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Single Case – General Neurology

Spinal Cord Ischemia Secondary to Aortic Dissection: Case Report with Literature Review for Different Clinical Presentations, Risk Factors, Radiological Findings, Therapeutic Modalities, and Outcome

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Keywords
Aortic dissection · Spinal cord ischemia

Abstract
Aortic dissection (AD) is a serious condition that causes transient or permanent neurological problems that include spinal cord ischemia (SCI), which occurs when AD extends into the descending aorta resulting in insufficient perfusion of segmental arteries that supplies the spinal cord. We report a 64-year-old male, presented with severe back pain, asymmetrical paresthesia, and weakness of both limbs, more in the left lower limb with loss of pinprick, temperature, and fine touch sensation on the lower left lower limb below the level of T5 with preserved proprioception and vibration and urine hesitancy. Computed tomography showed AD, Stanford type A, and spinal magnetic resonance imaging (MRI) showed hyperintense owl's eye sign at T5. The patient was diagnosed as anterior spinal artery syndrome secondary to an AD and referred for aortic surgical repair with good functional outcome. In our review to cases of SCI due to AD, it was more common in males above 55 years, pain only found in 47.8% of patients, with anterior cord syndrome on top of the clinical presentations, and hypertension is the most common risk factor. MRI spine showed thoracic location predominance. Surgical or endovascular repair especially for type A and complicated type B should be considered to

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avoid complications, and cerebrospinal fluid drainage is a very useful tool in reversing SCI specially if done early with favorable outcome. Only the old age is associated with increased risk of mortality. Early diagnosis and appropriate management are crucial for better outcome.

Introduction

Spinal cord infarction is a rare condition with few reliable estimates of its incidence. According to the previous studies, it accounts for 1.2% of all strokes [1] and 5–8% of all myelopathies [2]; however, recent studies have shown that myelopathy related to ischemic diseases accounts for 14–18% of patients with transverse myelitis, suggesting the underdiagnosis of SCI [3]. The age of onset ranges from the first decade to the tenth decade, with a median age between 50 and 70 years old [4]. SCI usually presents as anterior spinal artery syndrome or anterior spinal cord syndrome (ASCS) in up to 87.2% of the cases [5, 6]. SCI in territory of posterior spinal artery is very rare and involves posterior columns of the spinal cord. It presents with paresthesias and abolition of deep sensation below the level of the infarct. Occlusion of a central sulcal artery rarely produces small lesions in half of the spinal cord. This can present as an incomplete Brown-Séquard syndrome. Total transverse SCI involves both anterior and posterior spinal artery territory and may be misdiagnosed as transverse myelitis [7].

In one larger study of ASCI, 33% of cases were attributed to atherosclerotic disease, 16% to aortic pathology, and 16% to degenerative spine disease [2]. Approximately, 1% of patients presenting with acute type A aortic dissection will have spinal cord stroke [8]. A case of thoracoabdominal aortic aneurysm, with or without associated dissection, is also associated with spinal cord ischemia [9]. Also a significant number of cases of spinal cord ischemia occur in the periprocedural setting with up to 45% of all reported cord infarctions that are iatrogenic [10].

In this paper, we report a case of anterior spinal cord ischemia caused by aortic artery dissection (AAD) with literature review for other similar cases, aiming to come out with certain criteria for patient at risk, common clinical presentations, imaging findings, different therapeutic modalities, and outcome, hoping to help in improving the diagnostic and therapeutic yield of such rare yet devastating cases.

Case Presentation

We report a case of 64-year-old male known to be diabetic, hypertensive, dyslipidemic, and heavy smoker complicated with chronic obstructive pulmonary disease. In March 2020, he was presented to ER in our hospital with sudden severe progressive excruciating tearing interscapular back pain, which was radiating over the thorax posteriorly and spreading into the sides down the spine of 1-day duration, and it was continuous and progressively worsening. The pain was soon followed with weakness of both lower limbs, more on the left side. He was unable to walk, with loss of sensation in the left lower limb and hesitancy of micturition. There was no history of trauma or other cardiac or neurological symptoms.

On examination, he was fully conscious, oriented to time, place, and person, with normal speech, memory, and cranial nerves. Regarding the upper limbs, motor, sensory examination, and coordination were all normal including deep tendon reflexes). In the lower limbs, tone

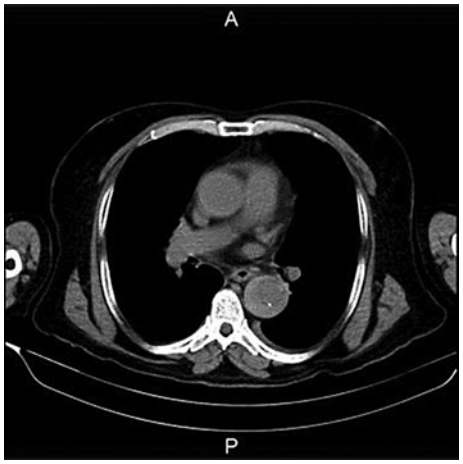

Fig. 1. CT axial view without contrast shows dilated ascending and descending aorta, and intramural thickening with wall calcification. CT, computed tomography.

was normal and power was 2 over 5 in the left lower limp and 4 over 5 on the right on MRS scale, and deep tendon reflexes including knee and ankle jerks were brisker on the left side than on the right. Planter response was extensor on the left and equivocal on the right side. Vibration and position sensation were normal in both lower limbs with no sacral hypoesthesia. However, pinprick, temperature, and fine touch sensations were impaired in the whole left lower limb up to L1, and it was normal on the right side. Examination of the spine revealed no tenderness, deformities, or bruises. On the second day, after 48 h of symptom onset, the patient showed a partial improvement to grade 4/5 on the left and 5/5 on the right side, but still sensory impairment was the same.

His blood pressure at admission was 177/92 mm Hg in both arms, and pulses in the upper limbs and carotids were normal but impaired in both lower limbs (femoral and popliteal). Cardiac examination revealed normal heart sound with regular rate and rhythm without any murmurs or gallops in auscultation. Respiratory and abdominal examinations were normal.

Regarding investigations, cardiac enzymes were done, including creatinine kinase (552 U/L) and troponin I (0.073 Ng/mL), and revealed an elevation, which is a suggestive of heart ischemic injury. ECG showed sinus rhythm/with diffuse deep *T* wave inversion in all leads. In addition, the lipid profile was elevated, in which total cholesterol was 244.7 mg/dL, cholesterol (HDL) was 58.10 mg/dL, cholesterol (LDL) was 163.3 mg/dL, and triglycerides were 187.90 mg/dL. Routine CBC and chemistry were all normal. In addition, thyroid function profile, coagulation profile, hemoglobin A1C, prostate-specific antigen, autoimmune profile, electrolyte profile (Na+, K+, Cl−), and creatinine level were all done and revealed normal results.

