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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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ARTICLE INFO

Article history:

Received 24 January 2016

Received in revised form

14 September 2016

Accepted 8 November 2016

Available online 19 November 2016

Keywords:

Aortic dissection

Aortic imaging

Carotid ultrasound

Cerebral infarction

Neurological complications

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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Peer review under the responsibility of the Lithuanian University of Health Sciences.



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<http://dx.doi.org/10.1016/j.medici.2016.11.001>

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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. contrast-enhanced 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.

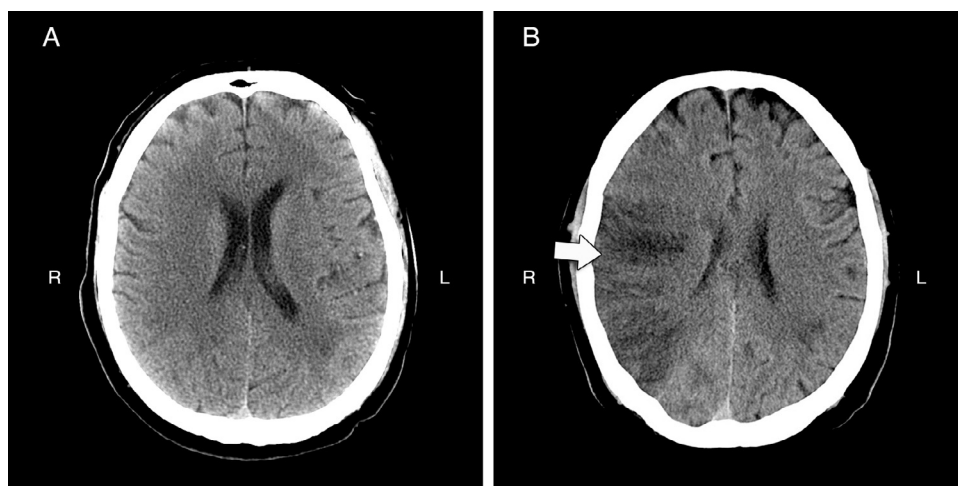


Fig. 1 – Initial (on day 0, panel A) and follow-up (on day 3, panel B) cranial computed tomography (CT) images. Arrow indicates signs of ischemic brain infarction in the right middle cerebral artery blood supply zone on the repeated brain CT. R, right; L, left.

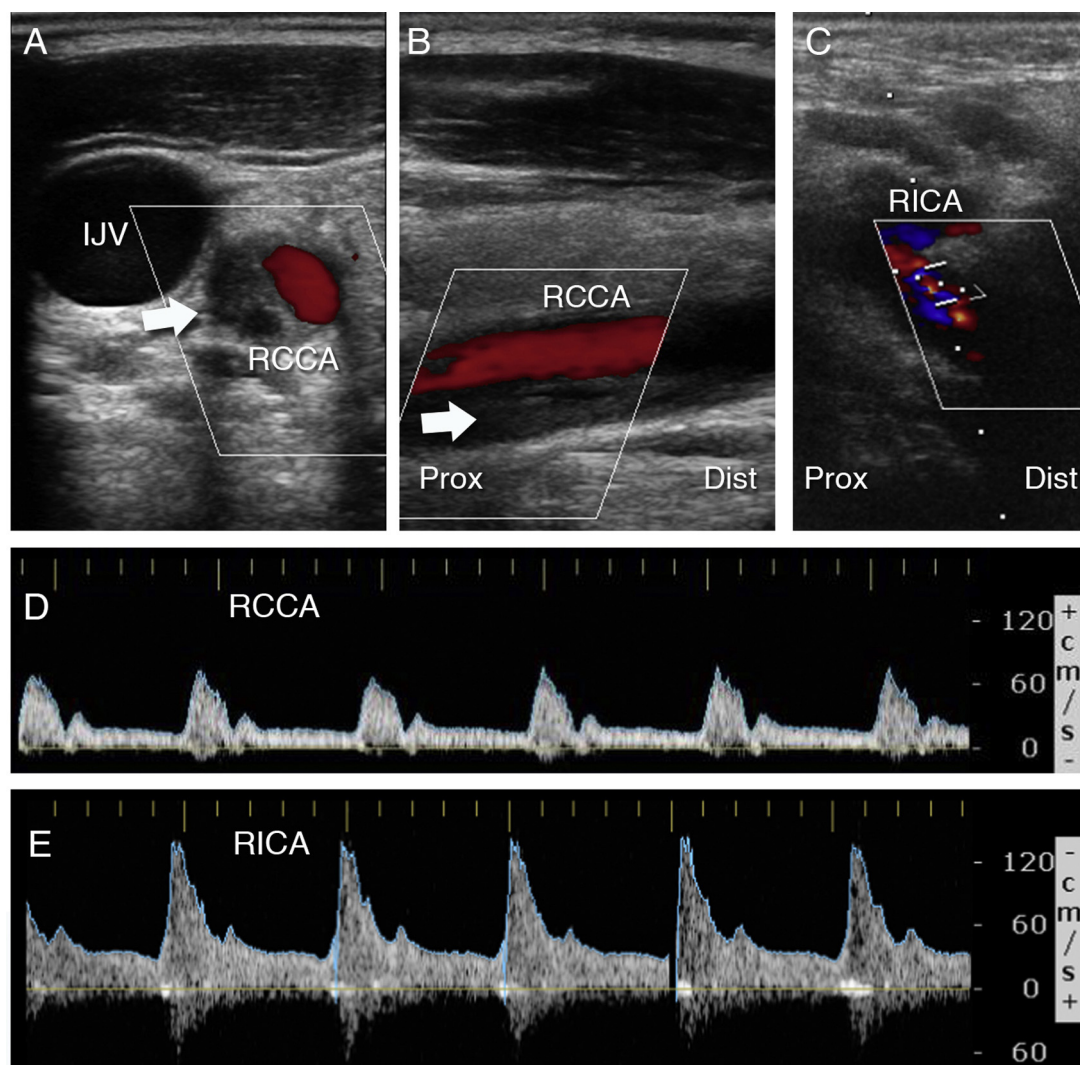


Fig. 2 – Urgent duplex ultrasound images of extracranial vessels on day 1. Arrows mark hyperechogenic signal along all the length, mostly lower part of the RCCA (panels A and B) extending toward RICA (panel C). Doppler blood flow velocities in the RCCA were in normal ranges, however, the shape of Doppler curve was abnormal (panel D), and altered in the RICA (panel E). RCCA, right common carotid artery; IJV, internal jugular vein; RICA, right internal carotid artery; Prox, proximally (from the brachiocephalic trunk); Dist, distally.

The patient's hemodynamic condition was stabilized after intravenous injection of atropine and infusion of crystalloid fluids. The neurological deficits recovered to the status of hemiparesis after the patient's HR increased to 80 beats per minute and BP roused to 110/70 mmHg resulting in the NIHSS score of 13 points. However, a time window for IVT had already exceeded 4.5 h. The patient's vital functions remained stable and for further investigations he was transferred from the ED to the Department of Neurology (DN).

