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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Abstract

Background: Aortic dissection (AD) is a serious condition, that causes transient or permanent neurological problems that include spinal cord ischemia, which occurs when AD extend into the descending aorta resulting in insufficient perfusion of segmental arteries that supplies the spinal cord.

Case Description: We report a 64-year-old Saudi male, hypertensive, dyslipidemic, presented with severe excruciating upper back pain along with 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 on both lower limbs, with urine hesitancy. Computed tomography (CT) showed aortic dissection, Stanford type A and spinal MRI showed hyperintense owl’s eye sign at T5. The patient was diagnosed as anterior spinal artery syndrome secondary to an aortic dissection and referred for aortic surgical repair with good functional outcome.

Conclusion: In our review to cases of SCI due to AD we found that it is more common in males above 55 y, 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 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 be should be considered to avoid complications, CSF drainage is a very useful tool in reversing spinal cord ischemia in setting of AD specially if done early with favourable outcome. Only old age associated with increased risk of mortality. Early diagnosis and appropriate management is crucial for better outcome.

Keywords: Aortic dissection; Spinal cord ischemia; SCI

Abbreviations: AD: Aortic Dissection; HTN: Hypertension; MRI: Magnetic Resonance Imaging; CT: Computed Tomography; CSF: Cerebrospinal Fluid; SCI: Spinal Cord Infarction; ASAS: Anterior Spinal Artery Syndrome; ASCS: Anterior Spinal Cord Syndrome; COPD: Chronic Obstructive Pulmonary Disease; BSS: Brown-Sequard Syndrome; AADS: Aortic Artery Dissections; PSA: Posterior Spinal Artery; CSFD: Lumbar Cerebrospinal Fluid Drainage; TEVAR: Thoracic Endovascular Aortic Repair
Background

Spinal cord infarction (SCI) 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 under diagnosis of SCI [3]. The age of onset ranges from the 1st decade to the 10th decade, with a median age between 50-70 years old [4]. SCI usually presents as anterior spinal artery syndrome (ASAS) 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-Sequard 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 ASCs, 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 are iatrogenic [10]. In this paper, we report a case of anterior spinal cord ischemia caused by aortic artery dissection 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 (COPD). 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 one day duration, 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, nor other cardiac nor neurological symptoms. On examination, he was fully conscious, oriented to time, place and person, with normal speech, memory, as well as cranial nerves. Regarding the upper limbs; motor, sensory examination and coordination were all normal including DTRs (Deep Tendon Reflexes). In the lower limbs; tone was normal, and power was 2 over 5 in the left lower limb and 4 over 5 on the right on MRS scale, DTRs including knee and ankle jerks were brisker on the left side than 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 hypohesthesia. 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, nor bruises. On the second day, after 48h of symptom onset, patient showed partial improvement to grade 4/5 on left and 5/5 on right side but still sensory impairment was the same. His Blood Pressure (BP) at admission was 177/92 mmHg in both arms, 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 examination 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. Additionally, 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 (PSA), 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 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 of type A Stanford classification. The intramural hematoma is seen of high attenuation in the pre-contrast phase with the total filling of the lumen at the post-contrast phase (Figure 1 and 2).

Figure 1: CT axial view without contrast shows dilated ascending and descending aorta, intramural thickening with wall calcification.
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 4x3.6 cm with residual patent lumen = 2.4 x 1.8 cm. An echocardiogram showed moderate to severe left ventricular systolic dysfunction (EF<30%), grade 1 diastolic dysfunction, multi walls 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 intra-medullary signal. It was noticed opposite to the T5 vertebral body, exhibiting bright signal in T2 weighted image and short tau inversion recovery (STIR), the iso-intense signal in T1 weighted image (Figure 3).

It mainly affects the ventral para-median aspects of the cord, with subtle cord expansion. It measures about 2 cm in maximum cranio-caudal length. No significant contrast enhancement could be detected (Figure 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-Sequard syndrome (BSS) nor complete anterior cord syndrome. The following conditions were considered; spinal cord infarction, myelitis, sudden compression from secondary versus deposits, hematomyelia and

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**Figure 2:** CT sagittal view with contrast shows filling defect consistent with thrombus extending from the ascending to descending aorta.

**Figure 3:** (A) MRI T2 sagittal view show small focal linear intra-medullary hypodensity at level of T5. (B) MRI T1 sagittal view show iso-intense spinal cord.