A posterior-anterior and lateral chest X-ray revealed bilateral accentuated bronchovesicular markings and dilated unfolded aorta, unfolded knuckle with right-side tracheal shift. Computed tomography (CT) of the brain was done and revealed normal findings. CT of thorax demonstrated that an enlarged left ventricle with extensive intramural hematoma extends along the whole course of the aorta down to its bifurcation (ascending, arch, and descending). This represents an atypical type of aortic dissection (AD) of type A Stanford classification. The intramural hematoma is seen of high attenuation in the precontrast phase with the total filling of the lumen at the postcontrast phase (Fig. 1, 2). No obvious intimal flap as well as no evidence of contrast leak could be detected. The aortic arch measures about 4.3 cm with residual patent lumen = 2.2 cm. In addition, the descending thoracic aorta measures about 4 × 3.6 cm with residual patent lumen = 2.4 × 1.8 cm. An echocardiogram showed

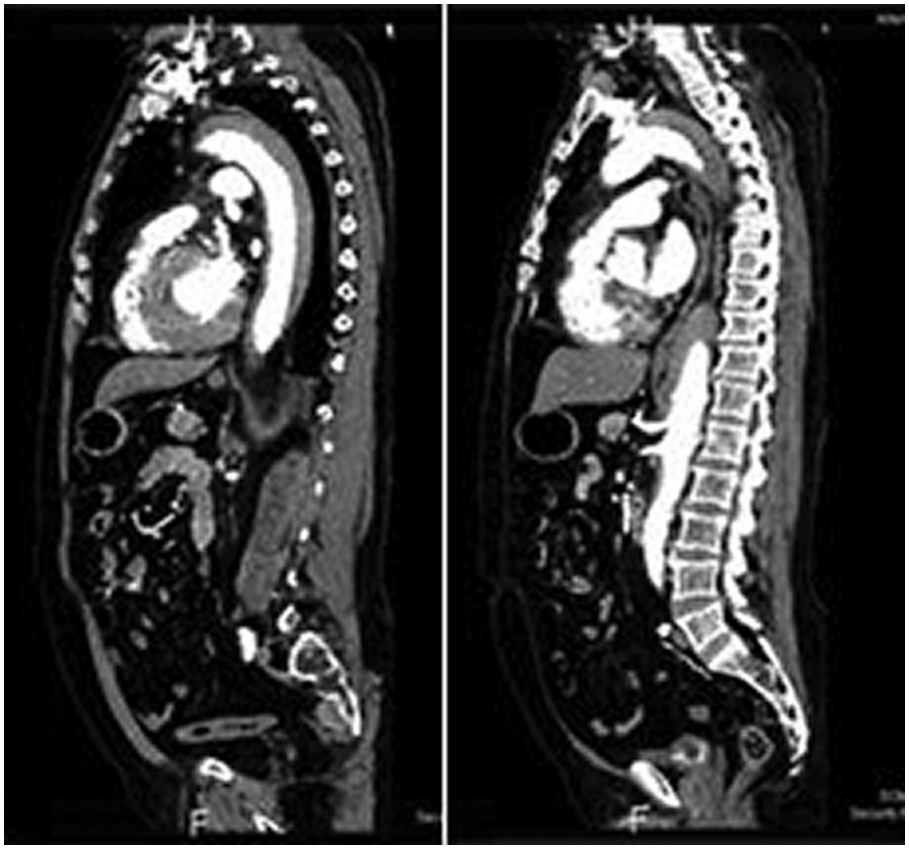

Fig. 2. CT sagittal view with contrast shows filling defect consisting of thrombus extending from the ascending to descending aorta. CT, computed tomography.

moderate-to-severe left ventricular systolic dysfunction (EF <30%), grade 1 diastolic dysfunction, multiwall motion abnormality, and mild dilatation in aortic root, and the rest of cardiac valves were normal.

Magnetic resonance imaging (MRI) of the dorsal spine revealed a small focal linear area of the abnormal intramedullary signal. It was noticed opposite to the T5 vertebral body, exhibiting a bright signal in T2-weighted image, short tau inversion recovery, and the isointense signal in T1-weighted image (Fig. 3). It mainly affects the ventral paramedian aspects of the cord, with subtle cord expansion. It measures about 2 cm in maximum craniocaudal length. No significant contrast enhancement could be detected (Fig. 4). MRI of the lumbar spine showed multiple degenerative features, L4 and L5 disc bulge and facet arthropathy, and L5 bilateral pars break. No evidence of cauda equina compression noticed.

The neurological findings were consistent with acute asymmetric anterior cord syndrome, rather than Brown-Séquard syndrome nor complete anterior cord syndrome. The following conditions were considered: spinal cord infarction, myelitis, sudden compression from secondary versus deposits, hematomyelia, and acute demyelination. The presence of significance and continuous interscapular back pain was in the presence of normal strangle, and suggestion of AD supports the diagnosis of acute vascular lesion of the spinal cord.

During the hospital course, the patient was on paracetamol and naproxen. In addition, he was managed with oral amlodipine and this resulted in reduction of blood pressure. The patient referred to a cardiac surgeon where a Bentall surgery was performed for him. The

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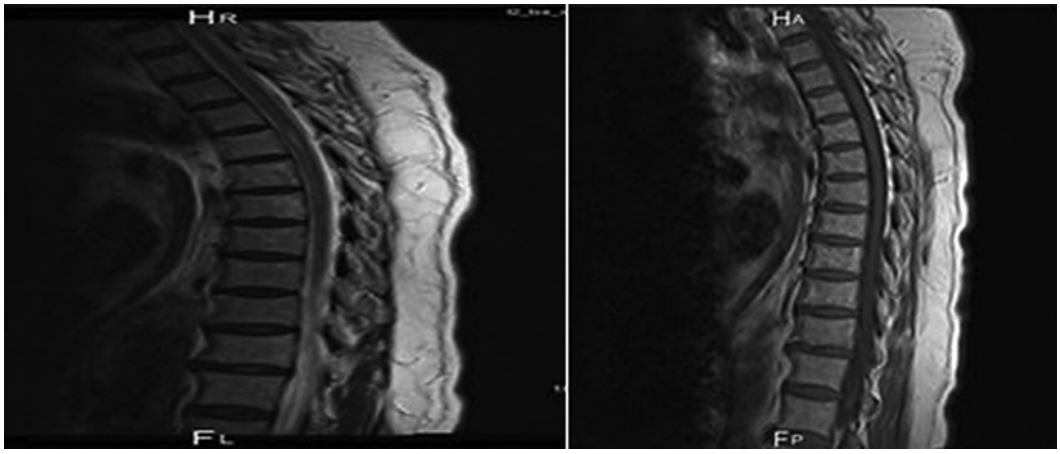

Fig. 3. a MRI T2 sagittal view show small focal linear intramedullary hypodensity at the level of T5. **b** MRI T1 sagittal view shows iso-intense spinal cord. MRI, magnetic resonance imaging.

