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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.

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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)

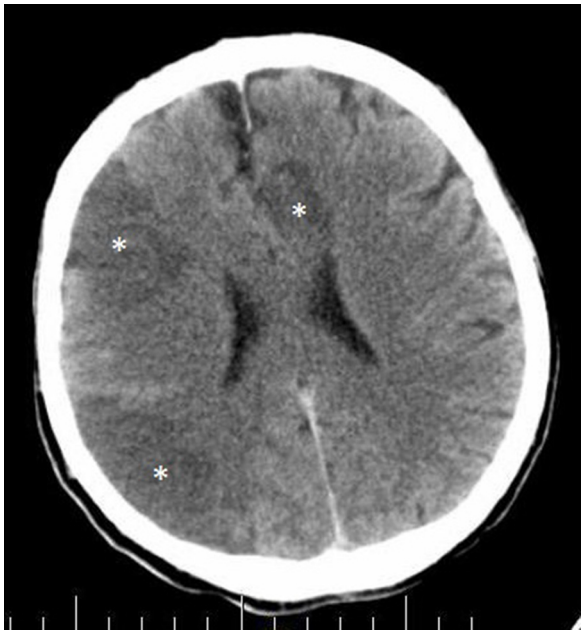

Fig. 1 – Ischemic lesions in both hemispheres (*marked with asterisks*) – ischemic lesion in frontal and parieto-occipital right region and left upper frontal and cingulate gyri.

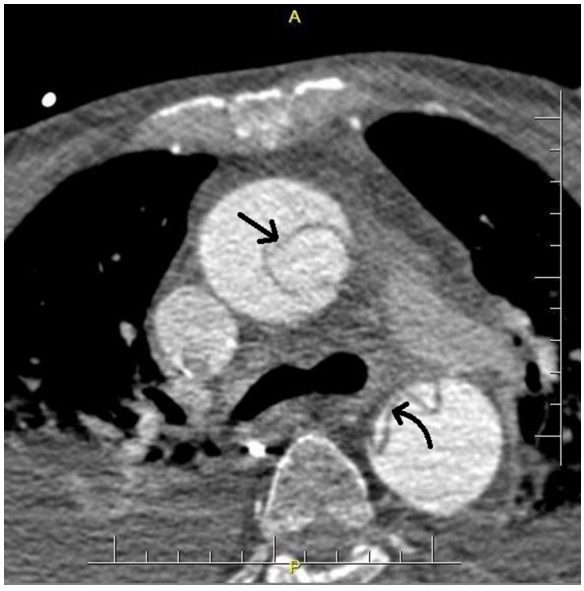

Fig. 2 – A CT scan of the chest revealing dissection of type I DeBakey. Dissection of ascending (*straight arrow*) and descending (*curved arrow*) portion of the aorta.

The patient was immediately transferred to the Heart Surgery Department and qualified to operation because of life saving indications. The patient's general condition decreased rapidly. At the moment of transfer the patient was unconscious, reactive to pain. Another CT scan of the head performed in the Heart Surgery Department excluded hemorrhagic transformation. Angio-CT of the aorta showed dissection of the aorta, including innominate artery, right common carotid artery, right internal carotid artery bulb, left common carotid artery and left internal carotid artery bulb and left subclavian artery. Vertebral arteries were patent (Fig. 3).

Operation was under extracorporeal circulation, in deep hypothermia. The prosthesis GELSOFT 24 mm was placed and angioplasty of the ascending aorta was made. Epicardial electrode was sutured. In perioperative period patient was in serious condition, needed inotropic support, blood-based products and antibiotics – initially empirical broad-spectrum later targeted.

Due to recurrent right pleural effusion and frequent drainages, CT of thorax was made showing in both lungs extensive atelectatic and infiltrative lesions in dorsal segments, fluid in pleura on both sides – up to 3 cm on left and 1 cm on right side.

Due to persistent serious condition after operation, CT scan of the head was repeated, revealing hemorrhagic transformation of ischemic stroke regions and subarachnoid hemorrhage (Figs. 4 and 5).

