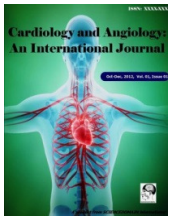

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Extensive Type A Aortic Dissection; from Internal Carotid Artery to Iliac Artery: A Case Report and Review of the Literature

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Authors' contributions

This work was carried out in collaboration between all authors. Author YG wrote the manuscript. Authors AE and KA did carotid ultrasonography and CT angiography. Author LTA managed the literature searches. Author ZO did neurologic examination of the case. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aortic dissection is defined as separation of the layers within the aortic wall. An acute aortic dissection is associated with high morbidity and mortality rates, indeed, many patients die before presentation to the emergency department (ED) or before diagnosis is made in the ED. The location, size and etiology of a dissection all impact on the clinical outcome. All arteries may be affected by the dissection, and clinical signs and symptoms such as neurologic, renal and extremity complications may vary depending on the involvement.

We reported the case of a 59-year-old woman who presented with sudden-onset pain, weakness and numbness of the right leg, with no chest or back pain. A combination of features on transcervical carotid Doppler and CT angiograhy demonstrated type A aortic dissection extending to the common iliac artery and involving the supra-aortic branches, with distal extension to the carotid vessels.

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Keywords: Aortic dissection; complication; carotid artery; iliac artery.

1. INTRODUCTION

Acute aortic dissection is associated with high morbidity and mortality rates, indeed, many patients die before presentation to the emergency department (ED) or before diagnosis is made in the ED [1,2]. Herein, we reported the case of a 59-year-old woman diagnosed by the combination of features on transcervical carotid Doppler and CT angiograhy demonstrated type A aortic dissection extending to the common iliac artery and involving the supra-aortic branches, with distal extension to the carotid vessels.

2. CASE REPORT

A 59-year-old woman was admitted to our emergency department because of sudden onset of pain, weakness and numbness of the right leg, nause and headache. She had been well until 3 hours before admission. She had no chest, or back pain. She had history of hypertension for ten years but no history of smoking or previous cervical or cranial trauma and family history of stroke. On admission, her vital signs were as follows: blood pressure 170/100 mmHg, and pulse rate 82 beats/min and regular, respiration rate 16 breaths/min, body temperature 36.9℃ and oxygen saturation on pulse oximetry 96%. 12-lead electrocardiogram (ECG) demonstrated *negative T waves* on anterolateral leads. Laboratory study did not show any evidence of acute myocardial infarction, but the total creatine phosphokinase was *extremely* high. She had pain, weakness and numbness of the right leg, and she was transfered to neurology department. Neurologic examination confirmed that the patient's right leg were pale, pulseless, numbness and muscle strength was 1/5. Brain diffusion-weighted imaging on the same day revealed chronic cortical and subcortical infarction in the right middle cerebral artery territory. Conventional carotid ultrasonography showed no atherosclerotic changes in the bilateral carotid arteries. However, longitudinal view showed a septum (arrow) separating the true lumen from a false lumen, narrowing of the true lumen (arrowhead) and B-flow imaging visualized different directions of blood flow through true and false lumen indicating artery dissection (Fig. 1, Video 1, 2 see Supplimentary File). Urgent carotid and thoracoabdominal contrast CT angiography revealed an extensive Stanford type A aortic involving the aortic root, ascending, arch, descending segments of the aorta, with involvement of the left subclavian-axillary, carotid arteries and brachiocepahilic arteries, and ascending aneurysm (55 mm), aortic rupture leading to hemopericardium, hemomediastinum compressing pulmonary arteries (Fig. 2). In addition, the dissection flap was seen in the abdominal aorta extends to celiac artery, origins of the mesenteric arteries, and into the left renal artery (Figs. 3 a, b, c), and into the right common iliac artery resulting in nearly complete occlusion (Fig. 3d).

