

# Ischemic stroke as a comorbidity in a patient with Stanford A aortic dissection: A case report

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## ABSTRACT

**Background.** Aortic dissection is one of the leading causes of death in cardiovascular disease. The clinical course of aortic dissection is often atypical and can mimic a variety of clinical manifestations. Stroke is one of the conditions that can make the management of aortic dissection becoming more complex.

**Case presentation.** A 55-year-old female patient came with complaints of severe chest pain. The patient was diagnosed with Stanford aortic dissection, De Bakey type II, hypertension heart disease, mild mitral regurgitation, mild circumferential pericardial effusion. After the second day of treatment, the patient also complained of sudden slurred speech and weakness on right extremities. The administration of antiplatelet were postponed in consideration of the current patient with aortic dissection. On the 7th day of treatment, the patient experienced a worsening motor strength on the right extremities. We decided to continue antihypertensive therapy on acute phase of ischemic stroke with the consideration that aortic dissection can cause higher mortality.

**Conclusions.** The management of ischemic stroke in a patient with aortic dissection is challenging. In ischemic stroke patient with aortic dissection, antihypertensives need to be given to reduce the stress on the aortic wall and prevent expansion of the dissection which has a higher mortality rate.

**Keywords:** aortic dissection, ischemic stroke, acute

## BACKGROUND

Aortic dissection is one of the leading causes of death in cardiovascular disease. Aortic dissection is reported to occur in 1/10,000 patients who come to the hospital with a mortality rate of up to 50% if left untreated [1,2]. This condition is usually caused by an etiology that causes disturbances in the stabilization of the aortic blood vessel lining such as direct trauma, hypertension, genetic disorder or post-surgery. A tear can cause a variety of clinical conditions such as cardiac tamponade, shock, and organ malperfusion [3,4].

The clinical course of aortic dissection is often atypical and can mimic a variety of clinical manifestations, which poses challenges in the management of patients with aortic dissection [1]. Stroke is one of the conditions that can arise either as a result

of aortic dissection, or as an early manifestation that can make the management of stroke cases with aortic dissection complex [2].

We report a case of a patient with Stanford A aortic dissection with comorbid non-hemorrhagic stroke with considerable potential problems in both diagnosis and case management.

## CASE PRESENTATION

A 55-year-old female patient came with complaints of severe chest pain that radiated to the back since 2 days before being admitted to the hospital. This complaint is first felt by the patient and occurs suddenly when the patient is resting. The pain was continuous, feels sharp like a tear and did not improve with a change in position, there were no com-

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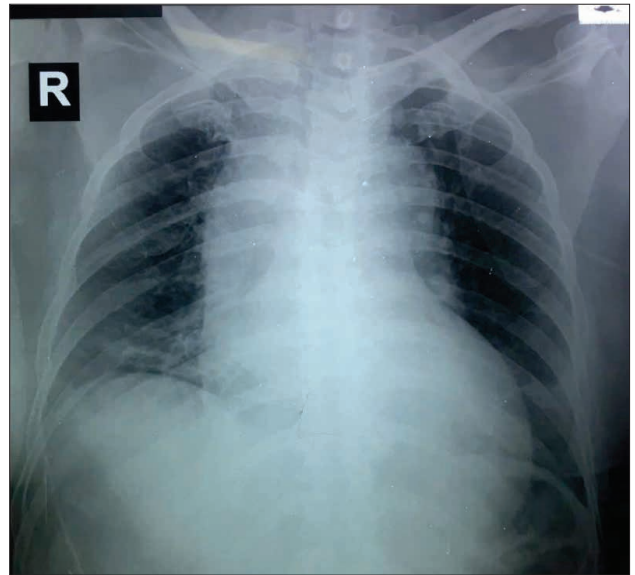
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plain of palpitations, nausea, vomiting, cold sweat or severe shortness of breath. After the second day of treatment, the patient also complained of sudden slurred speech. This complaint is also accompanied by weakness of the right side of the body.

On physical examination the patient was well conscious with GCS E4V5M6, blood pressure at arrival 149/92 mmHg, pulse 92 beats per minute regular, respiration 20 times per minute, oxygen saturation 98% in room air, axillary temperature 36.5°C, and VAS 2/10. On cardiac examination: inspection: no visible ictus cordis; palpation: ictus cordis at 5<sup>th</sup> ICS, at the left mid clavicular line; auscultation: S1 normal, S2 normal, regular, no murmur heard. On neurological status, there was paresis of right cranial nerves VII and XII, flaccid paralysis on the right extremities grade 4, pathological reflexes were negative.