The patient was consulted with neurosurgeon and disqualified from operation. In neurological examination the patient was unconscious, sedated, intubated, ventilated in SIMV mode, pupils diameter 2 mm, without reaction to light, weakened ciliary and corneal reactions, flaccid paresis of four limbs, tendon reflexes preserved, no plantar reflexes, ankle clonus on the both sides.

Angio-CT of the head excluded malformation of brain vessels. In the subsequent neurological examination the patient reacted to voice, opened his eyes, concentrated sight, tension decreased in four limbs, deep reflexes were symmetrical, plantar reflexes preserved.

During the course of hospitalization, patient's general condition continued to gradually improve, thus intensive rehabilitation was implemented. Echocardiographic examination after 3 weeks from operation showed: left ventricle ejection fraction (LVEF) of 35%, left ventricle with general loss

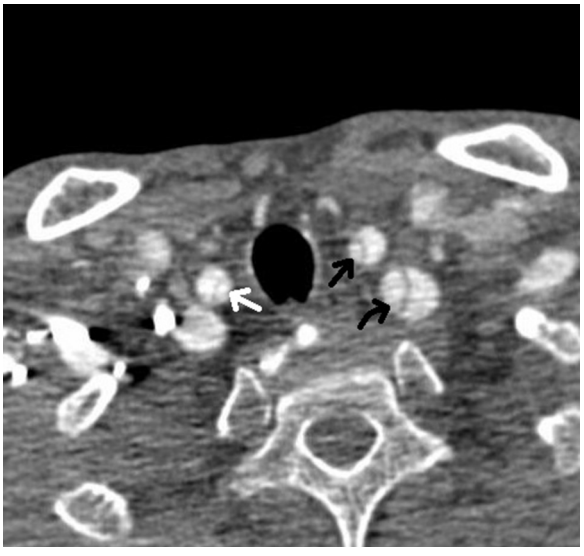

Fig. 3 – Angio-CT of the aorta showing dissection of right common carotid artery (*white arrow*), left common carotid artery (*black straight arrow*) and left subclavian artery (*black curved arrow*).

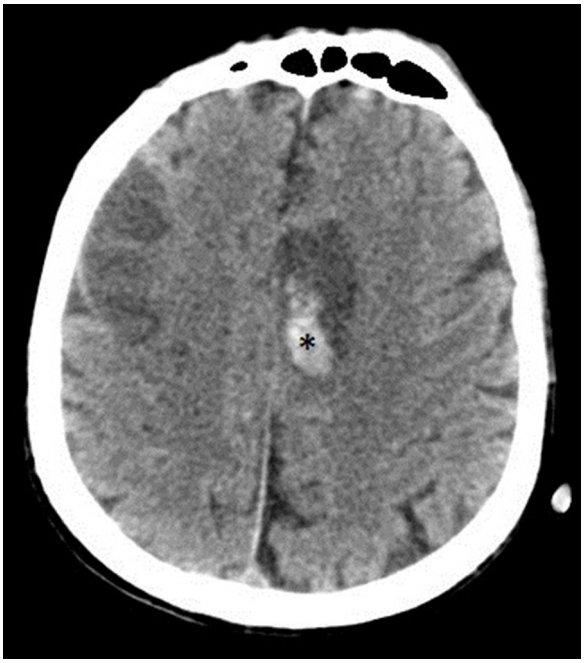

Fig. 4 – Hemorrhagic transformation of ischemic stroke regions (*marked with asterisk*).

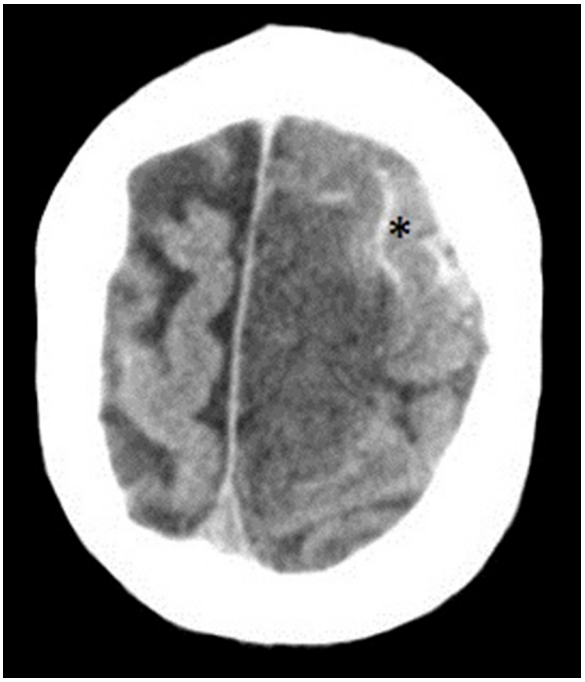

Fig. 5 – Subarachnoid hemorrhage (*marked with asterisk*).