On day 1 in the DN, carotid duplex ultrasound was urgently performed. It revealed hyperechogenic signal all along the length of the right common carotid artery (CCA) wall (the most evident abnormal features were detected in the lower part of the right CCA, in its lateral, posterior walls), which extended toward internal carotid artery (ICA) (Fig. 2). Changes in the linear blood flow velocities were observed, especially in the right ICA, which were altered (peak systolic velocity 141 cm/s,

end-diastolic velocity 29 cm/s; increased carotid index (ICA/CCA) which equaled 1.96); flow deceleration in the right CCA 72/15 cm/s; Pourcelot indexes (RI) of the right CCA 0.793 and ICA 0.789) with waveform changes. Examination of the vertebral arteries revealed a smaller diameter (2.91 mm) of the right vertebral artery compared to the left one (5 mm), but without impaired Doppler profile in these vessels.

Acute aortic dissection was suspected and urgent contrast-enhanced CT of thoracic aorta was subsequently performed. This showed dilation of its ascending part of up to 4.8 cm. The diameter of aortic arch was 3.9 cm and non-contrasting thrombosed lumen 0.8 cm in diameter form embranchment of brachiocephalic trunk, with extension to the right CCA being evident (Figs. 3, panel A and 4). Transthoracic echocardiography (TTE) revealed third grade aortic valve insufficiency. The patient was then immediately consulted by a cardiothoracic surgeon. The specialist decided not to perform urgent aortic

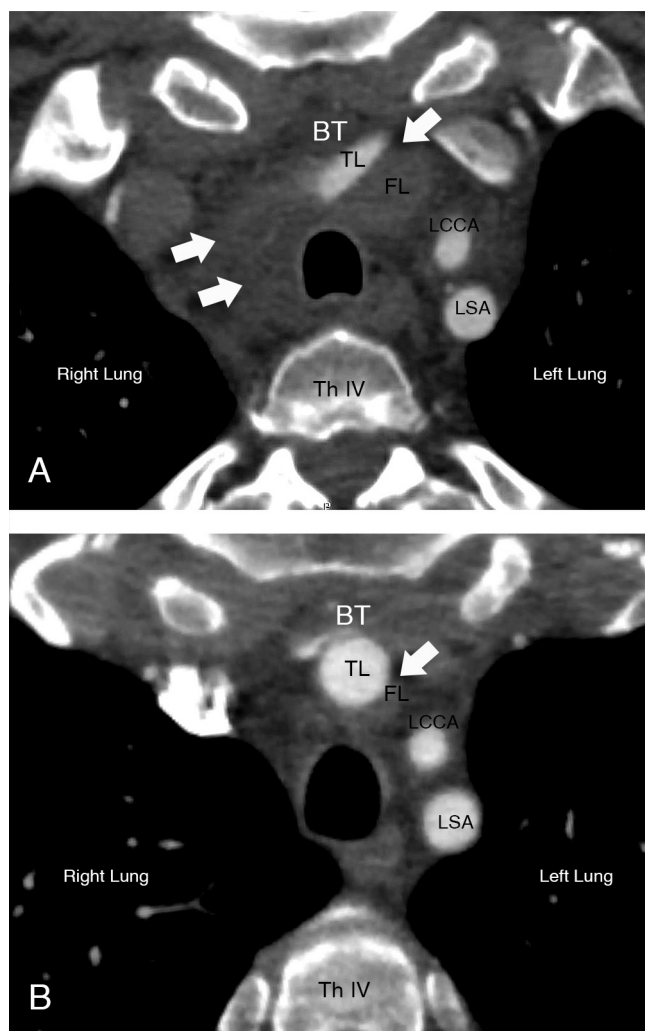


Fig. 3 – Contrast-enhanced CT angiography of the thoracic aorta on day 2 (panel A) and after 4 months (panel B); axial slice through aortic arch branch level. Arrows on the right shows thrombosed “false” lumen of BT which is more prominent in the acute stage of dissection. Two arrows indicate mild tissue infiltration in the right upper part of mediastinum (panel A), which had reduced later on follow-up image (panel B). BT, brachiocephalic trunk; TL, “true” lumen; FL, “false” lumen; LCCA, left common carotid artery; LSA, left subclavian artery.

surgery, and the decision was based on the thrombosed arterial lumen on CT images and low risk of rupture, thus only conservative medical management strategy was recommended with repeated imaging.

Further clinical course of the patient was complicated by malignant arterial hypertension (AH), meaning part of the in-hospital admission time (13 days out of 41 day) was spent in the Intensive Care Unit (ICU). Resistant AH was managed initially with labetalol and sodium nitroprusside by continuous intravenous infusion, followed with seven antihypertensive medications taken orally: metoprolol 100 mg/day, indapamide 1.5 mg/day, moxonidine 0.6 mg/day, olmesartane/amlodipine 40 mg/10 mg/day, spironolactone 25 mg/day, and doxazosine

16 mg/day. Low-molecular-weight-heparin (nadroparine) was also prescribed up to 1.2 mL/day subcutaneously.

On day 3, in the ICU, the second cranial CT scan showed a large area of ischemic cerebral infarction in the right middle cerebral artery (MCA) supply territory, and a small zone of secondary hemorrhagic transformation (Fig. 1, panel B).

On day 11 in the ICU, the second neurosonological evaluation by the same neurosonographer (KL) revealed that the wall of the right CCA still remained uneven (Fig. 5). However, the previously observed hyperechogenicity, which was proved to be the thrombosed lumen of the CCA wall was reducing. Spectral Doppler analysis showed the pattern of reduced linear blood flow velocities in the right ICA to 51/18 cm/s (the left ICA 83/31 cm/s), as well a markedly reduced carotid index to 0.40, and Pourcelot index in the right ICA to 0.647. However, the linear flow velocities in both CCA were increased, compared to the initial evaluation (right from 72/15 cm/s to 126/18 cm/s and left from 97/16 cm/s to 134/18 cm/s, and RI to 0.86), indicating that not only morphological, but also hemodynamic changes were taking place. Also, a clearly visible reverse component became apparent in the waveforms. After carotid ultrasound, a CT scan of the aorta was repeated, this confirmed the positive changes, and aortic dissection remained the same. TTE showed second grade aortic valve insufficiency, not third grade as previously found.

On day 22, after the patient was transferred back to the DN the third duplex carotid evaluation (KL), was performed, showing continued signs of improvement (Fig. 6). The extent of the pathological signal was markedly reduced and there was only a mild thickening in the inner vessel wall of the right CCA. The transcranial color-coded ultrasonographic examination of intracranial vessels did not show any abnormal changes. The conservative management and close follow-up strategy was kept. The patient was examined for primary causes of aortic dissection, but no evidence of infection, vasculitis, genetic or other specific causes were found. Left-sided hemiparesis gradually and totally resolved. The patient scored 2 points on the NIHSS, was independent in activities of daily living, and able to walk.