ECG examination was sinus rhythm HR 91 x/min, with T-inversion on V5-V6, I, avL (Figure 1). From the complete blood count with mild normochromic normo-chromic anemia. Thorax roentgen showed a cardiomegaly and aortic dilatation (Figure 2). CT scan of the head showing a hypodense lesions with ill-defined boundaries on the internal and external capsules which suggest a subacute cerebral infarction (Figure 3). Blood gas analysis shows metabolic acidosis. Bedside echocardiography examination revealed normal cardiac chamber dimensions, left ventricle (LV) concentric hypertrophy, with normal LV systolic function (EF BP 78%), with impression of normal valves, intimal flap impressions were obtained at the aortic arch and descending aorta, intimal flap on the ascending aorta not clearly visible with mild dilatation of the aortic root and ascending aorta, mild circumferential pericardial effusion was seen with the largest diameter 0.9 cm in the posterior LV thorax (Figure 4). CT scan with contrast suggesting an aortic dissection (ascending aorta to aortic arch) according to the Stanford A classification with a thickness of +/- 1 cm (Figure 5).

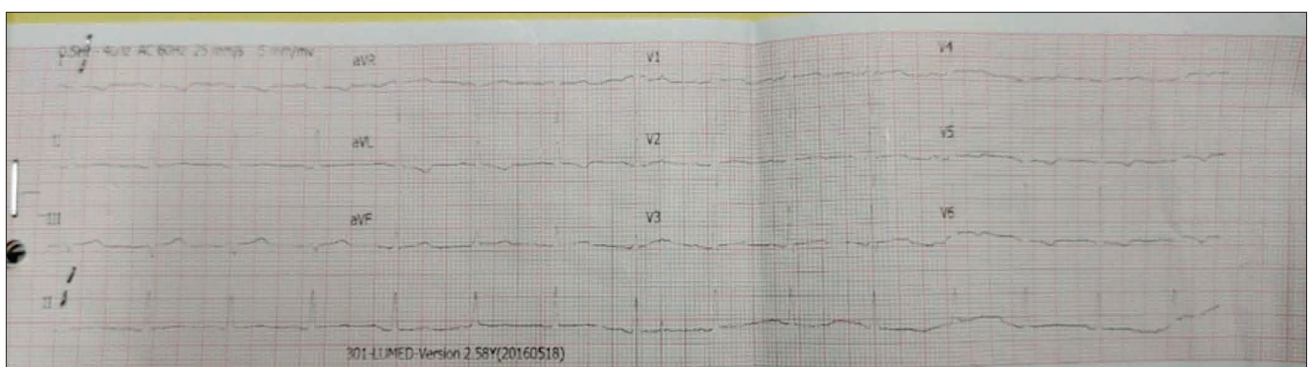
The patient was diagnosed with Stanford aortic dissection, De Bakey type II, hypertension heart dis-