of contractility, no signs of tamponade and no hypertrophy of left ventricle. After 40 days in Intensive Care Unit the patient was transferred to the Department of Neurology.

In the neurological examination during the day of transfer: the patient was conscious, in logic contact, but reluctant to talk, with reduced global muscles' strength, spastic hemiparesis on the right side (3/5 in Lovett scale in lower limb and 4+/5 in Lovett scale in upper limb), no Babinski signs, hyperreflexion of right tendon reflexes. Echo examination that day showed: normal size and thickness of left ventricle wall, lower contractility – akinesia of apex, small degree hypokinesia of other segments; LVEF 45%, right chambers of heart not enlarged, the ascending aorta diameter – 38 mm.

Because of fever and persistent lesions in the chest X-ray, antibiotic therapy was modified but four days later on the base of culture – antimycotic treatment was initiated.

Unfortunately on the 6th day of hospitalization in the Department of Neurology the patient's condition decreased rapidly. Hypotension and oliguria occurred, requiring catecholamines and diuretics. He was conscious, but without logic contact. Later he developed heart failure with peripheral oedema. He was tachycardiac, with low saturation of about 73–85%, anuria occurred. On the next day sudden cardiac arrest in the mechanism of asystolia occurred and despite 45 min resuscitation patient died on the 47th day after operation of aortic dissection. Post mortem examination revealed extensive thrombus on the aortic graft which closed the lumen of the aorta.

3. Discussion

Aortic dissection is a rare condition with an incidence ranging from 5 to 30 cases per million people per year [3]. Moreover, ischemic stroke induced by aortic dissection is not common among patients with aortic dissection reaching 15.7% according to Gaul et al. [2]. Although, majority of patients with neurological symptoms at the onset of dissection present with initial chest or back pain, up to one-third of patients give no history of significant pain. Moreover, half of patients with aortic dissection who do not report typical chest pain show neurological symptoms at the beginning. The frequency of aortic dissection with neurological involvement varies from 17 to 40% [2,4,5]. The most common initial neurological finding in patients with aortic dissection is ischemic stroke – it stands up for 53.3% of those with neurological symptoms followed by ischemic neuropathy (36.7%), somnolence (13.3%) and tonic-clonic seizures (10%). Neurological symptoms are often transient, fluctuating, and fully remitted before the patient reaches emergency room. Neurological manifestations usually appear at or soon after the onset of dissection. Rapid improvement in such cases is probably the result of only temporary arterial occlusion at the moment of propagation of the dissection [2]. Furthermore, strokes associated with aortic dissection are predominantly right-sided – in different publications described as 69.2–71% of all strokes [2,6] and thus it is not a common presentation for aortic dissection to begin with bilateral stroke. Tendency to be right sided frequently is accounted for the fact that carotid artery origins are much more vulnerable to the advancing dissection because of their proximity to the aortic arch [2,4,7,8]. Strokes that occur in patients with aortic dissection after surgical treatment are often bilateral. This fact is probably connected not only with extension of dissection toward supra-aortic vessels but also with various perioperative factors such as circulatory arrest or thromboembolism during operation [8,9]. DeBakey type I aortic dissection is the most common and dangerous type of aortic dissection, involving a tear in the ascending and descending portion of the aorta. The most common predisposing conditions are hypertension (65.3%), obesity (35.6%) and cigarette smoking (31.7%) [2]. Surgery is the treatment of choice in aortic dissection type A [1]. Estrera et al. in their publication from 2006 suggested that stroke does not contraindicate surgical repair in patients with type A aortic dissection. In their examined group of Stanford A type aortic dissection there were 10.6% of patients (16 persons) with stroke at the beginning, usually accompanied by a typical chest pain. Operation was started at different time from the beginning of symptoms. As a result improvement was noticed only with those who had undergone operation during first 10 h from the beginning of symptoms. Overall hospital mortality in this group was 18.8% [10]. In other publications, where group of patient consisted of those with neurological symptoms at the onset, without typical chest pain, mortality was higher. In Gaul et al. publication it reached 30% while overall mortality among all patients with aortic dissection was 22.6% [2]. Lee et al. presented their group with overall mortality among Stanford A type aortic dissection of 15.8% and among patients with neurological symptoms at the beginning – 43.9% mortality [9]. This examples show that neurological symptoms at the beginning, masking underlying condition decrease the chances for correct diagnosis and stand for higher risk of death among the group of patients with aortic dissection. However, when aortic dissection is recognized early, neurological symptoms are not necessarily associated with an increased mortality [2]. Herein, the time to operation was more than 24 h that decreased chances of survival. Even though acute aortic dissection is a contraindication to thrombolysis [11], there is a danger of performing thrombolytic therapy if aortic dissection is overlooked. In the presented case the patient's late admission to hospital prevented thrombolysis implementation that would certainly increase the risk of early death. However, there are numerous reports in literature of recombined tissue plasminogen activator (rt-PA) therapy in patients with acute aortic dissection starting with acute stroke. Mortality rate in such patients is approximately 71% [12–17]. On the other hand the incidence of stroke far exceeds that of aortic dissection, and most feel it would be a disservice to delay thrombolytic therapy for a large number of patients who could benefit in order to protect the minority of those with aortic dissection. Because aortic dissection is a rare cause of ischemic stroke, the aggressiveness of screening for this entity is questioned [18]. Nevertheless, presented case sets out clearly the need for a proper and effective clinical work-up of patients with acute stroke and suspicion of aortic dissection, especially those admitted to emergency room within fibrynolytic window. The protocol for acute stroke includes chest X-ray during hospitalization if not performed within previous 6 months [11]. However, up to 20% of chest X-rays may be negative in patients with aortic dissection revealing no