After 4 months, a CT scan of the thoracic aorta was repeated in the out-patient Department of Cardiology (Fig. 3, panel B), which showed further improvements.

After 5 months, the patient arrived for a follow-up visit in the out-patient DN. No neurological deficits were observed and carotid ultrasound findings were the same as on the last examination.

3. Review of the literature and discussion

3.1. Pathogenesis and hemodynamic properties of aortic dissection with neurological presentation

Aortic dissection occurs as a consequence of an intimal tear that allows access of blood under systolic pressure into the medial layer of the aorta with propagation that is more antegrade than retrograde [16]. Numerous congenital and acquired factors that lead to aortic medial degeneration are considered to be the central underlying process in aortic dissection, which result in an increased arterial wall stress.

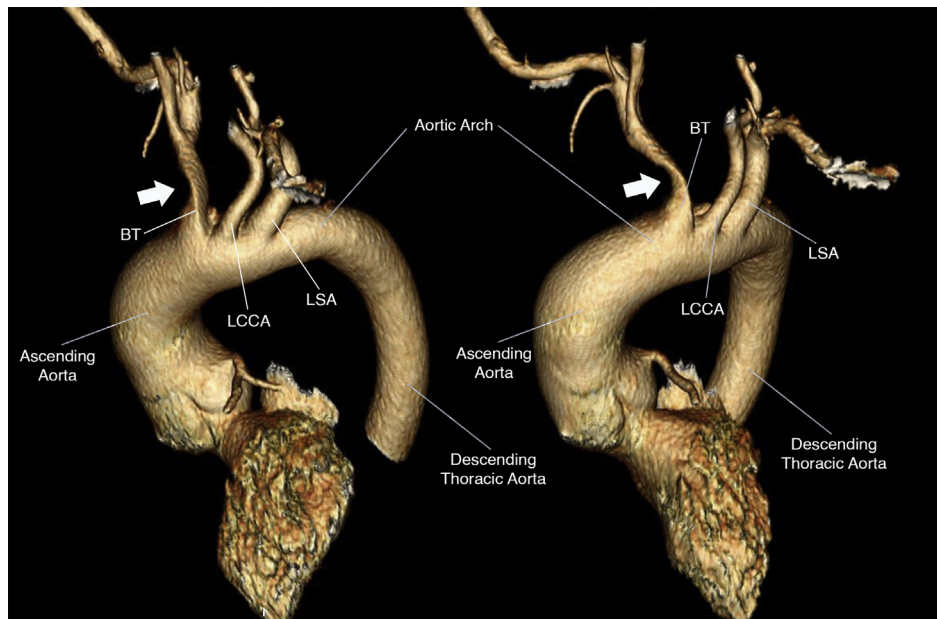


Fig. 4 – Three-dimensional (3D) reconstruction of contrast enhanced CT angiography images of the thoracic aorta on day 2. Arrows indicate narrowed “true” lumen of BT. BT, brachiocephalic trunk; LCCA, left common carotid artery; LSA, left subclavian artery.

This process is indicated by the law of Laplace and it is written as $\sigma = P \times r/h$, where σ is arterial wall stress, P is luminal pressure, r is vessel radius, and h is wall thickness [17]. This law demonstrates the detrimental effect of atherosclerosis, which causes medial necrosis and thinning by limiting the supply of oxygen and nutrients. The resulting flap of tissue divides aorta into a “true” lumen, which represents the original vessel lumen, and a “false” lumen, which is the space created by the medial disruption [18]. The intima flap may occlude or compromise branch vessels and lead to malperfusion [18]. The dissecting hematoma may re-enter the true lumen via one or more distal re-entry tears, which provide a mechanism for false lumen decompression. Alternatively, there may be no functioning re-entry site, causing the false lumen to act as a “wind sock” with increasing false lumen pressures, propagation of the dissection, aneurysm formation, compression of the true lumen or branch vessels, or rupture [13,19]. The inflammatory response to thrombus in the media is likely to initiate further necrosis and apoptosis of smooth muscle cells and degeneration of elastic tissue, which potentiates the risk of medial rupture [20,21]. Neurological symptoms may occur because of occlusion of carotid, vertebral, spinal arteries, *vasa nervorum* of the peripheral nerves, or because of hypotension and related cerebral perfusion deficit [13]. Signs and symptoms of IS are the most common initial neurological finding [22,23]. Pathogenetic mechanisms of stroke include extension of dissection toward supra-aortic vessels, thromboembolism or severe hypotension [22]. In published case series, strokes were more frequently hemispheric compared to vertebro-basilar circulation, and they were predominantly right-sided. The dominance of right hemisphere stroke despite mostly bilateral carotid dissection perhaps could be explained by different mechanical dynamics in the progression of the dissecting hematoma [23].

3.2. Predisposing and risk factors of aortic dissection

AH is the most prevalent risk factor for acute aortic dissection and is present in up to 68%–75% of cases [21,24]. The predisposing and risk factors of aortic dissection that have to be kept in mind are classified into further groups: (i) inflammatory diseases, (ii) genetic conditions, (iii) trauma and iatrogenic, and (iv) miscellaneous.

Reported inflammatory diseases associated with aortic dissection were: (a) autoimmune: giant cell arteritis, Takayasu arteritis, Behçet arteritis, rheumatoid arthritis or systemic lupus erythematosus; (b) infection: syphilis, tuberculosis [21]. Genetic conditions that are associated with aortic dissection are Marfan's syndrome, Loeys-Dietz's syndrome, Ehlers-Danlos syndrome (especially vascular type), Turner syndrome, familial syndrome, bicuspid aortic valve, and aortic coarctation. Aortic dissection may be also traumatic, such as deceleration injury or iatrogenic. Cocaine, amphetamine stimulant use and Valsalva maneuver may also be associated with this AAS [17,20,25]. Other important factors include age (sixth and seventh decade), male gender predominance (ratio 1.5:1), history of smoking (prevalence about 60%), dyslipidemia, atherosclerosis (prevalence about 30%) [19]. All the mentioned conditions and risk factors alone or in combination affect the wall of the aorta and may precipitate its disruption of various extent and lead to a number of complications.

3.3. Classification of aortic dissection and its relation to neurological deficits

Aortic dissection is classically classified according to anatomical location of the lesion. Widely accepted classification systems are the following: (i) DeBakey, (ii) Stanford, (iii) Svensson, and (iv) Penn.