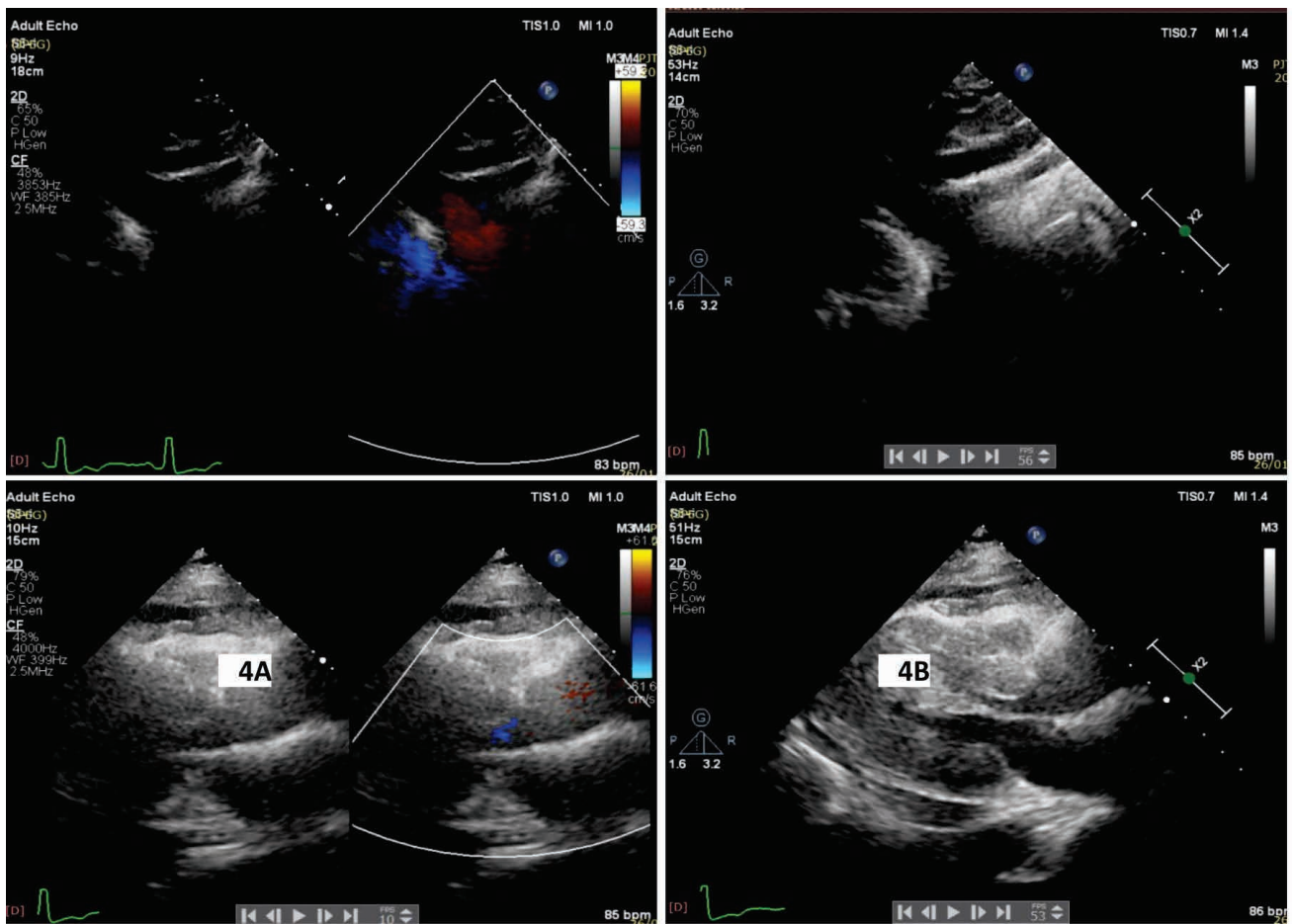
**FIGURE 2.** Thorax Rx showed a cardiomegaly and aortic dilatation



**FIGURE 3.** CT scan of the head showed a hypodense lesions with ill-defined boundaries on the left internal and external capsules which suggest a subacute cerebral infarction



**FIGURE 1.** ECG showed sinus rhythm rates 91 x/min, with T-inversion on V5-V6, I, avL



**FIGURE 4.** Transthoracic echocardiography: **4A** and **4B.** Impressing an intimal flap on aortic arch and Aorta Descendent at suprasternal view; **4C.** Intimal flap on the ascending aorta not clearly visible with mild dilatation of the aortic root and ascending aorta; **4D.** Mild circumferential pericardial effusion was seen with the largest diameter 0.9 cm in the posterior LV thorax

ease, mild mitral regurgitation, mild circumferential pericardial effusion. The patient is planned for hemiarch replacement, if necessary, Total arch replacement would be performed during surgical evaluation. Surgical management has been planned which runs simultaneously with the administration of antihypertensives and other therapies to optimize the patient's condition according to the guidelines for the management of aortic dissection, such as nitro-glycerine drip titrated according to hemodynamic and Ramipril 10 mg, Amlodipine 10 mg, bisoprolol 10 mg, simvastatin 40 mg.

From the neurology department, patient was diagnosed non-hemorrhagic stroke caused by thrombus, and antiplatelets were not given with consideration of ongoing and suspected acute aortic dissection and controlled blood pressure according to aortic dissection management guidelines while evaluating neurological status patient were also given Citicoline as a neuroprotectant.