**Figure 4:** MRI sagittal view post-enhanced was unremarkable.
acute demyelination. The presence of significance and continuous interscapular back pain was in the present of normal strangle, suggestion of aortic dissection supports the diagnosis of acute vascular lesion of the spinal cord. During the hospital course, patient was on paracetamol and naproxen. In addition, he was managed with oral amlodipine and this resulted in reduction of BP. The patient referred to a cardiac surgeon where a Bentall surgery was performed for him. The composite graft replacement was done regarding ascending aorta, arch, and coronary artery. Post-surgery outcomes include; significant improvement of the pain, remarkable recovery, and no postoperative complications were detected. After the surgery patient undergone physiotherapy and started to walk independently 1 w after surgery.

**Discussion**

Transient or permanent neurological symptoms at onset of aortic dissection 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 to 32%), ischemic neuropathy (in 4.2 to 24%) and less commonly spinal cord ischemia (in 1-8.9%), also hypoxic encephalopathy and syncope in some patients [10,12-16]. Spinal cord ischemia on the basis of aortic dissection is a rare syndrome and more common with distal aortic dissections. In a study by Sandhu et al., 2018, from 1999 to 2014, they managed 978 aortic artery dissections (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). Neurologic 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, 42 males. Chest pain present in 40 patients, HTN in 22 patients, smoking in 16 patients, genetic syndrome in 5 patients. 24 patients undergone open surgery and 3 patients undergone endovascular surgery. 10 patients died partial recovery in 5 patients and complete recovery in 26 patients [17].

In our search to literature for cases of SCI caused by aortic dissection, we found another 66 cases fulfil our search criteria plus our case. Analysing 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].

**Table 1:** Summary of literature review for cases of spinal cord ischemia secondary to aortic dissection

| N | Citation | Sex | Age | Bladder | Symmetrical/Asymmetrical | Sensory Level | Affected Artery | Stanford AD Type | Chest Pain | Outcome | Treatment | MRI Spine | Risk Factors |
|---|----------|-----|-----|---------|--------------------------|---------------|----------------|-----------------|------------|---------|-----------|-----------|-------------|
| 1 | Waltimo et al., 1980 [18] | M | 52 | None | Symmetrical | No | N/A | A | No pain | Death | Conservative | N/A | HTN |
| 2 | Waltimo et al., 1980 [18] | M | 52 | None | Symmetrical | No | N/A | A | No pain | Death | Conservative | N/A | HTN |
| 3 | Waltimo et al., 1980 [18] | M | 56 | None | Symmetrical | No | N/A | A | No pain | Death | Conservative | N/A | HTN |
| 4 | Gerber et al., 1986 [19] | F | 69 | None | Symmetrical | No | T11 and T5 | N/A | A | Death | None | HTN |
| 5 | Gerber et al., 1986 [19] | M | 78 | None | Symmetrical | No | N/A | A | No | Plegic | None | N/A | HTN |
| 6 | Rosen et al., 1988 [20] | F | 67 | None | Symmetrical | No | N/A | A | No | Death | Conservative | N/A | None |
| Authors               | Sex | Age | Symmetry | Type                          | Location         | Lesion | CT | Outcome | Treatment | Risk Factors | Additional Notes |
|----------------------|-----|-----|----------|-------------------------------|------------------|--------|----|---------|-----------|--------------|------------------|
| Zull et al., 1988    | M   | 67  | None     | Symmetrical                  | None             | Anterior spinal |   A  | no      | Death     | None         | None            |
| Zull et al., 1988    | F   | 63  | None     | Symmetrical                  | None             | Anterior spinal |    A  | yes     | Death     | None         | None            |
| Tanaka et al., 1990  | M   | 66  | None     | Symmetrical                  | Below T9         | Anterior and posterior spinal | B    | no      | Walk      | Conservative  | N/A HTN          |
| Holloway et al., 1993| F   | 92  | No       | Symmetrical                  | None             | Anterior spinal |    A  | no      | Death     | Conservative  | N/A HTN          |
| Krishnamurthy et al.| M   | 80  | No       | Symmetrical                  | N/A              | N/A A B Back and leg pain | Death | None    | Conservative | N/A None     | None            |
| Kellett et al., 1997 | M   | 65  | No       | Symmetrical                  | No               | N/A A B Chest pain | Walk  | Conservative | N/A IHD MI | Emphysema, Angina, HTN, MVP, Smoking |
| Beach et al., 1998   | F   | 58  | None     | Asymmetrical                 | N/A              | Left femoral    | A    | No      | Walk      | Open surgery  | N/A HTN          |
| Lacerda et al., 1998 | F   | 67  | N/A      | Symmetrical                  | N/A              | N/A A B No      | Death | Conservative | N/A N/A   | Small lacunar stroke, HTN |
| Donovan et al., 2000 | F   | 77  | No       | Symmetrical                  | T6-S5            | Adamkiewicz     | A    | No      | Plegic    | Conservative  | N/A chronic arthritis |
| Joo et al., 2000     | F   | 63  | No       | Symmetrical                  | T12-S5           | Adamkiewicz     | N/A  | no      | Walk      | Conservative  | N/A HTN          |
| Inamaou et al., 2000 | M   | 50  | No       | Symmetrical                  | L1-S5            | Adamkiewicz     | A    | no      | Plegic    | Conservative  | N/A HTN          |
| Killen et al., 2000  | M   | 57  | None     | Asymmetrical                 | N/A              | N/A A N/A       | severe upper back pain | Walk      | CSF drainage, intravenous naloxxone drip | N/A HTN, Brain stem stroke |
| Syed & Fiad, 2002    | M   | 32  | No       | Asymmetrical                 | Below knee       | N/A A           | Chest pain | Walk      | Open surgery | N/A Non-Hodgkin's lymphoma |
| Petal et al., 2002   | F   | 65  | Incontinence | Symmetrical                | L2               | N/A A           | Back pain | Death    | None      | Conservative  | N/A Smoking     |
| Ohmi et al., 2003    | NA  | NA  | None     | Symmetrical                  | T9-10            | N/A A           | Severe upper back pain | Plegic    | Conservative | Atrophy at T9-10 NA |