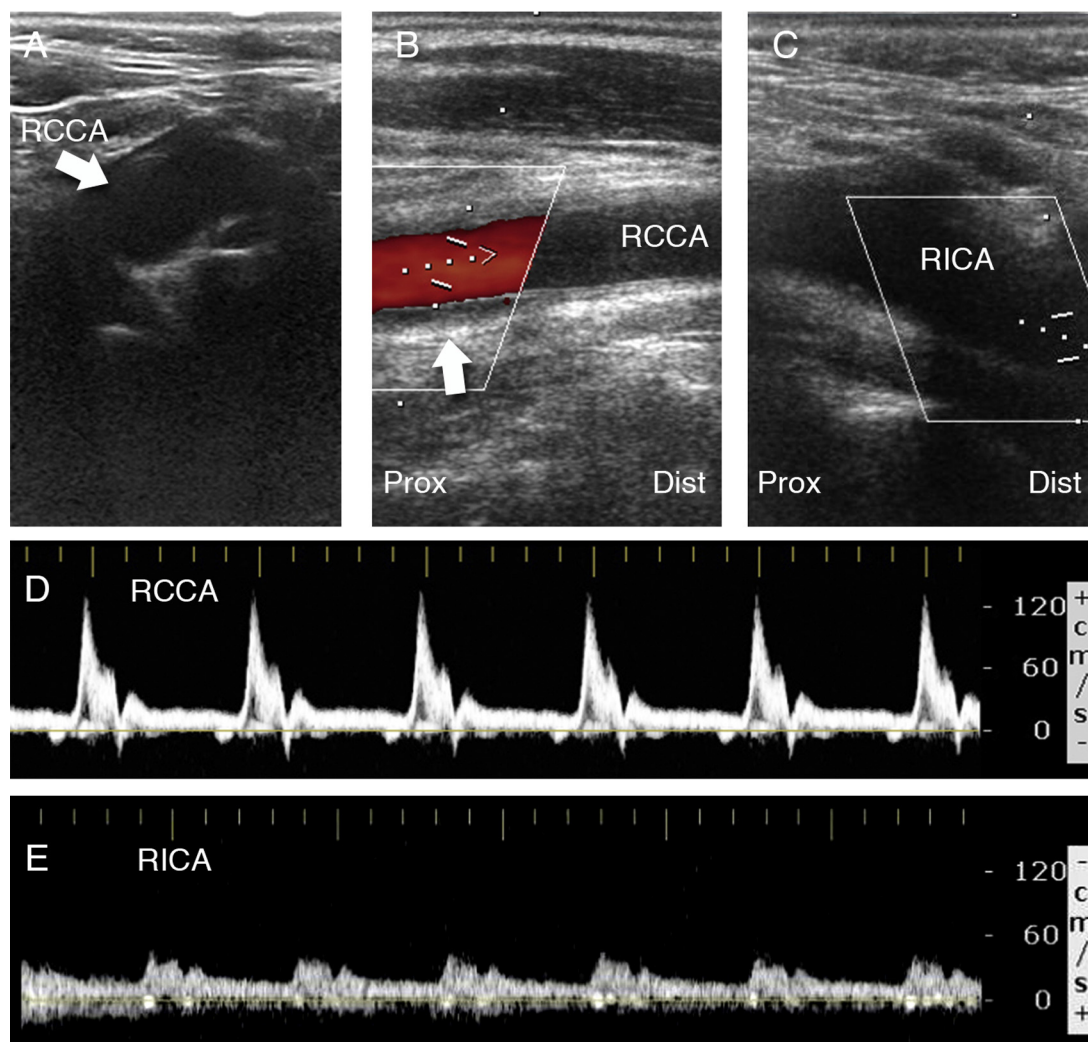


Fig. 5 – Follow-up duplex ultrasound images of extracranial vessels on day 12. Please note reduced previously observed hyperechogenicity of the signal along the vessel wall of right CCA (panels A and B). Characteristic Doppler blood flow profile remained evident, however, linear flow velocities raised in the RCCA (panel D), but significantly decreased in the RICA (panels C and E). RCCA, right common carotid artery; RICA, right internal carotid artery; Prox, proximally (from the brachiocephalic trunk); Dist, distally.

DeBakey type I dissection affects entire aorta. Type II dissection affects the ascending aorta, and type III (a or b) confines to the descending part of the vessel [21,26]. The Stanford classification is based on involvement of ascending aorta. In type A, an ascending aorta is affected and in type B, it is spared. A variety of aortic pathology is presented in Svensson classification, where class 1 is a classic dissection with true and false lumen, class 2 is an intramural hematoma or hemorrhage, class 3 is a subtle dissection without hematoma, class 4 is an atherosclerotic penetrating ulcer, and class 5 is an iatrogenic or traumatic dissection [27,28]. Penn system is a recently proposed system to estimate complications in type B aortic dissection [29].

According to time characteristics, aortic dissection is classified to acute (symptoms present within two weeks) and chronic (more than two weeks) [21,30], or according to the International Registry of Acute Aortic Dissection (IRAD)

classification system, to (i) hyperacute (<24 h), (ii) acute (2–7 days), (iii) sub-acute (8–30 days), and (iv) chronic (>30 days) [31].

Finally, the DISSECT system, that combines together six elements, integrates all the mentioned above topographic, temporal, and clinical aspects in order to help categorize aortic dissection and choose the best management strategy [32].

In patients presenting with neurological deficits due to acute aortic dissection the most frequent type is A. Syncope may complicate the presentation of acute type A dissection in five to ten percent of patients, and acute stroke occurs in about 16% of patients [23], however, in type B aortic dissection acute stroke is not an exception either [8]. Late IS has been reported in 1.4%–5% of patients with a type B dissection [8]. Spinal cord ischemia, due to interruption of the intercostal vessels, is more common in type B dissections and is seen in two to three percent of patients [33].