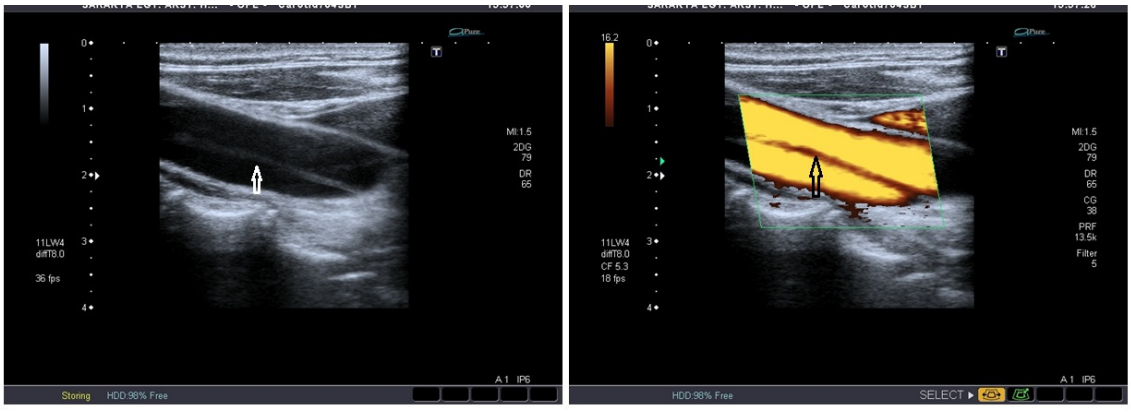

Fig. 1. Longitudinal view showed a septum (arrowhead) separating the true lumen from a false lumen, and B-flow imaging visualized different directions of blood flow through true and false lumen indicating artery dissection