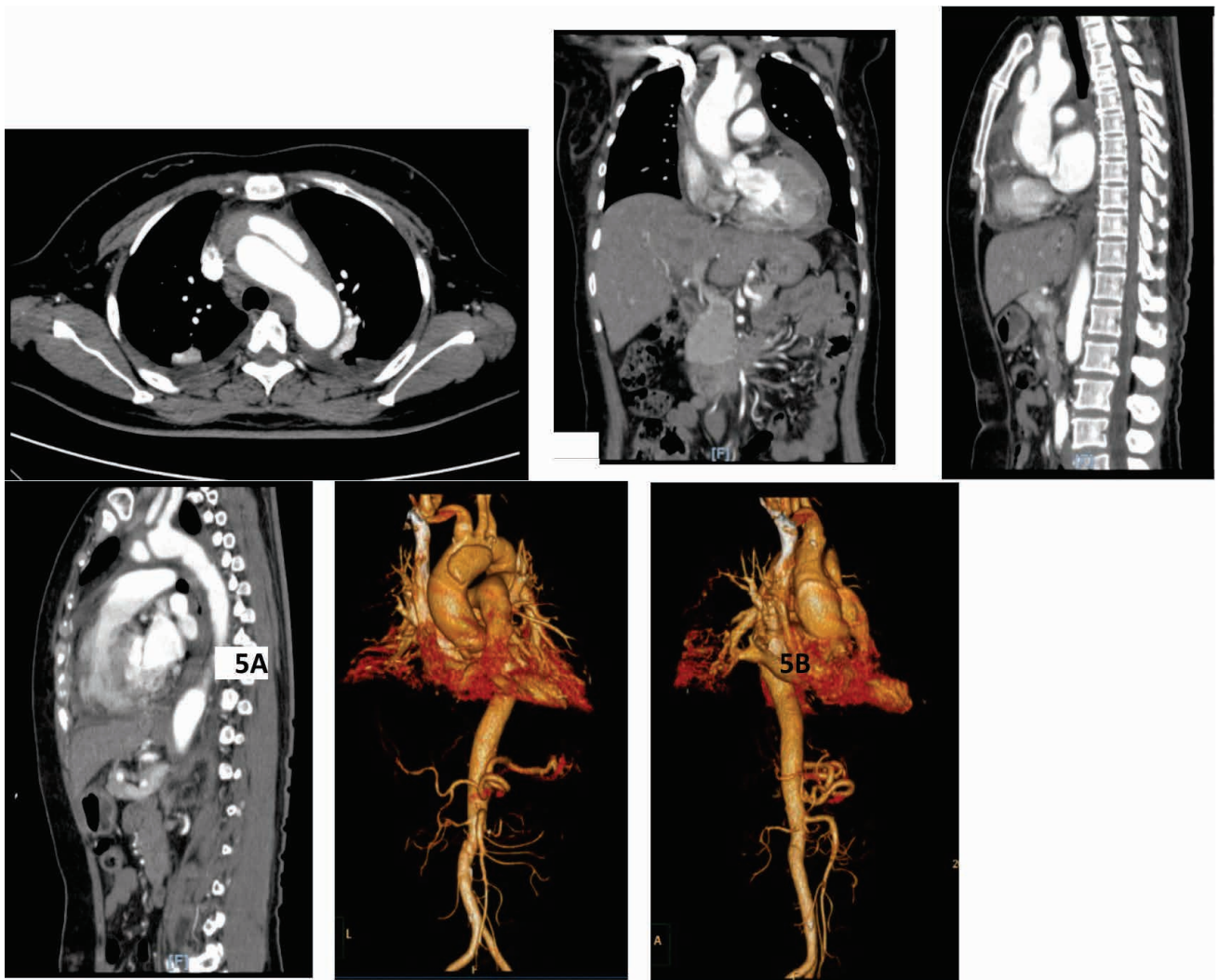
On the seventh day of treatment, the patient complained of worsening weakness on the right side of the body with a motoric grade of 2/2. We per-

formed a CT scan of the head, with a worsening of the ischemic area in the left caudate nucleus and left posterior internal capsule (Figure 6). After 14 days of treatment the patient was discharged, the hemodynamic was stable with right-sided motoric grade 3. The patient were planned for surgery at the next visit.