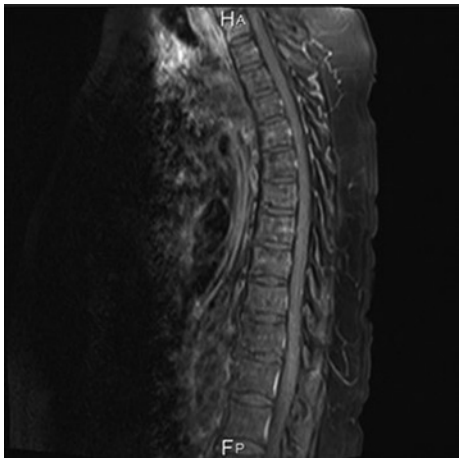

Fig. 4. MRI sagittal view postenhanced was unremarkable. MRI, magnetic resonance imaging.

composite graft replacement was done regarding ascending aorta, arch, and coronary artery. Postsurgery outcomes include significant improvement of the pain and remarkable recovery, and no postoperative complications were detected. After the surgery, the patient undergone physiotherapy and started to walk independently 1 week after surgery.

Discussion

Transient or permanent neurological symptoms at onset of AD are often dramatic and may mask the underlying condition especially in pain-free dissection (5–15%). They are usually caused by either dissection/occlusion of one or more aortic side branches supplying brain, spinal cord, or peripheral nerves or hypoperfusion. They usually appear at or shortly after the onset of dissection with rapid improvement resulting from transient arterial occlusion at the moment of propagation of the dissection [8, 11]. Their frequency varies between 17 and 40%, including persistent or transient ischemic stroke (in 2.6–32%), ischemic neuropathy (in 4.2–24%), and less commonly spinal cord ischemia (in 1–8.9%), as well as hypoxic encephalopathy and syncope in some patients [10, 12–16].

SCI on the basis of AD is a rare syndrome and more common with distal ADs. In a study by Sandhu et al. [17], from 1999 to 2014, they managed 978 AADs, comprising 482 with AD type A (88% DeBakey type I and 12% type II) and 496 with AD type B (96.4% type III and 3.6% abdominal). Neurological symptoms were present in 178 (18.2%), of which 52 (29.2%) presented with SCI. Isolated SCI (paraplegia/paraparesis in the absence of other malperfusion symptoms) was present in 28 AADs (2.9%). All SCIs occurred in DeBakey type I or III ADs. Of these 52 patients, 10 were females and 42 males. Chest pain was present in 40 patients, HTN in 22 patients, smoking in 16 patients, and genetic syndrome in 5 patients. Twenty-four patients undergone open surgery and 3 patients undergone endovascular surgery. Ten patients died, partial recovery in 5 patients, and complete recovery in 26 patients.

We conducted a systematic meta-synthesis literature review for cases of SCI caused by AD through searching the PubMed till April 2021. We found another 66 cases to fulfill our search criteria plus our case. Analyzing data from those 67 cases, we tried to explore the patient criteria, common presentations, risk factors, radiological findings, therapeutic interventions, and outcome in such cases (Table 1) [18–73].

Mean age and gender distribution in AD patients with neurological involvement do not differ from those without neurological involvement [11, 14]. In our 67 collected cases, 21/63 (33.3%) were females and 42/63 (66.6%) were males, with 4 cases sex not available (Table 2). So, the number of males doubles the number of females. As for age, it was ranged from 40 to 92 with 68.5% above 55 years, with a mean age of 60 and only 1 case aged 24 years (Table 2). This comes in agreement with Mayo Clinic who reported that male sex and age from 60 to 80 years old consider being one of the potential risks of AD [74].

Owing to the ischemic pathology, the onset of symptoms is usually acute and this was the case in all 67 cases. Pain is the most common presenting symptom of AD and could be the sign that directs the physician attention to think about AD as etiology for a case of paraplegia, with 95% of patients reported any pain, usually midline, in front and back of trunk depending on the location of dissection, localized to chest in 73%, anterior >posterior (61 vs. 36%), back in 53%, and abdomen in 30% of patients, which may extend down the back to the hips and legs in cases where dissection process extends distally [112]. Remarkably, chest pain is not an obligatory symptom of AD, and the frequency of pain-free dissections ranges between 5 and 15% [11, 12, 75, 112] especially in patients with neurological sequelae [11, 19, 20, 75–82]. In a study by Gaul et al. [8], only two-thirds of the patients with neurological symptoms at onset of dissection complained of pain, whereas most patients without neurological symptoms (94.4%) experienced initial pain. Approximately half of all patients who did not report pain showed neurological symptoms only [11], which make the diagnosis very challenging. In our review, pain was present in 32/67 (47.8%), which is much less expected in usual cases of AD (95%). Most patients experienced severe chest pain (18 cases [34.4%]), extended to the back in 2 cases, and localized to back only in another 11 (40.6%) cases (Table 3). The pain was usually severe, continuous, and excruciating.

Clinical manifestations of SCI comprise complete spinal cord infarction as well as ASCS, Brown-Séquard syndrome, progressive myelopathy, or transient spinal cord ischemia [79, 81, 83, 84]. Pure posterior spinal artery (PSA) infarction in SCI is relatively rare based on previous case studies [85–87]. In a recent study that comprised of the largest series of PSA infarctions (133 patients with SCI), 15 (11%) patients had a spontaneous PSA infarction [88]. This figure suggests that the diagnosis of PSA infarction might be underrecognized in SCI [86]. In our review, the presentation was anterior cord syndrome in 31/67 (46.2%) followed by pure motor in 26/67 (38.8%), then complete cord syndrome in 5/67 (7.4%), pure sensory in 2/60 (2.9%), Cauda equine syndrome in 2/67 (2.9%), and Brown-Séquard syndrome in 1/67 (1.49%). None of them had isolated posterior cord syndrome (Table 3).