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| Authors                  | Age | Sex | Symmetry | Classification | Spinal Level | Continuation | Treatment       | Outcome          | Other Factors                                      |
|-------------------------|-----|-----|----------|----------------|--------------|--------------|----------------|------------------|---------------------------------------------------|
| Blacker et al., 2003    | F   | 66  | Yes      | Complete ASAS  | N/A          | N/A          | B              | No               | CSF drainage                                   |
| Ogun et al., 2004       | M   | 46  | Sphincter dysfunction | Symmetrical | T6 - S5 | Anterior spinal | N/A | yes | Death        | Conservative                                   |
| Hsu et al., 2004        | M   | 55  | None     | Symmetrical paraplegia | T10 | Adamkiewicz | A | yes | Plegic        | Conservative                                   |
| Hsu et al., 2004        | F   | 64  | None     | Symmetrical paraplegia | None | N/A | A | No | Walk        | Open surgery                                   |
| Hsu et al., 2004        | M   | 67  | None     | Symmetrical paraplegia | T11 | Adamkiewicz | A | No | Death       | Open surgery                                   |
| Chiang et al., 2005     | F   | 74  | None     | Symmetrical | T8 - S5 | Adamkiewicz | A | Yes | Walk        | Open surgery                                   |
| Fujisawa et al., 2006   | N/A | N/A | N/A      | Symmetrical | N/A | N/A | B | N/A | Walk        | Conservative                                   |
| Altuwaijri et al., 2006 | F   | 51  | No       | Symmetrical | No | N/A | B | Chest pain | Walk        | Open aortic fenestration                  |
| Aktas et al., 2008      | M   | 54  | None     | Symmetrical | None | N/A | A | No | Walk        | None                                           |
| Aktas et al., 2008      | M   | 54  | None     | Symmetrical | None | N/A | A | No | Walk        | None                                           |
| Holper et al., 2009     | F   | 63  | Yes      | Anterior cord syndrome | N/A | N/A | B | Back pain | Plegic         | CSF drain                                      |
| Karacostas et al., 2010 | M   | 46  | Retention | Symmetrical | below T7 | Anterior spinal | B | No | Death | None                                           |
| TaHsieh et al., 2011    | M   | 24  | None     | Asymmetrical right LL only | N/A | Right common iliac | B | Abdominal Pain | Walk | Conservative                                   |