widening of superior mediastinum [19]. Although transthoracic echocardiography (TTE) does not allow for a full assessment of the aorta, it is useful for diagnosis of some aortic segments including the aortic arch. Its additional advantages are non-invasiveness and widespread availability at the point of care.

Chest CT scanning and transoesophageal echocardiography, while very sensitive for aortic dissection, are time consuming and expensive and should be considered in patients with acute stroke only if aortic dissection is highly probable [18].

Hence, we propose to consider the use of TTE focused on the aorta similarly to the one used in patients after trauma and commonly known as Focused Assessment with Sonography for Trauma (FAST). Focused Assessment with Sonography for The Aorta (FAST-A) including suprasternal and parasternal views would serve as a rapid bedside examination for patients with acute stroke and suspicion of aortic dissection, especially those considered for thrombolytic therapy. It seems appropriate to consider FAST-A as a routine screening tool only in patients with acute stroke and at increased risk of aortic dissection (men over 50 years old, with a history of hypertension, obesity or cigarette smoking) while avoiding delay in initiating thrombolytic therapy is of paramount importance for evaluation of patients with acute stroke. The utility of FAST-A as a rapid screening tool to exclude aortic dissection in patients with acute stroke is to be established.

4. Summary

We believe that thorough physical examination including heart auscultation, peripheral pulse assessment and blood pressure monitoring in all patients with acute stroke is necessary in order not to overlook a patient with aortic dissection presenting initially with neurological symptoms. In case of suggestive findings of aortic dissection (chest or back pain or significant trauma, hypotension, reduced peripheral pulses, aortic regurgitation murmur) further evaluation including adequate aorta imaging modalities is required, particularly when communication with the patient is impaired. While chest CT scanning and transoesophageal echocardiography are time consuming and should be reserved for patients with a strong suspicion of aortic dissection, we propose the use of TTE focused on the aorta as a potential adjunct to existing stroke protocols in patients at increased risk of aortic dissection, especially when thrombolytic therapy is considered. This could reduce the risk of fatal rt-PA administration in patients with acute aortic dissection starting with acute stroke. The utility of this approach is to be evaluated.

Conflict of interest

None declared.

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None declared.

Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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