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Table 1. Summary of literature review for cases of spinal cord ischemia secondary to AD

N	Citation	Sex	Age	Bladder	Symmetrical/ asymmetrical	Sensory level	Affected artery	Stanford AD type	Chest pain	Outcome	Treatment	MRI spine	Risk factors
1	Waltimo and Karli [18]	M	52	None	Symmetrical	No	N/A	A	No pain	Death	Conservative	N/A	HTN
2	Waltimo and Karli [18]	M	46	None	Symmetrical transient paraparesis	No	N/A	B	Intensive chest pain	Walk	Conservative	N/A	N/A
3	Waltimo and Karli [18]	M	56	None	Symmetrical paraplegia	No	N/A	B	Intensive chest pain	Plegic	Repair	N/A	N/A
4	Gerber et al. [19]	F	69	None	Symmetrical	T11 and T5	N/A	A	No	Death	None	N/A	Hypothyroidism, HTN
5	Gerber et al. [19]	M	78	None	Asymmetrical left leg weakness and ischemia	N/A	Right superficial femoral	A	No	Plegic	None	N/A	HTN
6	Rosen [20]	F	67	None	Transient symmetrical paraplegia	None	Adamkiewicz artery	A	No	Death	Conservative	N/A	None
7	Zull and Cydulka [21]	F	63	None	Symmetrical	None	Anterior spinal	A	Yes	Death	None	N/A	HTN
8	Zull and Cydulka [21]	M	67	None	Symmetrical transient	None	Anterior spinal	A	No	Death	None	N/A	HTN
9	Tanaka et al. [22]	M	66	None	Symmetrical paraplegia	Below T9	Anterior and posterior spinal	B	No	Walk	Conservative	N/A	HTN
10	Holloway et al. [23]	F	92	No	Symmetrical paraplegia	No	N/A	A	No	Death	Conservative		N/A
11	Krishnamurthy et al. [24]	M	80	No	Symmetrical paraplegia, parasthesia	N/A	N/A	B	Back and leg pain	Death	None	N/A	None
12	Kellett et al. [25]	M	65	No	Symmetrical paraparesis	No	N/A	B	Chest pain	Walk	Conservative	N/A	IHD, MI
13	Beach and Manthey [26]	F	58	None	Asymmetrical	N/A	Left common femoral	A	No	Walk	Open surgery	N/A	HTN
14	Lacerda et al. [27]	F	67	N/A	Symmetrical paraplegia	N/A	N/A	B	No	Death	Conservative	N/A	N/A
15	Donovan et al. [28]	F	77	No	Symmetrical	T6–S5	Adamkiewicz	A	No	Plegic	Conservative	N/A	Emphysema, angina, HTN, MVP, smoking

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Table 1 (continued)

N	Citation	Sex	Age	Bladder	Symmetrical/ asymmetrical	Sensory level	Affected artery	Stanford AD type	Chest pain	Outcome	Treatment	MRI spine	Risk factors
16	Joo and Cummings [29]	F	63	No	Symmetrical (pure sensory)	T12–S5	Adamkiewicz	N/A	No	Walk	Conservative	N/A	Chronic arthritis
17	Inamasu et al. [30]	M	50	No	Symmetrical	L1–S5	Adamkiewicz	A	No	Plegic	Conservative	N/A	Small lacunar stroke, HTN
18	Killen et al. [31]	M	57	None	Asymmetrical paraparesis, no sensory	N/A	N/A	N/A	Severe upper back pain	Walk	CSF drainage, intravenous naloxone drip	N/A	HTN, brain stem stroke
19	Syed and Fiad [32]	M	32	No	Asymmetrical paraparesis, paraesthesia Transient	Below knee	N/A	A	Chest pain	Walk	Open surgery	N/A	Non-hodgkin's lymphoma
20	Petal et al. [33]	F	65	Incontinence	Symmetric paraplegia	L2	N/A	B	Back pain	Death	None	N/A	Smoking
21	Ohmi et al. [34]	NA	NA	None	Symmetrical paraplegia	T9–10	N/A	B	Severe upper back pain	Plegic	Conservative	Atrophy at T9-10	NA
22	Blacker et al. [35]	F	66	Yes	Complete ASAS	N/A	N/A	B	No	Walk	CSF drainage	N/A	HTN
23	Ogun et al. [36]	M	46	Sphincter dysfunction	Symmetrical	T6–S5	Anterior spinal	N/A	Yes	Death	Conservative	N/A	HTN
24	Hsu and Lin [37]	M	55	None	Symmetrical paraplegia	T10	Adamkiewicz	A	Yes	Plegic	Conservative	Spinal MRI Lesion from T10 downward	HTN
25	Hsu and Lin [37]	F	64	None	Symmetrical paraplegia	None	N/A	A	No	Walk	Open surgery	Normal	HTN
26	Hsu and Lin [37]	M	67	None	Symmetrical paraplegia	T11	Adamkiewicz	A	No	Death	Open surgery	N/A	HTN, CHF
27	Hsu and Lin [37]	M	68	None	Symmetrical paraplegia	T5	Adamkiewicz	B	Yes	Death from sepsis	Conservative	N/A	HTN, DM, MI
28	Chiang et al. [38]	F	74	None	Symmetrical	T8–S5	Adamkiewicz	A	Yes	Walk	Open surgery	N/A	None
29	Fujisawa et al. [39]	N/A	N/A	N/A	Symmetrical	N/A	N/A	B	N/A	Walk	Conservative	N/A	N/A
30	Fujisawa et al. [39]	N/A	N/A	N/A	Symmetrical	N/A	N/A	B	N/A	Walk	Conservative	N/A	N/A

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Table 1 (continued)

N	Citation	Sex	Age	Bladder	Symmetrical/ asymmetrical	Sensory level	Affected artery	Stanford AD type	Chest pain	Outcome	Treatment	MRI spine	Risk factors
31	Altuwaijri et al. [40]	F	51	No	Symmetrical transient	No	N/A	B	Chest pain	Walk	Open aortic fenestration	Normal	HTN, smoking, peptic ulcer with Roux-en-Y bypass
32	Aktas et al. [41]	M	54	None	Symmetrical	None	N/A	A	No	Walk	None	N/A	None
33	Aktas et al. [41]	M	54	None	Symmetrical	None	N/A	A	No	Walk	None	N/A	HTN
34	Holper et al. [42]	F	63	Yes	Anterior cord syndrome	N/A	N/A	B	Back pain	Plegic	CSF drain	N/A	None
35	Karacostas et al. [43]	M	46	Retention	Symmetrical	Below T7	Anterior spinal	B	No	Death	None	Cord dilation at the T9–T12 level on sagittal T1- with No enhancement and increased signal on T2	Ankylosing spondyloarthritis, HTN
36	TaHsieh et al. [44]	M	24	None	Asymmetrical right LL only	N/A	Right common iliac	B	Abdominal pain	Walk	Conservative	N/A	Aortic insufficiency aortic valve replacement Marfan's syndrome
37	Zeggeren et al. [45]	M	62	Retention	Symmetrical paraplegia	T9-T12	Anterior spinal	B	No	Walk	Conservative	MRI showed a patchy increased signal intensity of the anterior spinal cord between T3 and T7 on the T2-weighted images	AF
38	Hayatsu et al. [46]	M	65	None	Symmetrical	T10	Adamkiewicz	B	Sever back pain	Walk	CSF drainage	N/A	None
39	Sui et al. [47]	M	50	Retention	Symmetrical	N/A	Anterior and posterior spinal	A	No	Death	Conservative	Long T2 signal at thoracic level	HTN
40	Colak et al. [48]	M	51	None	Symmetrical	N	N/A	A	No	Walk	Open surgery	N/A	Coronary artery bypass grafting, HTN
41	Lynch et al. [49]	M	54	Incontinence	Symmetrical, proximal	T4-T7, spare deep	Anterior spinal	B	Severe chest pain	Walk	CSF drainage	Abnormal hyperintense signal within the anterior portion of the central grey matter from level T4–T7 with associated mild cord swelling	HTN
42	Tsiouris et al. [50]	N/A	N/A	N/A	Symmetric paraplegia	N/A	Adamkiewicz	A	No	N/A	N/A	N/A	N/A
43	Hui and Lau [51]	M	70	No	Symmetrical paraparesis	No	Adamkiewicz	B	Chest, abdominal and back pain	Walk	Conservative	T2 hyperintensity in central aspect of spinal cord extending from T11/12 to L1	Smoking