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| Authors           | Year | Sex | Age | Symmetry | Area | Spinal Location | Spinal Location | Spinal Location | Spinal Location | Treatment | MR Findings                                                                 |
|-------------------|------|-----|-----|----------|------|-----------------|-----------------|-----------------|-----------------|-------------|-----------------------------------------------------------------------------|
| Zeggeren et al., 2011 | 45   | M   | 62  | Retention| Symmetrical | T9-T12 | Anterior spinal | B               | No              | Walk           | Conservative                                                                 |
| Hayatsu et al., 2011 | 46   | M   | 65  | None    | Symmetrical | T10   | Adamkiewicz | B               | Severe back pain | Walk           | CSF drainage                                                                                                                                 |
| Sui et al., 2012   | 47   | M   | 50  | Retention| Symmetrical | N/A   | Anterior and posterior spinal | A               | No              | Death           | Conservative | long T2 signal at thoracic level |                                                                 |
| Colak et al., 2012 | 48   | M   | 51  | None    | Symmetrical | N    | N/A            | A               | No              | Walk           | Open surgery                                                                 |
| Lynch et al., 2012 | 49   | M   | 54  | Incontinence| Symmetrical | T4-T7 | 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                                                                 |
| Tsiodris et al., 2012 | 50  | N/A | N/A | N/A      | Symmetrical paraplegia | N/A       | Adamkiewicz | A               | No              | N/A          | N/A                                                                                          |
| Hui et al., 2013    | 51   | M   | 70  | No      | Symmetrical | No    | Adamkiewicz | B               | Chest, abdominal and back pain | Walk           | Conservative | T2 hyper-intensity in central aspect of spinal cord extending from T11/12 to L1 | Smoking                                                                 |
| Rabadi et al., 2014 | 52   | M   | 60  | None    | Symmetrical | T6    | N/A            | B               | No pain         | Death           | None          | Normal                                                                                          |
| Ullery et al., 2015 | 53   | F   | 64  | None    | Symmetrical | N/A   | N/A            | A               | Yes             | Walk           | conservative | N/A                                                                                           | HTN, Dyslipidaemia, Aortic aneurysm repair 10y back |
| Fuliang et al., 2015 | 54  | M   | 40  | Yes     | Cauda equine syndrome | N/A    | Feeding arteries of cauda equina | B               | LBP             | Walk           | Endovascular aortic repair                                                                 |
| Yu et al., 2015     | 55   | M   | 56  | None    | Symmetrical | No    | Infrarenal abdominal aorta and bilateral iliac | A               | Severe back pain | Walk           | Open surgery | N/A                                                                                           | None                                                                 |
| Authors               | Gender | Age | Symmetry | Location | Lesion | Clinical Symptom | Therapeutic Modality | Radiological Findings                                                                 |
|-----------------------|--------|-----|----------|----------|--------|------------------|----------------------|---------------------------------------------------------------------------------------|
| Almenara et al., 2016 | M      | 64  | No       | L1 - S5  | 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. |
| Hdiij et al., 2016    | M      | 70  | Retention| Symmetrical| N/A    | A No Death       | None                 | None                                                                   |
| Hughes et al., 2016   | F      | 56  | None     | Asymmetrical| below T10| Anterior spinal  | Open surgery         | N/A COPD, Smoking                                                        |
| Martinez GG et al., 2016 | M      | 72  | None     | Symmetrical| N/A    | B Yes Death      | None                 | N/A HTN, DM                                                            |
| Prakash et al., 2017  | F      | 45  | Incontinence| Symmetrical| T10    | A No Plegic      | Open surgery         | N/A Marfan syndrome                                                  |
| Yildiz et al., 2017   | M      | 74  | None     | Symmetrical| below T12, preserved deep sensation | N/A B Back pain | Death | Conservative  |
| Sekine et al., 2017   | M      | 69  | Yes      | Brown sequard | T10 | Anterior and the posterior spinal | Walk | Conservative  |
| Niclauss et al., 2017 | MMM    | M49 | None     | Symmetrical transient paraplegia | T10 | N/A B sudden onset of chest and back pain | Walk | Repair | N/A No |
| Cheng et al., 2018    | F      | 53  | No       | Symmetrical| T8 - S5| Adamkiewicz | Yes Plegic | Open surgery         | N/A Marfan syndrome, History of tuberculosis                                                                                 |
| Atsuyuki et al., 2018 | F      | 85  | Retention| Symmetrical| T4 - S5| Sulcal artery | B No | Walk with use T-cane | Conservative  |