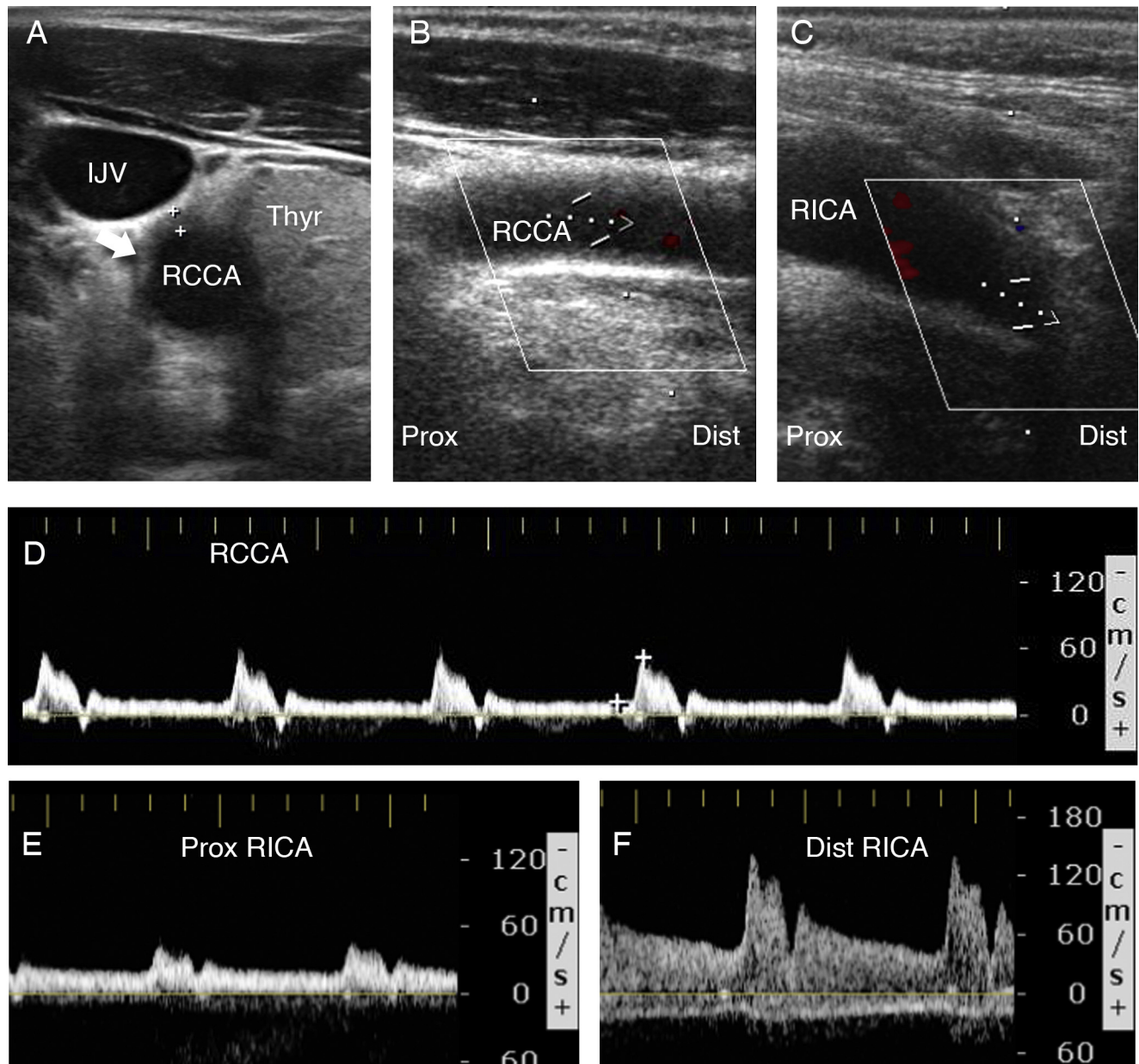


Fig. 6 – Follow-up duplex ultrasound images of extracranial vessels on day 22. Please note the extent of pathological signal was markedly reduced and only a mild thickening in the inner RCCA wall remained (panels A and B). Linear blood flow velocities reduced in the RCCA (panel D), remained low in the RICA (panels C and E), however, in the distal portion of the RICA increased (panel F). IJV, internal jugular vein; RCCA, right common carotid artery; RICA, right internal carotid artery; Prox, proximally (from the brachiocephalic trunk); Dist, distally; Thyr, thyroid gland.

3.4. Neurological symptoms in acute aortic dissection

Neurologic complications occur in 13%–42% of patients with acute aortic dissection, and the patients with neurologic complications have higher mortality compared to those without [23,34]. The main non-neurological complications of acute aortic dissection include: aortic regurgitation, cardiac tamponade, myocardial ischemia or infarction, heart failure, pleural effusion, mesenteric ischemia, acute renal failure or/and lower limb ischemia [4,20].

The main neurological complications of aortic dissection are as follows: (i) major neurological deficit (coma or IS), (ii)

transient ischemic attack (TIA), (iii) spinal cord ischemia (paraparesis, paraplegia), (iv) ischemic neuropathy, and (v) hypoxic-ischemic encephalopathy [13,20]. Among them, IS is reported as the most frequent complication, which affects up to one-third of patients with aortic dissection [35]. Other neurological consequences include syncope (6%–19%), generalized tonic-clonic seizures, somnolence, transient global amnesia, altered mental status, Horner's syndrome, vocal cord paralysis, hoarseness or dysphagia due to organ displacement [4,35,36]. In half of the patients, neurological symptoms are transient. Rapid improvement in such cases is probably the result of transient arterial occlusion at the

moment of propagation of the dissection. Gaul et al. have reported that the majority of patients (87%) with aortic dissection complained of initial chest or back pain; however, among the patients with neurological symptoms at the onset of dissection, only two-thirds gave a history of pain [23].

The mechanism of painless aortic dissection is not very clear so far and may be related to the following factors: aortic blood flows slowly into the film without an impact of external forces or small impact force on the occurrence of aortic dissection, and the extravascular membrane force generated is so small that patients do not feel the pain; painless patients have lower systolic BP which reduces the aortic pressure and the tension of vascular adventitia, resulting in failure of pain generation [37].

3.5. Imaging in (hyper)acute aortic dissection

Taking into account that in 25%–50% of patients with an aortic dissection the wrong diagnosis is made [38], the most important task for every consulting specialist in the ED is as follows: (a) as fast as possible to suspect an aortic dissection as underlying cause of acute stroke and other complications, and (b) to rule out or to confirm suspected diagnosis by choosing an appropriate diagnostic tool.

Aortic emergencies have to be detected quickly [39]. Ancillary diagnostic armamentarium may consist of: (i) ultrasonography (extracranial and intracranial vessel ultrasound, TEE), (ii) CT with contrast-enhanced angiography (CTA), (iii) MRI with contrast-enhanced angiography (MRA), and (iv) conventional angiography (aortography).

If acute aortic dissection is suspected, an immediate CTA needs to be performed [4]. Multi-slice CTA is the fastest and most robust imaging modality [39]. Imaging of the whole aorta (48 cm) may take 20 s (16-slice CT with small collimation), 10 s (16-slice CT with large collimation), 6 s (for 64-slice CT) or well below 3 s (for 256–320-slice or dual CT) [39]. However, the investigations that carry the highest sensitivity and specificity, CT, TEE, and MRI are not usually the first-line investigations in most patients so a high index of clinical suspicion of alarming signs remains a key [5,36,40]. According to the IRAD investigators, the overall sensitivity (including true-positive rates of the first imaging modality to detect aortic dissection) of TEE, CT, MRI and aortography was 88%, 93%, 100%, and 87%, respectively [5]. If required, the imaging has to be repeated [3]. A mean radiation dose of single conventional dual-source contrast-enhanced CT aortography is 3.9 ± 0.9 mSv [41].