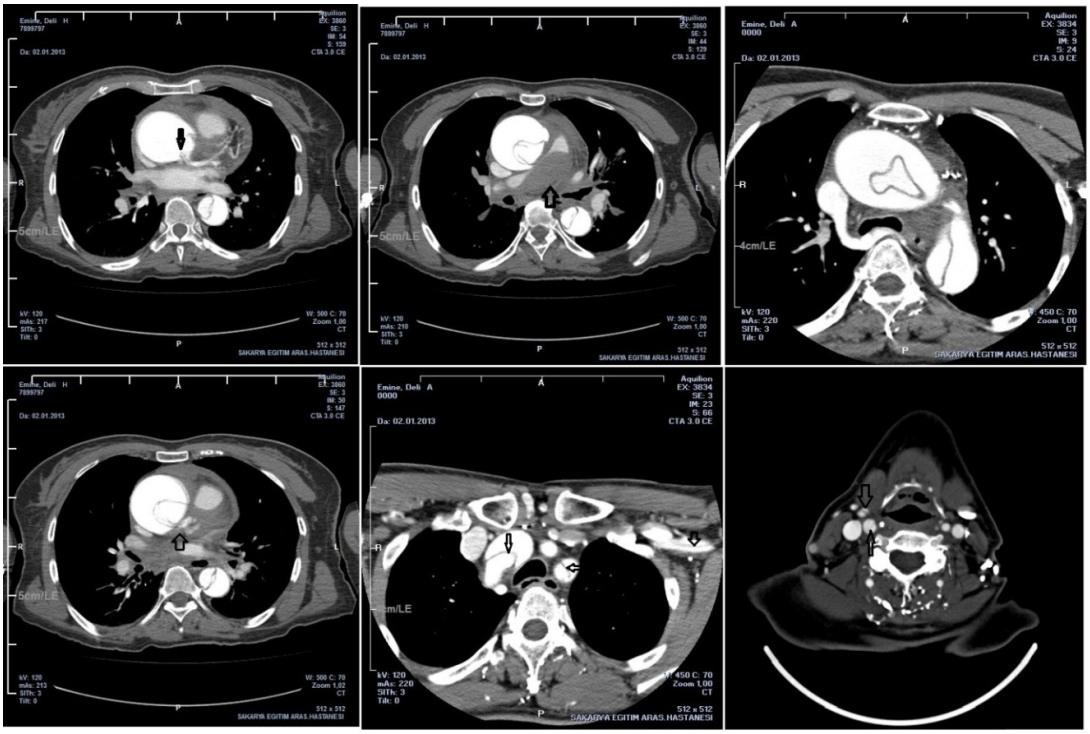

Fig. 2. Urgent carotid and thoracoabdominal contrast CT angiography revealed an extensive Stanford type A aortic involving the aortic root, ascending, arch, descending segments of the aorta, with involvement of the left subclavian-axillary, carotid and brachiocepahilic arteries, and ascending aneurysm, aortic rupture leading to hemopericardium, hemomediastinum compressing pulmonary arteries

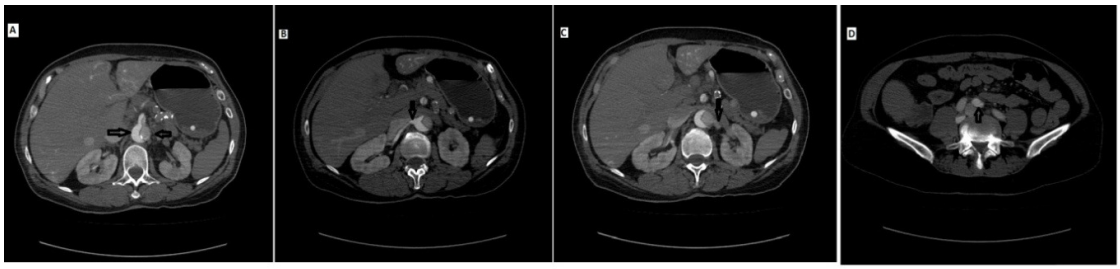

Fig. 3. The dissection flap was seen in the abdominal aorta extends to celiac artery, origins of the mesenteric arteries, and into the left renal artery, and into the right common iliac artery resulting in nearly complete occlusion

Immediately after finishing the acquisition of CT images, the patient developed severe hypotension, pulmonary edema, and a soft diastolic murmur consistent with aortic regurgitation. ECG was repeated showing slow sinus rhythm (35/per minute), and anterolateral ST segment depression. Because of rapid general deterioration and critically ill condition, the patient required orotracheal intubation in the emergency room. The patient underwent emergency surgery of the aorta and coronary bypass grafting. Despite of that, she unfortunately died in a few hours.

3. DISCUSSION

In type A acute aortic dissection, the extent and nature of the involvement of the branches, including the coronary arteries, are important determinants of the clinical syndrome with which the patient presents. These include electrocardiographic changes, or elevated creatine kinase–MB fraction or cardiac troponin

levels (indicating cardiac malperfusion or myocardial dysfunction) [3]. In our patient, CT angigraphy showed partial obstruction of the left main coronary artery by the intimal flap with turbulent flow at its ostium. The ECG showed ST-segment depression in anterolateral leads. But she had no chest pain and had normal cardiac troponin levels.

In aortic dissection extending to the arch, carotid arteries may be involved. Dissections of cervico-cerebral arteries cause transient ischemic attacks or strokes [4]. The most frequently reported site of spontaneous wall dissection is the cervical part of the internal carotid artery (ICA) [5]. The common carotid artery is often poorly visualized at aortic arch angiography, and this might explain why the association of aortic and common carotid artery dissection, although common at autopsy, was never reported intravitam [6].

US is rapidly performed in experienced hands and is suited for emergency use as a noninvasive bedside tool and can help to detect vascular dissections [7,8]. The characteristic ultrasonographic features of a dissection can vary from minor lesions (irregularities and thickening of the vessel wall with a hypoechoic intramural hematoma and/or narrowing of the lumen without hemodynamic alterations) to major structural lesions such as a severe stenosis or occlusion. The true and false lumen can be clearly discriminated because both fast and slow flows are seen simultaneously within the lumen of the vessel. The pulsating membrane is clearly identified (Fig. 1, video 1 see Supplimentary File). B-flow improves the visualization of appositional thrombi, because slow flow, vessel wall and low-echogenic material can be differentiated. Even if the membrane itself cannot be recognized, different color coding in a vessel may indicate different flows in the same lumen, which is an indirect sign of dissection (Fig. 1, video 2 see Supplimentary File). But, one disadvantage of all ultrasound methods is that they cannot cover all areas, such as the thoracic or the intracranial area. For this reason, the distal end of a dissection can often not be identified, and complications here such as appositional thrombi cannot be detected and the estimation of the true extent of the dissection may be limited. [9,10].