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Table 1 (continued)

N	Citation	Sex	Age	Bladder	Symmetrical/ asymmetrical	Sensory level	Affected artery	Stanford AD type	Chest pain	Outcome	Treatment	MRI spine	Risk factors
44	Rabadi [52]	M	60	None	Symmetrical	T6	N/A	B	No pain	Death	None	Normal	HTN, dyslipidaemia, aortic aneurysm repair 10y back
45	Ullery et al. [53]	F	64	None	Symmetrical paraplegia	N/A	N/A	A	Yes	Walk	Conservative	N/A	HTN, AF
46	He et al. [54]	M	40	Yes	Cauda equina syndrome	N/A	Feeding arteries of cauda equina	B	LBP	Walk	Endovascular aortic repair	N/A	N/A
47	Yu et al. [55]	M	56	None	Symmetrical paraplegia	No	Infrarenal abdominal aorta and bilateral iliac	A	Severe back pain	Walk	Open surgery	N/A	None
48	Almenara et al. [56]	M	64	No	Symmetrical	L1–S5	Left renal and external iliac	N/A	No	Plegic	Open surgery	Widening of the spinal canal and spinal cord with hyperintensity in the T2-weighted and FLAIR sequences, between T9 and T10 and the end of the conus medullaris, and abnormal diffusion restriction	None
49	Hdiji et al. [57]	M	70	Retention	Symmetrical	None	N/A	A	No	Death	None	Normal	None
50	Hughes et al. [58]	F	56	None	Asymmetrical	Below T10	Anterior spinal	A	No	Death	Open surgery	N/A	COPD, smoking
51	Martínez et al. [59]	M	72	None	Symmetrical	N/A	Lumbar spine's spinal	B	Yes	Death	None	N/A	HTN, DM
52	Prakash et al. [60]	F	45	Incontinence	Symmetrical	T10	N/A	A	No	Plegic	Open surgery	N/A	Marfan syndrome
53	Yildiz et al. [61]	M	74	None	Symmetrical	Below T12, preserved deep sensation	N/A	B	Back pain	Death	Conservative	Sagittal T2 hyperintense, "pencil-like" signal change on the anterior of the spinal cord at T8-12 levels Axial T2 weighted showed T2 hyperintense signal change patterns called "snake eyes" or "owl's eyes" in the center of the spinal cord	HTN
54	Sekine et al. [62]	M	69	Yes	Brown-Séquard	T10	Anterior and posterior spinal	B	No	Walk	Conservative	SCI in the right posterior area of the spinal cord at level T7/8 and the conus medullaris	Previous coronary artery bypass grafting
55	Niclauss et al. [63]	M	M 49	None	Symmetrical transient paraplegia	T10	N/A	B	Sudden onset of chest and back pain	Walk	Repair	N/A	No

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Table 1 (continued)

N	Citation	Sex	Age	Bladder	Symmetrical/ asymmetrical	Sensory level	Affected artery	Stanford AD type	Chest pain	Outcome	Treatment	MRI spine	Risk factors
56	Cheng et al. [64]	F	53	No	Symmetrical	T8–S5	Adamkiewicz	A	Yes	Plegic	Open surgery	N/A	Marfan syndrome, history of tuberculosis
57	Atsuyuki et al. [65]	F	85	Retention	Symmetrical	T4–S5	Sulcal artery	B	No	Walk with use T-cane	Conservative	T2 high signal intensity lesion at T3–T10	HTN
58	Atsuyuki et al. [65]	M	68	Retention	Symmetrical	L2–S5	Adamkiewicz	B	No	Plegic	Conservative	T 2high signal intensity in the conus medullaris With restricted diffusion	None
59	Strohm et al. [66]	M	61	Yes	Bilateral lower extremity weakness	T4	Not identified	B	Severe chest pain	Walk	CSF drainage	Normal	HTN, HLD
60	Tsushima et al. [67]	M	57	None	Symmetrical	N/A	N/A	A	No	Death	None	T2 signal intensity and diffusion restriction predominantly involving the central gray matter of the spinal cord extending from the T4–T11 level	None
61	Quintana et al. [68]	M	42	None	Symmetrical paraplegia	N/A	Adamkiewicz	A	Yes	Walk	Open surgery	Normal	None
62	Memon et al. [69]	F	45	None	Numbness in LL below umbilicus	T11	N/A	A	Severe back pain	Walk	Repair	N/A	HTN, smoking
63	Takeda et al. [70]	M	62	None	Asymmetrical	N/A	Adamkiewicz	B	No pain	Walk	Repair	Normal	Smoking
64	Sabugueiro and Olson [71]	F	56	Yes	Symmetrical (cauda equina syndrome)	Saddle anesthesia	N/A	A	LBP	Death	None	Abnormal high signal detected within the distal cord and conus	None
65	Kim et al. [72]	M	62	None	Asymmetrical	N/A	Left renal artery, left intercostal and left lumbar branches	B	Yes	Walk using a q-cane	Open surgery	Left asymmetric increased T2 signal intensity of the spinal cord from T11–L2 level	HTN
66	Nahed and Rizk [73]	M	53	None	Symmetrical	T4	N/A	A	No pain	Walk (transient)	Repair	Normal	None
67	Our case	M	64	Yes	Asymmetrical paraparesis and sensory	T5	N/A	A	Severe back and chest pain	Walk	Open surgery	T5 hyperintense lesion	HTN, DM, asthmatic, dyslipidaemia

N/A, not available; M, male; F, female; HTN, hypertension; DM, diabetes; COPD, chronic obstructive pulmonary disease; AF, atrial fibrillation; MI, myocardial infarction; CHF, congestive heart failure; T, thoracic; L, lumbar; S, sacral; AD, aortic dissection; CSF, cerebrospinal fluid.