The main clinical clues enhancing suspicion of aortic dissection (“red flags”) in the case of acute IS are as follows: (i) history of aortalgic pain or chest discomfort, (ii) left hemiparesis, (iii) transient or fluctuating neurological symptoms, (iv) transient hypotension or shock, (v) bradycardia, (vi) asymmetrical pulses, (vii) cardiac murmur, and (viii) carotid bruits. In our presented case, the patient had a history of pain; however, it was not a typical aortalgic pain. Left hemiparesis, fluctuating neurological symptoms, transient hypotension and bradycardia were also characteristic clinical findings. On physical examination at presentation some patients with an aortic dissection are described to be anxious and in distress [42].

There is no consensus, whether aortic dissection is a contraindication for IVT in acute IS. However, clinical data

shows that IVT might be associated with high risks. IVT for acute IS should be avoided in ascending aortic dissection, but is safe in carotid dissection and appears to be safe in intracranial artery dissection with no evidence of subarachnoid hemorrhage on baseline imaging [10]. This opinion is also supported by the fact that aortic dissection is an absolute contraindication to IVT in case of acute myocardial infarction [43]. An inappropriate IVT possibly has an adverse effect on the outcomes by causing further expansion of the tear, and by idiopathic prolonged coagulation. It also carries a high risk of aortic rupture leading to hemopericardium, cardiac tamponade and intrapleural hemorrhage (hemothorax) [35,44–47].

Due to rare incidence of aortic dissection, clinical evidence on a rapid and specific screening method is lacking. Laboratory testing that can increase alert for the differential diagnosis is evaluation of D-dimer levels. Typically, the level of D-dimers is immediately very high in aortic dissection, compared with other disorders in which the D-dimer level increases gradually. D-Dimers yielded the highest diagnostic value during the first hour [20]. Extracranial vessel duplex ultrasound may serve as a valuable tool to diagnose patients with dissections of brain supplying arteries in anterior and posterior circulation. There is evidence that involvement of supra-aortic branches in aortic dissection is not a rarity [23,48]. Among published single case reports and case series, where carotid ultrasound was the first method leading to the correct diagnosis, Vassileva et al. have described five cases of stroke and TIA due to aortic dissection [49], and Baguet et al. reported four cases, where the first method of detecting underlying aortic pathology was extracranial ultrasound [24].

Routine carotid ultrasound can identify stroke patients ineligible for IVT therapy due to aortic dissection [7,9,15]. It is very important to keep in mind that extracranial vessel ultrasonography should not delay IVT. That is why it is essential to perform a short scan just in B-mode and then color-coded imaging within minutes [15]. However, Noel and colleagues described a successful IVT in a coexisting aortic dissection that was suspected after a carotid ultrasound examination with characteristic changes [50].

In the present case an abnormal signal in the right CCA was observed. However, other frequent ultrasonographic findings which may be detected include: (a) occlusion of one or both CCA, (b) double lumen in one or both CCA, extending to the carotid bifurcation with hemodynamically relevant stenosis of the CCA or ICA, as well as (c) a subclavian steal syndrome. These specific and characteristic findings may be unilateral or bilateral. Different flow velocities on pulsed Doppler wave analysis can be detected within the “true” and the “false” lumen with a “to-and-fro” spectral profile in the latter. Also abnormal carotid ultrasound findings can be combined with typical simultaneous flow changes in vertebral or subclavian arteries [13,15,49,51]. However, aortic dissections might located be more distally, which often limit their visualization by duplex sonography [52,53]. Finally, although positive findings can be detected by ultrasonography, aortic CTA is still required to differentiate between aortic dissection extending into the cervical arteries and primary cervical artery dissection, where IVT should not be withheld [54].

Carotid ultrasound can be useful not only for the rapid diagnosis of underlying dissection, but also for a follow-up, as

it was in the presented case. The hemodynamic variables such as flow pattern assessed with carotid ultrasound can have prognostic value. The conservative management strategy of aortic dissection was chosen for the patient because the pathological changes were resolving and clinical condition was improving, which postponed surgical procedure. Extracranial vessel ultrasound together with other imaging methods helped to evaluate the process of vessel wall repairment and blood flow restoration, which indicated that the risk of repetitive ischemic event was low. Repeated carotid ultrasound evaluation showed a gradually reducing hyperecho-genic signal area in the wall of the right CCA and changing blood profile dynamics. However, it is worth mentioning that results of repeated neurosonologic examinations may vary from one neurosonology expert to another, so it would be more valuable, if a single examiner would examine one patient over different periods of time.

Renal impairment, pulse deficit, neurologic complication and nonsurgical treatment were described as independent variables for determining in-hospital mortality in patients with type A dissection [22]. Refractory AH or pain, and an aortic diameter of 5.5 cm or greater were also listed according to the IRAD investigators [5,21,31,55]. Retrospective analysis of our patient uncovered four of the mentioned above variables, which could have been associated with unfavorable prognosis: neurologic complication, slight renal impairment, refractory AH and nonsurgical treatment. However, initial and further medical-only treatments were prescribed strictly in compliance with international experience and recommendations, which probably ensured the successful outcome. Initial management of acute dissection aims to limit propagation by control of BP and reductions in the change in pressure over time, ideally in the ICU [21]. Reduction of pulse pressure to maintain sufficient end-organ perfusion is a priority; intravenous β -blockade (labetalol with both α -blockade and β -blockade properties) is the first-line treatment [21]. According to the IRAD experience, drugs should be adjusted to achieve a systolic pressure of 100–120 mm Hg and a HR of 60–80 beats per minute, which often needs a poly-pharmacy approach [17,21]. Further management (open surgery or endovascular intervention) depends on the site of the tear, evidence of complications and disease progression on serial imaging. There is still controversy over whether surgery should be performed in patients with type A aortic dissection presenting with neurological deficits or coma. Although commonly associated with a poor post-operative prognosis, recovery has been reported when rapid brain reperfusion is achieved, especially, if the time between symptom onset and arrival at the operating room is less than 5 h [20,21]. The IRAD investigators in a recently published review of the registry concluded, that surgical management for type A aortic dissection increased from 79% to 90%, endovascular management of type B increased from 7% to 31%, and most importantly, type A in-hospital mortality decreased significantly (31%–22%), as well as surgical mortality (25%–18%). As it is stated in the European Society of Cardiology (ESC) Guidelines on the diagnosis and treatment of aortic diseases, in case of acute type A aortic dissection, surgery is the treatment of choice. Acute type A aortic dissection has a mortality of 50% within the first 48 h if not operated. Based on that evidence, all patients with type A

aortic dissection should be sent for surgery. The superiority of surgery over conservative treatment has been reported, even in patients with unfavorable presentations and/or major comorbidities. However, the course of type B aortic dissection is often uncomplicated, in the absence of malperfusion or signs of (early) disease progression, the patient can be safely stabilized under medical therapy alone, to control pain and blood pressure [20].

