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Clinical Case Report

Crucial role of carotid ultrasound for the rapid diagnosis of hyperacute aortic dissection complicated by cerebral infarction: A case report and literature review

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ABSTRACT

Aortic dissection is a life-threatening rare condition that may virtually present by any organ system dysfunction, the nervous system included. Acute cerebral infarction among multiple other neurological and non-neurological presentations is part of this acute aortic syndrome. Rapid and correct diagnosis is of extreme importance keeping in mind the possibility of thrombolytic treatment if a patient with a suspected ischemic stroke arrives to the Emergency Department within a 4.5-h window after symptom onset. Systemic intravenous thrombolysis in the case of an acute brain infarction due to aortic dissection may lead to fatal outcomes. In this neurological emergency it is important to rule out underlying aortic dissection by choosing appropriately quick and accurate diagnostic tool. We aimed to present a prospective follow-up case, where carotid ultrasound examination was the primary key method that led to a correct diagnosis in hyperacute (<24 h) Stanford type A aortic dissection presenting as an acute ischemic stroke, and thereafter with a repeated contrast-enhanced computed tomography and transthoracic echocardiography, helped to monitor topography of intravascular processes and hemodynamic properties during the clinical course of a disease, which influenced treatment decisions. Thus, we reviewed the literature mainly focusing on the various neurological aspects associated with aortic dissection.

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1. Introduction

Aortic dissection is a life-threatening vascular emergency that is characterized by disruption of the arterial wall integrity. Sometimes the condition is called a catastrophe of the principal artery of the body, which is associated with a great diversity of clinical presentations which may be caused by dysfunction of virtually any organ system. It is important for every clinician not to miss this underlying condition, since it is rarely seen in clinical practice. The incidence of aortic dissection is low, and estimated to be 3–6 per 100 000 persons per year [1–3].

Timely diagnosis determinates the clinical outcomes. If untreated, the mortality rate is 25% in the first 24 h, 50% in 48 h, 75% by two weeks [4,5]. Rapid and correct diagnosis might unfortunately be delayed due to the fact that the imaging studies of the largest sensitivity and specificity (e.g. contrastenhanced computed tomography (CT)) are not of the first-line investigations in the Emergency Department (ED), and they are time consuming (magnetic resonance imaging (MRI), repeated imaging by ECG-synchronized CT, transesophageal echocardiography (TEE)), or invasive (conventional aortography) [3,5,6]. Furthermore, these imaging methods may only be available in specialized cardiovascular referral centers.

Acute cerebral infarction among other multiple neurological and non-neurological presentations is part of this acute aortic syndrome (AAS). AAS constitute a spectrum of conditions characterized by disruptions in the integrity of the aortic wall that may lead to potentially catastrophic outcomes. They include classic aortic dissection, intramural hematoma, and penetrating aortic ulcer [3]. The diagnosis of aortic dissection can be challenging and not straightforward due to atypical presentations [7–9]. According to the guidelines of the European Stroke Organization and the American Heart Association/Stroke Association, intravenous administration of alteplase (recombinant tissue-type plasminogen activator, r-tPA) within 4.5 h of symptoms onset and/or endovascular treatment remain the only approved therapy that may reverse neurological deficit in patients with acute ischemic

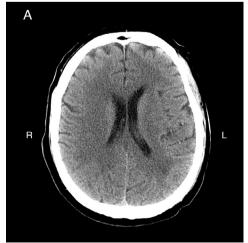
stroke (IS) [10–12]. A consulting neurologist takes full responsibility on the decision of whether to perform systemic intravenous thrombolysis (IVT) in acute IS or if a patient is ineligible. IVT with r-tPA in the case of acute brain infarction caused by aortic dissection may lead to lethal outcomes [10,13–15].

2. Case presentation

A 45-year-old-man with no remarkable previous medical history, except smoking, was admitted to the ED of the Hospital of Lithuanian University of Health Sciences Kauno Klinikos with a sudden onset of left-sided hemiplegia. The patient was presented within 2.5 h after the first symptoms. Earlier that day the patient felt moderate pain in the right shoulder irradiating to the neck, which occurred after some physical activity at home. He took non-steroidal anti-inflammatory medications and pain evidently reduced.

The initial evaluation in the ED found his arterial blood pressure (BP) was 134/70 mmHg and the heart rate (HR) was 45 beats per minute. In the neurological examination the patient was conscious, alert, slightly anxious, showing signs of left-sided weakness of lower facial muscles and the left hemiplegia. In addition, a sensory decrease on the left side of the body, partial gaze palsy, partial hemianopia, unilateral hemispatial inattention, and severe dysarthria were present. The National Institutes of Health Stroke Scale (NIHSS) score was 17 points. Acute IS was suspected and the patient was considered a candidate for systemic IVT. The initial urgent cranial CT scan revealed no signs of acute brain infarction or hemorrhage (Fig. 1, panel A).

During the preparation for IVT, a sudden symptomatic hypotension (96/50 mmHg) accompanied by bradycardia (40 beats per minute) developed while in the ED, which significantly worsened the patient's neurologic status. During this episode, hemodynamic instability with altered consciousness developed, which became a contraindication for IVT. The patient was consulted by a cardiologist, however, no acute coronary syndrome was suspected by the specialist.



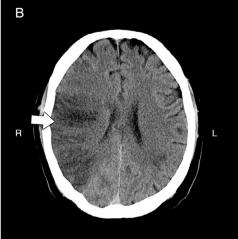


Fig. 1 – Initial (on day 0, panel A) and follow-up (on day 3, panel B) cranial computed tomography (CT) images. Arrow indicates signs of ischemic brain infarction in the right middle cerebral artery blood supply zone on the repeated brain CT. R, right; L, left.

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