While cerebral angiography is considered the gold standard for diagnosis, CT angiography has many advantages [11,12]. CT including helical CT and multisection CT, has the advantages of shorter acquisition time, wide availability, and high diagnostic accuracy and has, therefore, classically been the modality of choice for the evaluation of aortic dissection. Modern multisection CT allows rapid image acquisition and data reconstruction and aids in treatment planning. It helps differentiate type A from type B dissection, may localize the intimal entry site, and helps assess branch-vessel involvement and compromise and the relationship of the branch vessels to the true or false lumen. This information aids in planning treatment with either root replacement, intravascular stent placement, or fenestration [13]. In addition; in patients with thoracic aortic dissection, death can occur secondary to acute aortic regurgitation, major aortic branch obstruction, pericardial tamponade, or aortic rupture, 75% of which occur into the pericardium, left pleural cavity, or mediastinum [14]. In our case, post IV contrast CT aortic angiography revealed extensive dissection involving the aortic root, ascending, arch, descending segments of the aorta, with involvement of the left subclavian-axillary, brachiocepahilic, common carotid arteries, right carotid interna and externa arteries and ascending aneurysm (55 mm), aortic rupture leading to hemopericardium, hemomediastinum compressing pulmonar arteries (Fig. 2).

Main abdominal arterial branch involvement has been reported in 27% of cases [15], and the celiac trunk, superior and inferior mesenteric arteries, and renal arteries can all be involved. Occlusion of the celiac trunk may lead to splenic or hepatic infarction with pain and abnormal liver blood test results. Involvement of the mesenteric branches can lead to mesenteric ischemia with nausea, vomiting, nasogastric aspirates, abdominal pain, bloody diarrhea, recurrent sepsis, and abnormal hepatic and pancreatic enzymes. Compromise of the renal arteries can lead to ischemia with oliguria, anuria, and abnormal renal blood parameters. Renal arteries supplied by the false lumen are rarely compromised [16]. In our case, the dissection flap seen in the abdominal aorta extends to celiac artery, origins of the mesenteric arteries, and an unspecified false lumen flap extends into the left renal artery. There is no contrast enhancement of the left kidney, the right kidney is supplied by the true lumen and demonstrates normal enhancement (Figs. 3 a, b. c).

A dissection that involves the descending aorta can lead to lower extremity ischemia manifests

as a pulse deficit, defined as a weak of femoral pulse resulting from the intimal flap or compression by hematoma [17], or focal neurologic deficits due to spinal artery involvement and spinal cord ischemia [18]. In our case, CT images show extension of a dissection flap into the right common iliac artery (arrow) resulting absence of femoral pulse, pain and weakness of muscular strength (Fig. 3d). This finding emphasizes the need to include the entire aorta and pelvic vessels in the scanning field to detect the full extent of involvement.

4. CONCLUSION

Aortic dissection presents itself through a wide range of manifestations and findings, and every finding represents an important possibility for diagnosis [2]. The overall outcome is determined by the type and extent of dissection and the presence of associated complications; therefore, evaluation of the entire aorta, branch vessels, and iliac and proximal femoral arteries is recommended to aid in treatment planning [14]. The second, and perhaps more important message is that every patient in whom the diagnosis of AD is suspected or confirmed requires prompt, close and constant monitoring since ongoing complications can appear in the following first hours, or even minutes, after initial presentation as demonstrated in our case.

CONSENT

Al authors declare that written informed consent was obtained from the patient for publication of this case report and accompanying images.

ETHICAL APPROVAL

Not aplicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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