4. Conclusions

Aortic dissection is a great challenge for many medical specialists, including neurologists. There is data confirming the opinion that IVT should be withheld in the case of stroke with a primary cause of aortic dissection due to a high risk of complications. Ultrasound examination of extracranial vessels is a suitable screening method in a narrow time window of thrombolysis, where it may be a key method for revealing the underlying cause of stroke. In our presented case, extracranial ultrasound had a crucial role in promptly determining the correct diagnosis. Secondly, conservative treatment strategy was chosen, based on the follow-up imaging results. The patient was not eligible for IVT, no urgent surgery was performed in this hyperacute ascending aortic dissection (Stanford type A) setting either, and the surgical procedure was postponed. However, the outcomes were successful after medical-only management, which was prescribed strictly in compliance with international recommendations. Early ultrasound examination of extracranial vessels should be considered in the case of atypical presentation of IS. Importantly, “red flags” that require further investigation in addition to the conventional thrombolysis protocol should be identified. As in our case presented, symptoms, suggesting of underlying acute aortic dissection resulting in IS are listed as follows: presence of aortalgic pain before the onset of symptoms, young age, fluctuation of symptoms, transient hypotension or bradycardia. Patients with the highest risk should be referred to specialized centers with stroke team available 24/7 (including neurologist) and equipped with all the necessary diagnostic tools. To conclude with, duplex ultrasound examination can be a simple and rapid tool to rule out potential aortic dissection and to avoid a fatal sequel of thrombolytic therapy.

Conflict of interest

The authors declare no conflict of interest.

Informed consent

Informed consent has been obtained from the patient to reproduce the images and other clinical data anonymously.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

REFERENCES

- [1] Howard DP, Banerjee A, Fairhead JF, Perkins J, Silver LE, Rothwell PM, Oxford Vascular Study. Population-based study of incidence and outcome of acute aortic dissection and premorbid risk factor control: 10-year results from the Oxford Vascular Study. *Circulation* 2013;127:2031–7.
- [2] Olsson C, Thelin S, Ståhle E, Ekbom A, Granath F. Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. *Circulation* 2006;114:2611–8.
- [3] Meier C, Lichtenberg M, Lebedez P, Breuckmann F. Pitfalls in suspected acute aortic syndrome: impact of appropriate and if required repeated imaging. *Case Rep Emerg Med* 2015;2015:573256.
- [4] van Bree SH, van Bree MD, Somsen GA. Horner's syndrome in a patient presenting with chest pain. *Neth J Med* 2015;73:293–5.
- [5] Moore AG, Eagle KA, Bruckman D, Moon BS, Malouf JF, Fattori R, et al. Choice of computed tomography, transesophageal echocardiography, magnetic resonance imaging, and aortography in acute aortic dissection: International Registry of Acute Aortic Dissection (IRAD). *Am J Cardiol* 2002;89:1235–8.
- [6] Raymond CE, Aggarwal B, Schoenhagen P, Kralovic DM, Kormos K, Holloway D, et al. Prevalence and factors associated with false positive suspicion of acute aortic syndrome: experience in a patient population transferred to a specialized aortic treatment center. *Cardiovasc Diagn Ther* 2013;3:196–204.
- [7] Fernandes A, Caetano F, Cachulo MC, Gonçalves L. A straightforward stroke? Maybe not: atypical presentation of type A aortic dissection. *BMJ Case Rep* 2015;2015.
- [8] AlGhamdi A, Alqahtani S, Ricketti M, Aziz S. Early acute ischaemic stroke in two patients with acute type B aortic dissection: an unusual complication. *BMJ Case Rep* 2015;2015.
- [9] Seliger C, Turmanidze N, Schmid E, Wiethölter H, Boy S, Bogdahn U, et al. Three cases of stroke in patients with atypical presentation of type a aortic dissection – potential of neurosonography in the early diagnosis of atypical stroke. *Ultraschall Med* 2011;32:619–21.
- [10] Tsvigoulis G, Safouris A, Alexandrov AV. Safety of intravenous thrombolysis for acute ischemic stroke in specific conditions. *Expert Opin Drug Saf* 2015;14:845–64.
- [11] Powers WJ, Derdeyn CP, Biller J, Coffey CS, Hoh BL, Jauch EC, et al., American Heart Association Stroke Council. 2015 AHA/ASA focused update of the 2013 guidelines for the early management of patients with acute ischemic stroke regarding endovascular treatment: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2015;46(10):3020–35.
- [12] European Stroke Organisation (ESO) Executive Committee, ESO Writing Committee. Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. *Cerebrovasc Dis* 2008;25:457–507.
- [13] Gaul C, Dietrich W, Erbguth FJ. Neurological symptoms in aortic dissection: a challenge for neurologists. *Cerebrovasc Dis* 2008;26:1–8.
- [14] Shunsuke T, Yanagawa Y. Case report: ischemic stroke with left hemiparesis or shock should be evaluated by computed tomography for aortic dissection. *Am J Emerg Med* 2012;30:836.e3–4.
- [15] Hama Y, Koga M, Tokunaga K, Takizawa H, Miyashita K, Iba Y, et al. Carotid ultrasonography can identify stroke patients ineligible for intravenous thrombolysis therapy due to acute aortic dissection. *J Neuroimaging* 2015;25:671–3.
- [16] Tsai TT, Nienaber CA, Eagle KA. Acute aortic syndromes. *Circulation* 2005;112:3802–13.
- [17] Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, et al., Task Force on Aortic Dissection, European Society of Cardiology. Diagnosis and management of aortic dissection. *Eur Heart J* 2001;22:1642–81.
- [18] Bergmark BA, Sobieszczek P, Gravereaux EC, Bonaca M, Giugliano RP. Acute dissection of the descending aorta: a case report and review of the literature. *Cardiol Ther* 2013;2:199–213.
- [19] Bonaca MP, O'Gara PT. Diagnosis and management of acute aortic syndromes: dissection, intramural hematoma, and penetrating aortic ulcer. *Curr Cardiol Rep* 2014;16:536.
- [20] Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 2014 ESC guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The task force for the diagnosis and treatment of aortic diseases of the European Society of Cardiology (ESC). *Eur Heart J* 2014;35:2873–926.
- [21] Nienaber CA, Clough RE. Management of acute aortic dissection. *Lancet* 2015;385:800–11.
- [22] Lee SJ, Kim HJ, Na CY, Oh SS, Kim YM, Lee CK, et al. Eleven years of experience with the neurologic complications in Korean patients with acute aortic dissection: a retrospective study. *BMC Neurol* 2013;13:46.
- [23] Gaul C, Dietrich W, Friedrich I, Sirch J, Erbguth FJ. Neurological symptoms in type A aortic dissections. *Stroke* 2007;38:292–7.
- [24] Baguet JP, Chavanon O, Sessa C, Thony F, Lantelme P, Barone-Rochette G, et al. European Society of Hypertension scientific newsletter: hypertension and aortic diseases. *J Hypertens* 2012;30:440–3.
- [25] Singh A, Khaja A, Alpert MA. Cocaine and aortic dissection. *Vasc Med* 2010;15:127–33.
- [26] DeBaakey ME, Beall Jr AC, Cooley DA, Crawford ES, Morris Jr GC, Garrett HE, et al. Dissecting aneurysms of the aorta. *Surg Clin N Am* 1966;46:1045–55.
- [27] Gollidge J, Eagle KA. Acute aortic dissection. *Lancet* 2008;372:55–66.
- [28] Bossone E, Corteveille DC, Harris KM, Suzuki T, Fattori R, Hutchison S, et al. Stroke and outcomes in patients with acute type A aortic dissection. *Circulation* 2013;128:S175–9.
- [29] Augoustides JG, Szeto WY, Woo EY, Andritsos M, Fairman RM, Bavaria JE. The complications of uncomplicated acute type-B dissection: the introduction of the Penn classification. *J Cardiothorac Vasc Anesth* 2012;26:1139–44.
- [30] Steuer J, Björck M, Mayer D, Wanhainen A, Pfammatter T, Lachat M. Distinction between acute and chronic type B aortic dissection: is there a sub-acute phase? *Eur J Vasc Endovasc Surg* 2013;45:627–31.
- [31] Booher AM, Isselbacher EM, Nienaber CA, Trimarchi S, Evangelista A, Montgomery DG, et al. The IRAD classification system for characterizing survival after aortic dissection. *Am J Med* 2013;126: 730.e19–24.
- [32] Dake MD, Thompson M, van Sambeek M, Vermassen F, Morales JP, DEFINE Investigators. DISSECT: a new mnemonic-based approach to the categorization of aortic dissection. *Eur J Vasc Endovasc Surg* 2013;46:175–90.
- [33] DeBaakey ME, McCollum CH, Crawford ES, Morris Jr GC, Howell J, Noon GP, et al. Dissection and dissecting aneurysms of the aorta: twenty-year follow-up of five hundred twenty-seven patients treated surgically. *Surgery* 1982;92:1118–34.
- [34] Blanco M, Díez-Tejedor E, Larrea JL, Ramírez U. Neurologic complications of type I aortic dissection. *Acta Neurol Scand* 1999;99:232–5.

