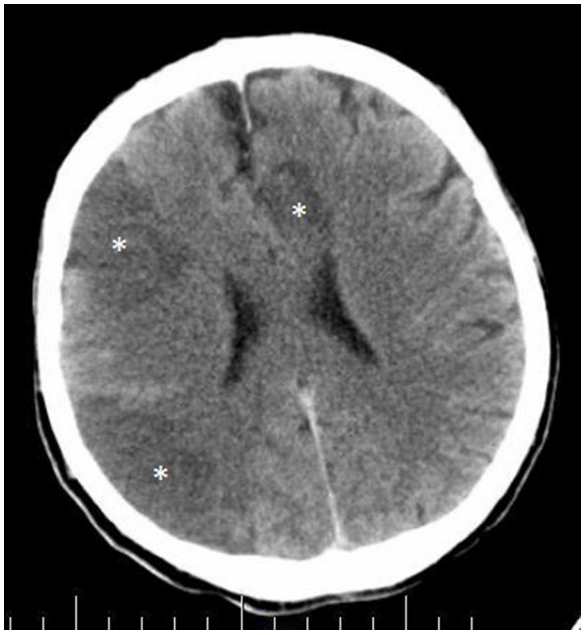

Fig. 1 – Ischemic lesions in both hemispheres (*marked with asterisks*) – ischemic lesion in frontal and parieto-occipital right region and left upper frontal and cingulate gyri.

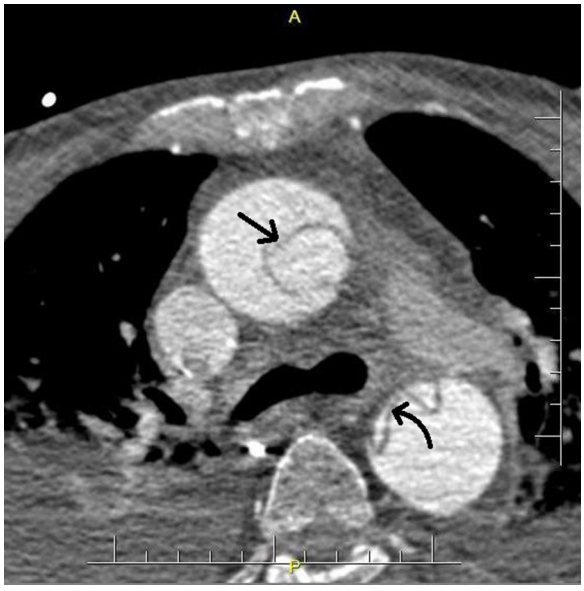

Fig. 2 – A CT scan of the chest revealing dissection of type I DeBakey. Dissection of ascending (*straight arrow*) and descending (*curved arrow*) portion of the aorta.