# Paraparesis and Acute Renal Failure as the Major Presentation of Aortic Dissection

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Aortic dissection (AD) is a serious condition with high mortality rate and needs early diagnosis and treatment. Most of the patients with AD present with suddenly severe pain in chest and mid-back area. However, painless dissection has occurred in some patients with AD. These patients have presented with the symptoms of vascular insufficiency in any organ which is supplied by the aorta. In this paper, I reported the patient with painless AD presenting with paraparesis and acute renal failure.

**บทคัดย่อ:** ผู้ป่วยภาวะ Aortic Dissection ที่มาด้วยอาการ ขาอ่อนแรงสองข้าง และไตวายเฉียบพลัน
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Aortic dissection เป็นภาวะฉุกเฉินและมีอัตราการเสียชีวิตสูง ดังนั้นการวินิจฉัยและการรักษาอย่างเร่งด่วนจึงเป็นสิ่งจำเป็น มิฉะนั้นอาจเป็นอันตรายถึงชีวิตได้ อาการเริ่มต้นที่พบบ่อยในผู้ป่วยภาวะนี้คืออาการเจ็บแน่นบริเวณหน้าอกและกลางหลัง อย่างไรก็ตามในผู้ป่วยบางรายอาจไม่มีอาการเจ็บในลักษณะเช่นนี้ได้ แต่จะมีอาการของอาการขาดเลือดในอวัยวะที่ได้รับเลือดจาก aorta เช่น ขาอ่อนแรง หรือไตวายเฉียบพลันเป็นต้น ต่อไปนี้เป็นรายงานผู้ป่วย Aortic dissection 1 ราย ที่มีอาการขาอ่อนแรงทั้ง 2 ขา ประมาณ 10 ชั่วโมงก่อนมาโรงพยาบาลและตรวจเลือดพบภาวะไตวายเฉียบพลัน แต่ไม่มีอาการเจ็บบริเวณหน้าอก

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**Introduction**

Aortic dissection (AD) is a serious condition with high mortality rate. Anagnostopoulus et al[1] reported the mortality rate of this condition was about 50% within first 48 hours of onset and about 80% within 2 weeks. More than 90% of the patients with AD present with a sudden, severe pain in the sub-sternal, interscapular or mid-back area. The pain can radiate to the abdomen, lower back, flank or extremities. However, painless dissection has occurred about 5% in patients with AD[2]. These patients have presented with the symptoms of vascular insufficiency in any organ that is supplied by the aorta such as acute myocardial infarction, cerebral ischemia, leg pain, acute renal failure (ARF) and paraparesis. In this paper I, reported the patient with painless AD who presented with paraparesis and ARF.

**Case Report**

A 40-year old Thai man with poor controlled hypertension (5 years), presented with paraparesis for 10 hours. One day ago, he developed pain at left side of back and radiated to left leg. He visited the rural hospital with this problem. Ten hours ago, the patient exhibited weakness and numbness at both legs. He was referred to Maharat Nakhon Ratchasima Hospital with the diagnosis of spinal cord disease and was admitted at Orthopedic department. On physical examinations, blood pressure 168/91 mmHg, he had normal level of consciousness but could not move his legs. The muscle power at both upper extremities was grade 5/5 according to Medical Research Council (MRC) Scale for Testing Muscle Strength and grade 0/5 at both lower extremities. The deep tendon reflexes at biceps, triceps and