- [35] Kamouchi M. Aortic dissection as a possible underlying cause of acute ischemic stroke. *Circ J* 2015;79:1697–8.
- [36] Kinner S, Eggebrecht H, Maderwald S, Barkhausen J, Ladd SC, Quick HH, et al. Dynamic MR angiography in acute aortic dissection. *J Magn Reson Imaging* 2015;42:505–14.
- [37] Liu Z, Zou Y, Chai B, Zeng H. Analysis of clinical features of painless aortic dissection. *J Huazhong Univ Sci Technol* 2014;34:73–5.
- [38] Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA* 2000;283:897–903.
- [39] Voitle E, Hofmann W, Cejna M. Aortic emergencies – diagnosis and treatment: a pictorial review. *Insights Imaging* 2015;6:17–32.
- [40] O'Brien D, White S, Wilson D, Haworth K, Williams A. Thoracic aortopathies in the military patient. *J R Army Med Corps* 2015;161(3):230–6.
- [41] Liu Y, Xu J, Li J, Ren J, Liu H, Xu J, et al. The ascending aortic image quality and the whole aortic radiation dose of high-pitch dual-source CT angiography. *J Cardiothorac Surg* 2013;8:228.
- [42] Lissin LW, Vagelos R. Acute aortic syndrome: a case presentation and review of the literature. *Vasc Med* 2002;7:281–7.
- [43] Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC), Steg PG, James SK, Atar D, Badano LP, Blömostrom-Lundqvist C, et al. ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J* 2012;33:2569–619.
- [44] Minematsu K, Toyoda K, Hirano T, Kimura K, Kondo R, Mori E, et al. Guidelines for the intravenous application of recombinant tissue-type plasminogen activator (alteplase), the second edition, October 2012: a guideline from the Japan Stroke Society. *J Stroke Cerebrovasc Dis* 2013;22: 571–600.
- [45] Flemming KD, Brown Jr RD. Acute cerebral infarction caused by aortic dissection: caution in the thrombolytic era. *Stroke* 1999;30:477–8.
- [46] Tsigkas G, Kasimis G, Theodoropoulos K, Chouchoulis K, Baikoussis NG, Apostolakis E, et al. A successfully thrombolysed acute inferior myocardial infarction due to type A aortic dissection with lethal consequences: the importance of early cardiac echocardiography. *J Cardiothorac Surg* 2011;6:101.
- [47] Hong KSH, Park SY, Whang SI, Seo SY, Lee DH, Kim HJ, et al. Intravenous recombinant tissue plasminogen activator thrombolysis in a patient with acute ischemic stroke secondary to aortic dissection. *J Clin Neurol* 2009;5:49–52.
- [48] Charlton-Ouw KM, Azizzadeh A, Sandhu HK, Sawal A, Leake SS, Miller 3rd CC, et al. Management of common carotid artery dissection due to extension from acute type A (DeBakey I) aortic dissection. *J Vasc Surg* 2013;4:910–6.
- [49] Vassileva E, Klissurski MG, Vavrek EV, Daskalov MP. Cerebrovascular ultrasound as a first method of detecting aortic dissection in acute stroke patients. *Int J Stroke* 2015;10(1):E8.
- [50] Noel M, Short J, Farooq MU. Thrombolytic therapy in a patient with acute ischemic stroke caused by aortic dissection. *Clin Neural Neurosurg* 2010;112(8):695–6. <http://dx.doi.org/10.1016/j.clineuro.2010.03.021> [Epub ahead of print]
- [51] Morelli N, Rota E, Mancuso M, Immovilli P, Spallazzi M, Rocca G, et al. Carotid ultrasound imaging in a patient with acute ischemic stroke and aortic dissection: a lesson for the management of ischemic stroke? *Int J Stroke* 2013;8:E53–4.
- [52] Valdueza JM, Schreiber SJ. *Neurosonology and imaging of stroke*. Stuttgart: Thieme; 2011. p. 80.
- [53] Iguchi Y, Kimura K, Sakai K, Kimura K, Sakai K, Matsumoto N, et al. Hyper-acute stroke patients associated with aortic dissection. *Intern Med* 2010;49:543–7.
- [54] Zinkstok SM, Vergouwen MD, Engelter ST, Vergouwen MD, Engelter ST, Lyrer PA, et al. Safety and functional outcome of thrombolysis in dissection-related ischemic stroke: a meta-analysis of individual patient data. *Stroke* 2011;42:2515–20.
- [55] Pape LA, Awais M, Woznicki EM, Suzuki T, Trimarchi S, Evangelista A, et al. Presentation, diagnosis, and outcomes of acute aortic dissection: 17-year trends from the International Registry of Acute Aortic Dissection. *J Am Coll Cardiol* 2015;66:350–8.