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| Authors                  | Year | Gender | Age | Symmetry | Extent | Peak Location | Vascular Impairment | Symptoms                  | Management | Radiological Findings                                                                 | Outcome |
|-------------------------|------|--------|-----|----------|--------|---------------|---------------------|-------------------------|-------------|--------------------------------------------------------------------------------------|---------|
| Atsuyuki et al., 2018   | 65   | M      | 68  | Retention| Symmetrical | L2 - S5 | Adamkiewicz | B | No | Plegic | Conservative | T2 high signal intensity in the conus medullaris with restricted diffusion | None |
| Strohm et al., 2018     | 66   | M      | 61  | Yes      | Bilateral lower extremity weakness | T4 | Not identified | B | Severe chest pain | Walk | CSF drainage | Normal | HTN, HLD |
| Tsushima et al., 2019   | 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 T4 - T11 level, | None |
| Quintana et al., 2019   | 68   | M      | 42  | None     | Symmetrical paraplegia | N/ A | Adamkiewicz | A | Yes | Walk | Open surgery | Normal | None |
| Memon et al., 2019      | 69   | F      | 45  | None     | Bilateral lower extremity weakness | T11 | N/ A | A | Severe back pain | Walk | repair | N/A | HTN, Smoking |
| Takeda et al., 2019     | 70   | M      | 62  | None     | Asymmetrical | N/ A | Adamkiewicz | B | No pain | Walk | Repair | Normal | Smoking |
| Sabugueiro et al., 2019 | 71   | F      | 56  | Yes      | Symmetrical [cada equina syndrome] | Saddle anaesthesia | N/ A | A | LBP | Death | None | Abnormal high signal detected within the distal cord and conus | None |
| Kim et al., 2020        | 72   | M      | 62  | None     | Asymmetrical | N/ A | Left renal artery, left intercostal and left lumbar branches | B | Yes | Walk using a | Open surgery | Left asymmetric increased T2 signal intensity of the spinal cord from T11 - L2 level | HTN |
| Nahed et al., 2021      | 73   | M      | 53  | None     | Symmetrical | T4 | N/ A | A | No pain | Walk [transient] | Repair | Normal | None |
| 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

Mean age and gender distribution in AD patients with neurological involvement do not differ from those without [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 number of males doubles the number of females. As for age, it was ranged from 40 to 92 with 68.5% above 55y, with mean age 60 and only one case aged 24y (Table 2). This comes in agreement with mayo clinic who reported that male sex and age from (60-80 years old) consider being one of potential risks of aortic dissection [74]. Owing to the ischemic pathology, the onset of symptoms usually acute and this was the case in all sixty seven cases. Pain is the most common presenting symptom of aortic dissection and could be the sign that direct the physician attention to think about aortic dissection 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 aortic dissection, the frequency of pain-free dissections ranges between 5 and 15% [112,11,75,12] especially in patients with neurological sequelae [11,19,20,75-82]. In a study by Gaul et al., [11] only two thirds of 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 aortic dissection (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, excruciating.
| 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**                                |   |
| Yes                                     | 32 (47.8) |
| No                                      | 35 (52.2) |
| **Pain location**                       | N = 32 |
| Chest                                   | 18 |
| Back                                    | 11 |
| Chest and back                          | 2 |
| Chest, back & abdomen                   | 1 |
| **Duration**                            |   |
| Transient                               | 7 (10.4) |
| Permanent                               | 60 (89.6) |
| **MRI findings**                        |   |
| N/A                                     | 43|
| Normal                                  | 8 |
| Thoracic                                | 10|
| Conus                                   | 2 |
| Thoracic and conus                      | 2 |
| Thoraco-lumber                          | 1 |
| Thoraco-lumber and conus                | 1 |
| **Treatment**                           |   |
| N/A                                     | 1 |
| No treatment                            | 14|
| Conservative                            | 25|
| CSF drainage                            | 6 |
| Endovascular                            | 2 |
| Open surgery                            | 19|
Clinical manifestations of SCI comprise complete spinal cord infarction as well as anterior spinal cord syndrome, Brown-Sequard syndrome, progressive myelopathy or transient spinal cord ischemia [83,84, 79,81]. Pure posterior spinal artery (PSA) infarction in SCI is relatively rare based on previous case studies. [85-87] In a recent study that comprised 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 under recognized 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-Sequard syndrome in 1/67 (1.49%). None of them had isolated posterior cord syndrome (Table 3).