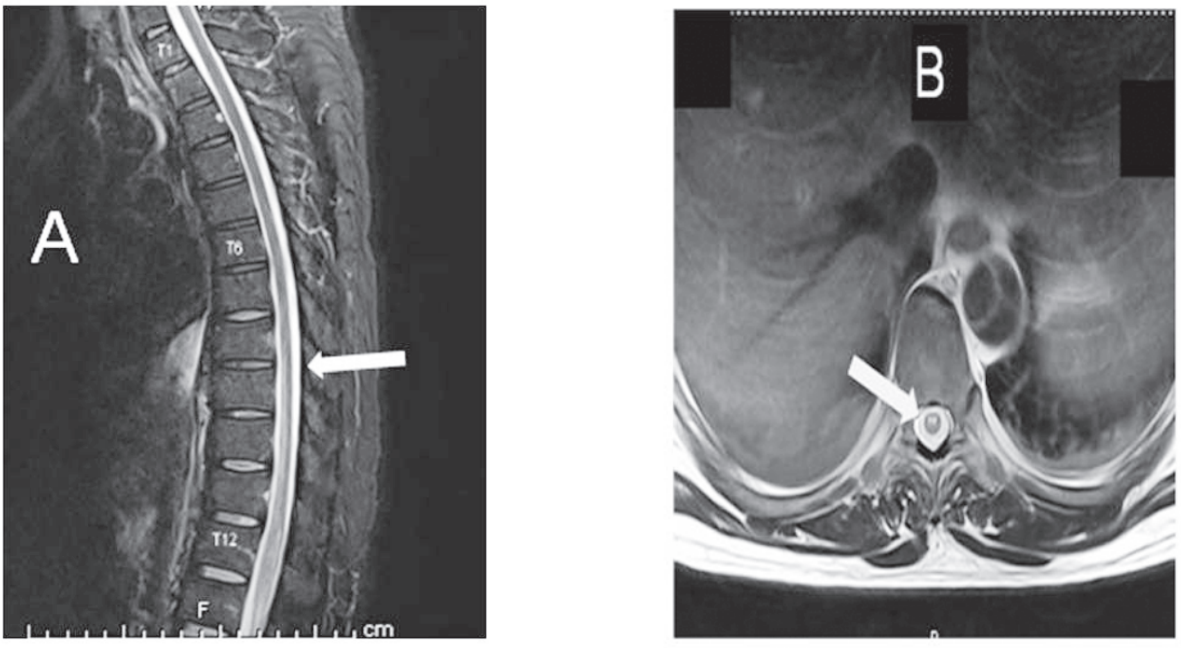


**Figure 1**: Abnormal high T2 signal intensity at grey matter (arrow) along 7th thoracic spinal cord level and downward to conus medullaris which is compatible with acute cord ischemia.

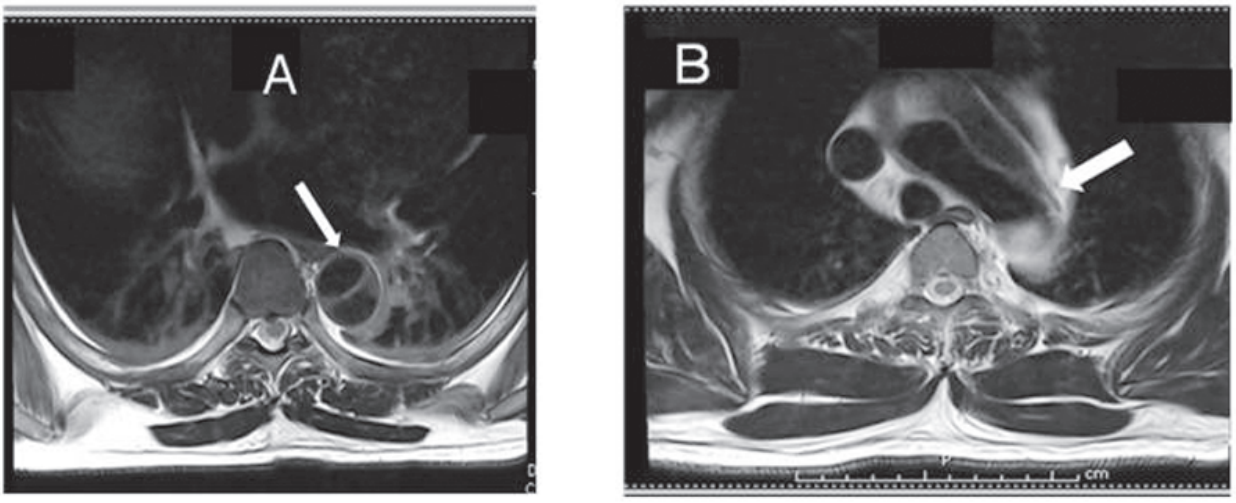


**Figure 2:** The intraluminal intimal flap (arrow) along ascending aorta (A) to aortic bifurcation (B), which is compatible with Stanford type A aortic dissection.

brachioradialises were normal bilaterally, but disappeared at quardriceps and gastrosoleuses both sides. The impairment of pinprick and joint positional sensation at the level below sternum ($6^{th}$ thoracic level) were found in this patient. His basic blood tests consisting of CBC and electrolyte were within normal range, except blood urea nitrogen (BUN) 41 mg/dL (normal range 7-18 mg/dL), creatinine 4.1 mg/dL (0.6-1.3 mg/dL), AST 2,530 unit/L (8-50 unit/L), ALT 1,278 unit/L (8-50 unit/L). The chest and spine films were unremarkable.