Table 2. Sociodemographic criteria among the studied patients

Parameters	The studied patients *N*= 67
Age, years	
Mean±SD	60.06±11.60
Range	24–92
Sex, *n* (%)	
N/A	4 (6.0)
Male	42 (62.7)
Female	21 (31.3)
Risk factors, *n* (%)	
HTN	32 (47.8)
Smoking	7 (10.4)
DM	3 (4.5)
Cardiovascular (MI, angina, stroke, AF, CHF, MVP, CABG)	12 (17.9)
Hypothyroidism	1 (1.5)
Respiratory (asthma, COPD)	4 (6.0)
Arthritis	2 (3.0)
Marfan syndrome	2 (3.0)
Non–Hodgkin's lymphoma	1 (1.5)

N/A, not available; MI, myocardial infarction; CHF, congestive heart failure; AF, atrial fibrillation; MVP, mitral valve prolapse; CABG, coronary artery bypass graft; COPD, chronic obstructive pulmonary disease; HTN, hypertension.

The symptoms are almost bilateral symmetrical since both halves of the anterior spinal cord are supplied from one anterior midline spinal cord. However, the ASCS with unilateral symptomatology has rarely been reported; this may be due to occlusion of unilateral sulcal arteries or collateralization from one posterior spinal artery [89] as it could be the explanation to our case with asymmetrical incomplete paraparesis with loss of sensation on left LL till L1 and also in 10 other cases from the literature totally 11/67 (16.49%), being strictly unilateral in only 1 case.

As we mentioned before, the symptoms could be permanent or transient. Seven out of sixty-seven cases (10.4%) had transient symptoms with spontaneous recovery in 4 cases, and conditioned recovery after intervention in 3 cases (1 after endovascular fenestration and 2 after CSF drainage).

If the location of the infarction involves the lateral horns within levels T1-L2 of the spinal cord, it will cause autonomic dysfunction, including neurogenic bowel/bladder, which requires bladder catheterization [90, 91]. Bladder symptoms in the form of hesitancy, retention, or incontinence were found in 17/63 (26.9%) patients, with the level between T3 and L1 (Table 3).

The most common risk factor for AD is poorly controlled hypertension (HTN) (65–75% risk with a history of HTN) [74]. Other risk factors include age, male sex, smoking, preexisting aortic diseases or aortic valve disease, family history of aortic diseases, history of cardiac surgery, direct blunt trauma, and the use of intravenous drugs (such as cocaine or amphetamines) [92]. In our review, HTN was on the top of the vascular risk factors being present in 32/62 (51.6%) cases. Out of 62 patients, 7 patients (11.29%) were smokers, 3 with DM, 2 with

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Table 3. Clinical data among the studied patients

Parameters	The studied patients (*N* = 67)
Bladder	
N/A	4
Yes	17
No	46
Symmetry	
Symmetrical	56
Asymmetrical	11
Affected artery	
N/A	35
Adamkiewicz	15
Anterior spinal	7
Anterior and posterior spinal	3
Femoral arteries	2
Iliac arteries	1
Sulcal arteries	1
Feeding arteries of cauda equine	1
Renal and iliac arteries	2
Stanford AD type	
N/A	4
A	32
B	31
Pain, *n* (%)	
Yes	32 (47.8)
No	35 (52.2)
Pain location	*N* = 32
Chest	18
Back	11
Chest and back	2
Chest, back, and abdomen	1
Duration, *n* (%)	
Transient	7 (10.4)
Permanent	60 (89.6)
MRI findings, *n* (%)	
N/A	43
Normal	8
Thoracic	10
Conus	2
Thoracic and conus	2
Thoracolumber	1
Thoracolumber and conus	1
Treatment, *n* (%)	
N/A	1
No treatment	14
Conservative	25
CSF drainage	6
Endovascular	2
Open surgery	19
Outcome, *n* (%)	
N/A	1
Walk	34 (52.2)
Plegic	11 (16.4)
Death	21 (31.3)

N/A, not available; CSF, cerebrospinal fluid; MRI, magnetic resonance imaging; AD, aortic dissection.

Marfan syndrome (3.2%), 2 had previous stroke, 2 had previous history of angina, 2 had coronary artery grafting, 2 with asthma, 1 with mitral valve prolapse (1.6%), 1 with emphysema, 1 with chronic obstructive pulmonary disease, 1 with atrial fibrillation, 1 with congestive heart failure, 1 with hypothyroid, and 1 with non–Hodgkin's lymphoma (Table 2).

Diagnosis of spinal cord ischemia is done by taking a detailed history, performing physical examination, and also neuroimaging studies. MRI of the spine is usually done to confirm the diagnosis, although, in the first 24 h, the results may appear negative. Hyperintensity in the anterior horns in the T2-weighted image is the hallmark observation. Anterior spinal artery syndrome findings indicate a thin "pencil-like" hyperintense region that spreads vertically affecting several spinal levels in the sagittal view, 2 bright dots at each anterior horn on the axial view identified as owl's eyes, T1-weighted hypointensity at the injured area, spinal cord expansion at the injury site due to early signs of inflammation/edema (diffusion-weighted images that help distinguish between ischemia and inflammation), and signs of vertebral body infarction that are not always present are other results indicating a spinal cord infarction [89, 93, 94]. In our review, out of the 24 cases undergone MRI spine, 8 cases were normal and the other 16 cases showed hyperintense T2 lesions in different locations, 1 delayed MRI shows cord atrophy, with the typical owl eye sign found only in 3 cases.

In a study by Hsu et al. [91], comparing SCI in patients with to those without vessel dissection, it was found that in the vessel dissection group, patients frequently had lesions involving the upper cervical (C1–C4) and lower thoracic (T10–T12) vertebral body levels. In contrast, patients without vessel dissection more frequently had lesions distributed in the cervical regions (C5–T7) than in the thoracolumbar regions, with more posterior involvement [92]. In our review, out of the 16 cases with positive MRI, the level was as high as T3 and as low as conus medullaris with no cases with cervical affection, being at a thoracic level in 10/24 (41.6%) cases, conus in 2/24 (8.3%) cases, thoracic and conus in 2/24 (8.3%) cases, thoracolumbar in 1/24 (4.16%) case, and thoracic, lumbar, and conus in 1/24 (4.16%) case (Table 3).