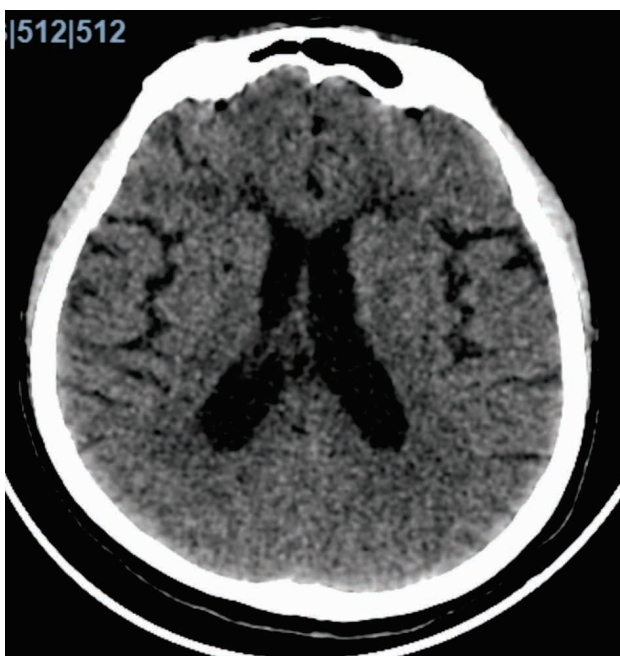
## DISCUSSION

The incidence of aortic dissection in the Oxford vascular study was 1/10,000 people per year. It is more common in men than in women and the incidence increases with age [3,4]. In the United States alone the incidence of aortic dissection is quite rare and estimates are based mostly on autopsy studies. Aortic dissection is reported in 1/10,000 hospital admissions and an estimated 2,000 new cases are reported annually in the United States [5].

The risk factors for thoracic aortic dissection are mostly due to conditions associated with increased wall stress (hypertension and physical trauma) and/or a primary abnormality in the aorta that makes



**FIGURE 5.** Thoracic CT scan axial, sagittal, transversal and coronal sections with contrast, and 3D reconstruction. **5A-5F.** An intimal flap that provides true lumen and false lumen of the ascending aorta and aortic arch (shown on arrows)



**FIGURE 6.** Follow-up CT scan of the head showed worsening of the ischemic area in the left caudate nucleus and left posterior internal capsule

the aorta susceptible to dissection (genetic abnormalities). Based on data from The International Registry of Acute Aortic Dissection (IRAD), the majority of patients (77%) had a previous history of hypertension [6].

The aortic wall is exposed to high pressure originating from the left ventricle and can cause shear stress, which makes the aorta more susceptible to injury and disease from mechanical trauma and more prone to tearing than other blood vessels especially in the presence of an underlying vascular aneurysm [5]. Acquired chronic conditions, such as hypertension and sometimes in combination with the atherosclerotic process, can cause thickening and fibrosis of the intima lining of blood vessels and degradation and apoptosis of smooth muscle cells. If the process continues, there can be necrosis and fibrosis of the elastic components of the arterial wall, which can diminish the elastic properties of the vessel wall and result in stiffness and weakness of the vessel wall, which can be the beginning of the tear that appears in this condition [5,6].

Other conditions that may increase the risk of acute aortic dissection include direct blunt trauma to the blood vessels, smoking, hyperlipidemia, cocaine use and pregnancy [7]. The possibility of aortic dissection acquired as a result of invasive procedures or cardiac surgery is also possible, but according to data from IRAD, the incidence is very low at only 0.06% with a good short-term and long-term prognosis [6].

The patient is a 55-year-old female patient, with a history of hypertension >10 years and not controlled by medication, no history of trauma, unknown and no congenital abnormalities in the blood vessels or direct manipulation of the aorta that caused this aortic dissection. Hypertension is the most likely cause of aortic dissection in patients.

Patients who present with a dominant neurological deficit with aortic dissection can cause delays in the diagnosis and treatment needed in patients with aortic dissection, especially with type A. The use of r-tPA therapy is currently indicated in patients with acute stroke onset. Stroke alone has been a poor prognostic factor in patients with aortic dissection [2,8].

Patients with ischemic stroke conditions usually present with hypertension. Where this can be a response to cerebral autoregulation to maintain per-

fusion in the penumbra so that there is no worsening of neurological status in the patient [9]. In the 2013 ASA guidelines, antihypertensive therapy is given to patients who are not candidates for fibrinolysis, with a systolic blood pressure threshold of > 220 mmHg or a diastolic blood pressure of > 120 mmHg. It is also of concern that in patients with aortic dissection the target systolic blood pressure of the patient is 100-120 mmHg and should be lowered immediately to reduce stress on the aortic wall and prevent expansion of the dissection [7]. Seeing that patient were still alert, we decided to continue antihypertensive therapy with the consideration that aortic dissection can cause higher mortality.

## CONCLUSIONS

The management of ischemic stroke in a patient with aortic dissection is challenging. Administration of antihypertensive drugs in the acute phase of stroke can impair perfusion to the penumbra, resulting in worsening of neurological symptoms. However, in ischemic stroke patient with aortic dissection, antihypertensives need to be given to reduce the stress on the aortic wall and prevent expansion of the dissection which has a higher mortality rate.

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