The Magnetic Resonance Imaging (MRI) was performed for identifying the pathology of the spinal cord. The study showed the abnormal high T2 signal intensity at grey matter along thoracic $7^{th}$ spinal cord level and downward to conus medullaris which was compatible with acute cord ischemia (figure 1A and 1B). The imaging also showed the intraluminal intimal flap along ascending aorta to aortic bifurcation, which was compatible with Stanford type A aortic dissection (figure 2A and 2B). The origin of both renal arteries arose from true lumen with relatively small luminal diameter of left renal artery, possibly dissection involving left renal artery (figure 3)

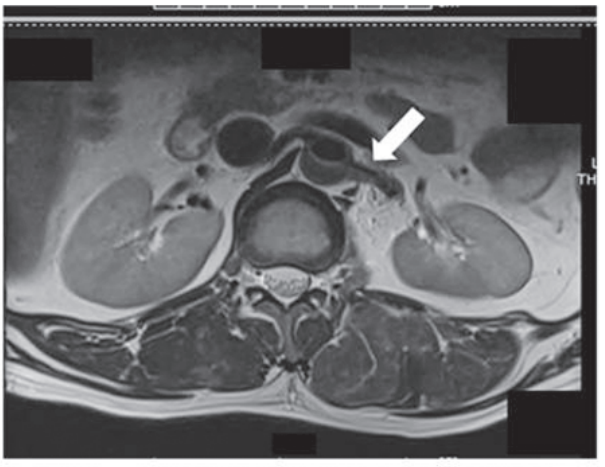

**Figure 3:** The origin of left renal artery arose from true lumen (arrow) with relatively small luminal diameter of left renal artery, possibly dissection involving left renal artery.

The patients was diagnosed as Stanford type A aortic dissection that involved along ascending aorta to aortic bifurcation, proximal to left renal artery and spinal cord infarction at 7th thoracic level to conus medullaris. The aortic graft replacement operation was performed immediately after the definite diagnosis. The patient had intra-operation uncontrolled bleeding. His blood pressure dropped persistently and he became unconscious. The patient expired 3 hours after the operation.

**Discussion**

There are three common neurological localizations that cause paraparesis: intracranial parasaggital, spinal cord and cauda equina. In this patient, sudden onset of paraparesis with loss of deep tendon reflexes and sensory impairment at the level below sixth-thoracic level indicated the thoracic cord lesion.

There are many causes of acute spinal cord disorder. Acute spinal cord ischemia is one of the causes of this condition. The causes of spinal cord infarction include the diseases or procedures which involve the thoracoabdominal aorta (aortic surgery, aortic dissection, traumatic rupture of aorta), intrinsic arterial occlusion resulted from arteriosclerosis, vasculitis (SLE, polyarteritis nodosa), infection (bacterial meningitis, syphilis), cardiogenic embolism and hypoperfusion (sepsis, hypovolumic)[3].

Spinal cord ischemia from AD can result in either anterior spinal artery syndrome or complete cord dysfunction and depends on the degree of disruption of spinal circulation. The pathophysiology of AD and the coexisting hemodynamic change can explain the relationship between AD and spinal cord ischemia. The tear of the aorta creates intimal hematoma and luminal occlusion progressing distally to its branches including the Adamkiewicz artery[4]. Normally, the Adamkiewicz artery is the most important arterial feeder of the lower one-third of the spinal cord. The most common level of the origin of this artery is 9th to 12th thoracic levels (89% in Thai population and 75% in Western population ) and left side more than right side of the aorta (64.1% versus 35.9%)[5-6]. In the previous study of 44 patients with spinal cord infarction, two patients (4.5%) had AD as the cause of spinal cord ischemia[7]. On the other hand, the incidence of AD which presented with paraparesis ranged from 2 to 8%[8].

There are two common causes of ARF in patients with AD. First, the compromise of blood supply to kidneys from dissection which involves both renal arteries and second, the pre-renal factors such as hypovolemic shock or exposure to contrast media[9]. However, the impairment of renal function from the latter in AD is mostly asymptomatic and only few cases of AD presenting with frank ARF have been reported in the previous literature[10]. In this patient, he had no pre-renal factors leading to ARF, so the involvement of renal arteries would be the cause of ARF.

In generally, the risk factors of AD are hypertension (about 70% of cases), male (2:1 ratio), atherosclerosis (about 30%), elderly (6th-7th decade), Marfan's syndrome, Ehlers-Danlos syndrome, bicuspid aortic valve (7-14% of cases), third trimester of pregnancy, trauma and cardiac surgery[11].

AD is divided into 2 types, Stanford A and B according to the ascending aorta involved[12]. If the dissection involves the ascending aorta and/or aortic arch, and possibly the descending aorta, it has been classified as Stanford type A. In addition, if the involvement has

limited at the descending aorta or the arch (distal to right brachiocephalic artery origin) without involvement of the ascending aorta, it has been classified as Stanford type B.

The prognosis of AD depends on type and management. In the study of 464 patients showed overall in-hospital mortality was 27%, and 30-day mortality was 26% in type A with surgical management, 58% in type A without surgical management, 31% in type B with surgical management and 11% in type B without surgical management[13].

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