Spinal cord involvement in patients with AD could be secondary to obstruction of the intercostal and lumbar arteries, the Adamkiewicz artery (arteria radicularis magna), or the thoracic radicular arteries. Most frequently, the middle thoracic spinal cord, the watershed zone between the territories of the artery of Adamkiewicz, and the thoracic radicular artery are affected [83]. Among the 32 cases with well-defined occluded artery, 15/32 (46.8%) had Adamkiewicz artery occlusion, followed by 7/32 (21.8%) with anterior spinal artery, 3/32 (9.3%) with combined anterior and posterior spinal artery occlusion, 2/32 (6.25%) with left renal left renal and external iliac arteries occlusion, and another 2 cases with right brachiocephalic and right iliac arteries. Each of sulcal artery, right superficial femoral artery, left common femoral artery, right common iliac artery, lumbar spine's spinal arteries, left renal, external iliac arteries, and bilateral iliac arteries was found in one case and one more case with occlusion of feeding arteries to cauda equina (Table 3).

For confirmation of the diagnosis, patients often require more than one noninvasive imaging study to characterize AD, with CT used in 61% of cases, echocardiography in 33%, aortography in 4%, and MRI in only 2% [74]. Imaging helps in diagnosis and classification of the AD in order to decide best the therapeutic plan. Two classifications are most commonly used for AD. The DeBakey system is classified into 3 types (types I, II, and III) according to the site of the first entry of dissection [95]. Type I has the first entry in the ascending aorta and propagates distally to the descending aorta. Type II has the first entry in the ascending aorta and does not propagate to the aortic arch. Type III has the first entry in the descending aorta and propagates distally above (type IIIa) or below (type IIIb) the diaphragm. The Stanford system is classified into 2 types (types A and B) based on the involvement of the ascending aorta [96]. Type A includes dissection in the ascending aorta regardless of the

site of first entry. Type B does not include dissection in the ascending aorta. The usual incidence of different types of AD in a previous study was 37 (71.2%) Stanford type A and 15 (28.8%) type B [97]. In our review, the type of AD was identified in 63 cases; it was type A in 32/63 (50.7%) cases and type B in 31/63 (49.2%) cases with almost equal incidence (Table 3).

Regardless of whether acute AD is type A or B, medical therapy to control pain and HTN is essential in all patients. Beta blockers have the desired effect of reducing blood pressure and heart rate to the normal range [98]. These medications also protect the myocardium against ischemia. Otherwise, vasodilators such as calcium channel blockers (nicardipine or diltiazem) or nitroglycerin are useful in reducing HTN in an emergent situation. Multiple synergistic medications may be necessary for adequate hemodynamic control [99]. General guidelines stipulate a target systolic blood pressure of 100–120 mm Hg (except in patients presenting with paraplegia, where a systolic range of 120–130 is generally employed) and a heart rate of 60–80 beats per minute [99, 100].

In patients with type A AD, surgical treatment is the gold standard; mortality is 50% within the first 48 h if surgery is not performed [101]. However, with type B AD, medical therapy including analgesia, antihypertensive drugs, and bed rest is performed. However, complicated type B AD, such as descending aortic rupture, uncontrolled pain, and malperfusion of the aortic branch or lower extremities, is an indication for urgent surgery [101]. More recently, thoracic endovascular aortic repair has become an alternative technique to treat complicated type B AD [102].

Lumbar cerebrospinal fluid (CSF) drainage helps prevent spinal cord injury for patients undergoing open or endoscopic thoracic or thoracoabdominal aortic aneurysm and thoracic endovascular aortic repair surgery [103, 104]. When combined with augmentation of the systemic blood pressure, CSF drainage reduces the risk of SCI by increasing the afferent spinal cord blood supply and perfusion pressure by creating a low ambient pressure in the subarachnoid space that surrounds the spinal cord [105], with up to 80% reduction in the relative risk of postoperative deficits in cases of SCI [106–108]. Combinations of lumbar drain and intrathecal papaverine have also been successful in reducing the severity of neurological injury. Prompt detection of spinal cord ischemia by neurological examination and imaging, combined with interventions that increase cord perfusion, is crucial in effectively treating or reversing acute paraplegia or paraparesis and may even reverse cases of delayed onset paraplegia [109].

In our review, 19 patients undergone open surgical repair (12 with type A, 4 with type B, 1 on unknown type of AD), with good recovery in 13/19 (68.4%), residual plegia in 4/19 (21%) patients, and death in 2/19 (10.5%) patients. Only 2 patient undergone endovascular repair with AD type B with favorable outcome in both cases. Six patients undergone CSF drainage; most of them are AD type B, with marked instant recovery in 5/6 (83.3%) patients, but only 1 left with marked residual due to delayed procedure. Twenty-five patients received conservative medical treatment and 3 patients of unknown management and 12 patients received no treatment due to death (Table 4).

The overall spinal cord infarction mortality rate is estimated to be between 9 and 23% [110, 111]. Most deaths occurred early after the SCI. In a study by Robertson et al. [10], older age, severe neurological impairment, and peripheral vascular disease were independently associated with increased mortality. In our review, death occurs in 21/67 (31.3%) patients, 12/21 (57.14%) of defined dissection type were A and 8/21 (38%) were B, 1 of them died of sepsis, 1 of the unidentified type of AD. Older age was the only significant risk factor for mortality (Table 4).

The degree of functional motor and sensory dysfunction of survivors will vary. In a study by Robertson et al. [10] on long-term outcome in 115 SCI patients, among survivors, 37 (42%)

Table 4. Outcome of the patients in relation to sociodemographic criteria of the patients

Parameters	The studied patients *N* = 67			*p* value
	walk *N* = 34	plegic *N* = 11	death *N* = 21	
Age, years				0.63[1]
Mean± SD	57.46±11.78	60.82±10.53	64.0±11.18	0.045[2]
Range	24–85	45–78	46–92	0.37[3]
Sex, *n* (%)				
N/A	2 (8.6)	1 (9.1)	0 (0.0)	0.78[1]
Male	23 (65.7)	6 (54.5)	13 (61.9)	0.29[2]
Female	9 (25.7)	4 (36.4)	8 (38.1)	0.37[3]
Risk factors, *n* (%)				
Smoking	4 (11.7)	1 (9.1)	2 (9.5)	0.96
HTN	16 (47)	4 (36.4)	12 (57.1)	0.50
DM	1 (2.9)	0 (0.0)	2 (9.5)	0.37
Cardiovascular	7 (20.58)	1 (9.1)	4 (19.0)	0.70
Hypothyroidism	0 (0.0)	0 (0.0)	1 (4.8)	0.33
Respiratory	1 (2.9)	2 (18.2)	1 (4.8)	0.17
Arthritis	1 (2.9)	0 (0.0)	1 (4.8)	0.75
Marfan syndrome	0 (0.0)	2 (18.2)	0 (0.0)	0.005
Non–Hodgkin's lymphoma	1 (2.9)	0 (0.0)	0 (0.0)	0.63

SD, standard deviation; N/A, not available; HTN, hypertension; DM, diabetes mellitus.
[1]Comparing walk and plegic.
[2]Comparing walk and death.
[3]Comparing between plegic and death.

were using a wheelchair, 23 (26%) were using a gait aid (cane or walker), and 29 (33%) walked unaided. The results from univariate analysis suggested that severe impairment on initial examination, absence of Babinski sign, presence of sensory level, longitudinally extensive MRI lesions, and MRI lesions with the highest level in the thoracic region were associated with wheelchair and catheter use at a final follow-up. Age, gender, and comorbidities were not associated with functional outcome. But when it was adjusted for time to last follow-up using multivariate logistic regression, severity of impairment was the only variable associated with requiring wheelchair. In a study by Nedeltchev et al. [2], 41% had regained full walking ability, 30% were able to walk with aids, 20% were wheelchair bound, and 9% had died. Severe initial impairment and female sex were independent predictors of unfavorable outcome.

In our review, outcome was good with almost complete recovery in 34/67 (50.7%), while 11/67 (20%) patients ended in wheel chair after prolonged rehabilitation. Tables 4 and 5 demonstrate the outcome in relation to patient sociodemographic criteria, clinical presentation, radiological findings, and therapeutic modality. Age was not an indicator of bad prognosis in our cases, but the initial degree of disability and the lack of early improvement were associated with bad motor outcome. Age, gender, and comorbidities were not associated with functional outcome, but it was noticed that the 2 cases of Marfan syndrome ended up being plegic. There was no association between outcome and radiological findings or location of the lesion. Early diagnosis and appropriate treatment can improve the functional outcome with

Table 5. Outcome of the cases in relation to clinical presentation, radiological findings and therapeutic modality

Parameters	The studied patients *N* = 67			*p* value
	walk *N* = 34	plegic *N* = 11	death *N* = 21	
Bladder, *n* (%)				0.89
N/A	2 (5.88)	0 (0.0)	1 (9.5)	
Yes	8 (23.52)	3 (27.3)	6 (23.8)	
No	24 (70.58)	8 (72.7)	14 (66.7)	
Symmetry, *n* (%)				0.09
Symmetrical	25 (73.5)	10 (90.9)	20 (95.2)	
Asymmetrical	9 (26.5)	1 (9.1)	1 (4.8)	
Affected artery, *n* (%)				0.40
N/A	19 (55.8)	4 (36.4)	12 (57.1)	
Anterior spinal	2 (5.8)	0 (0.0)	5 (23.8)	
Adamkiewicz	6 (17.6)	5 (45.5)	3 (14.3)	
Anterior and posterior spinal	2 (5.8)	0 (0.0)	1 (4.8)	
Femoral arteries	1 (2.9)	1 (9.1)	0 (0.0)	
Iliac arteries	1 (2.9)	0 (0.0)	0 (0.0)	
Sulcal arteries	1 (2.9)	0 (0.0)	0 (0.0)	
Feeding arteries of cauda equine	1 (2.9)	0 (0.0)	0 (0.0)	
Renal and iliac arteries	1 (2.9)	1 (9.1)	0 (0.0)	
Stanford AD type, *n* (%)				0.70
N/A	2 (5.8)	1 (9.1)	1 (4.8)	
A	13 (38.2)	6 (54.5)	12 (57.1)	
B	19 (53)	4 (36.4)	8 (38.1)	
Duration, *n* (%)				0.40
Transient	5 (14.7)	0 (0.0)	2 (9.5)	
Permanent	29 (85.3)	11 (100)	19 (90.5)	
Pain, *n* (%)				0.50
Yes	19 (55.9)	5 (45.5)	8 (38.1)	
No	15 (44.1)	6 (54.5)	13 (61.9)	
Pain location, *n* (%)				0.83
Chest	11 (57.89)	3 (60.0)	4 (50.0)	
Back	5 (26.3)	2 (40.0)	4 (50.0)	
Chest and back	2 (10.5)	0 (0.0)	0 (0.0)	
Chest, back, and abdomen	1 (5.2)	0 (0.0)	0 (0.0)	
MRI findings, *n* (%)				0.34
N/A	21 (61.7)	7 (63.6)	14 (66.7)	
Normal	6 (17.6)	0 (0.0)	2 (9.5)	
Conus	0 (0.0)	1 (9.1)	1 (4.8)	
Thoracic	5 (14.7)	1 (9.1)	4 (19.0)	
Thoracic and conus	1 (2.9)	1 (9.1)	0 (0.0)	
Thoracolumbar	1 (2.9)	0 (0.0)	0 (0.0)	
Thoracolumbar and conus	0 (0.0)	1 (9.1)	0 (0.0)	
Treatment, *n* (%)				
No treatment	2 (5.8)	1 (9.1)	11 (52.4)	0.96[1]
Conservative	12 (35.3)	5 (45.5)	8 (38.1)	0.007[2]
CSF drainage	5 (14.7)	1 (9.1)	0 (0.0)	0.13[3]
Endovascular	2 (5.8)	0 (0.0)	0 (0.0)	
Open surgery	13 (38.2)	4 (36.3)	2 (9.5)	

N/A, not available; CSF, cerebrospinal fluid; MRI, magnetic resonance imaging; AD, aortic dissection.
[1]Comparing walk and plegic.
[2]Comparing walk with death.
[3]Comparing plegic and death.

2/2 (100%) of patients undergone endovascular surgery, 5/6 (83.3) of patients undergone CSF drainage, and 13/19 (68.4%) undergone surgery versus 12/25 (48%) of patients received conservative treatment end up walking.

Conclusion

In our review to cases of SCI due to AD, we found that it is more common in males above 55 years, pain only found in 47.8% of patients, with anterior cord syndrome on top of the clinical presentations, whether permanent or transient, and HTN is the most common risk factor. MRI spine could be normal in up to third of cases specially if done early with thoracic location predominance in positive cases. Surgical or endovascular repair especially for type A and complicated type B should be considered to avoid complications, CSF drainage is a very useful tool in reversing spinal cord ischemia, which is setting of AD specially if done early with favorable outcome. Only the old age is associated with increased risk of mortality. Early diagnosis and appropriate management are crucial for better outcome.

Statement of Ethics

The study was performed in accordance with the Declaration of Helsinki. Written Informed consent to participate was obtained from the patient. The paper is exempt from Ethical Committee Approval as it is a case report not a case study.

Written consent to publish was obtained from study participants for the publication of this case report and any accompanying images.

Conflict of Interest Statement

The authors declare that they have no competing interests.

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Author Contributions

H.E. assisted with literature search, data acquisition, and analysis; and prepared and edited the manuscript. A.I.: prepared and edited the manuscript. A.A., M.A., W.A., and S.F. contributed to literature research and prepared the manuscript. All authors have read and approved the manuscript.

Data Availability Statement

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

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