The symptoms almost bilateral symmetrical since both halves of the anterior spinal cord are supplied vascularity from one anterior midline spinal cord. However the anterior spinal cord syndrome with unilateral symptomaticatology 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 total 11/67 (16.49%), being strictly unilateral in only one case. As we mentioned before, the symptoms could be permanent or transient. Seven out of our sixty seven cases (10.4%) had transient symptoms with spontaneous recovery in 4 cases, and conditioned recovery after intervention in 3 cases (one after endovascular fenestration and two after CSF drainage).

If the location of the infraction involves the lateral horns within levels T1-L2 of the spinal cord it will cause autonomic dysfunction, including neurogenic bowel/ bladder which require bladder catheterization. [90, 91] Bladder symptoms in form of hesitancy, retention or incontinence, was found in 17/63 (26.9%) patients, with level between T3 and L1 (Table 3). The most common risk factor for aortic dissection is poorly controlled hypertension (65–75% risk with a history of hypertension [74]. Other risk factors include age, male sex, smoking, pre-existing 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 amphetaamines) [90-92]. In our review, hypertension was on the top of the vascular risk factors being present in 32/62 (51.6%) cases. Out of 62 patients, seven patient (11.29%) were smokers, 3 with DM, 2 with Marfan syndrome (3.2%), 2 had previous stroke, 2 had previous history of angina, 2 had coronary artery grafting, 2 with asthma, one with mitral valve prolapse (1.6%), one with emphysema, one with COPD, one with atrial fibrillation (AF), one with congestive heart failure (CHF), one hypothyroid and one with Non Hodgkins lymphoma (Table 2).

Diagnosis of spinal cord ischemia is done by taking a detailed history, performing physical examination, also neuroimaging studies. MRI of the spine is usually done to confirm the diagnosis, although, in the first 24 hours the results may appear negative. Hyper-intensity in the anterior horns in the T2-weighted image is the hallmark observation. ASAS findings indicate a thin “pencil-like” hyper-intense region that spreads vertically affecting several spinal levels in the sagittal view, two 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 [93,94,89] In our review, out of the 24 cases undergone MRI spine, 8 cases was normal and the other 16 cases showed hyperintense T2 lesions in different location, one delayed MRI show cord atrophy, with the typical owl eye sign found only in 3 cases.

In a study by Hsu et al., 2019, comparing SCI in patient 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 thoraco-lumbar 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 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 aortic dissection 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

| Outcome | 1 | 34 (52.2) | 11 (16.4) | 21 (31.3) |
|---|---|---|---|---|
| N/A | | | | |
| Walk | | | | |
| Plegic | | | | |
| Death | | | | |

N/A: not available, CSF: cerebrospinal fluid
(46.8%) had Adamkiewicz Artery occlusion, followed by 7/32 (2.18%) 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, bilateral iliac arteries were 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 non-invasive imaging study to characterise aortic dissection, with CT used in 61% of cases, echocardiography in 33%, aortography in 4%, and magnetic resonance imaging (MRI) in only 2% [74]. Imaging helps diagnosis and classification of the aortic dissection in order to decide best therapeutic plan. Two classifications are most commonly used for aortic dissection. The DeBakey system is classified into three 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 two types (types A and B) based on 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 aortic dissection in a previous study were 37 (71.2%) Stanford type-A and 15 (28.8%) type-B [97]. In our review, type of aortic dissection 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 aortic dissection is type A or B, medical therapy to control pain and hypertension 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 hypertension 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 mmHg (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 aortic dissection, surgical treatment is the gold standard; mortality is 50% within the first 48 h if surgery is not performed [101]. However with type B aortic dissection, medical therapy including analgesia, antihypertensive drugs, and bed rest is performed. However, complicated type B aortic dissection, 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 (TEVAR) has become an alternative technique to treat complicated type B aortic dissection [102]. Lumbar cerebrospinal fluid drainage (CSFD) helps prevent spinal cord injury for patients undergoing open or endoscopic thoracic or thoracoabdominal aortic aneurysm and thoracic endovascular aortic repair (TAA/TAAA/TEVAR) surgery [103,104]. When combined with augmentation of the systemic blood pressure, CSFD reduces the risk of spinal cord infarction (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 neurologic 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 and 4 with type B and 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 AD type B, with marked instant recovery in 5/6 (83.3%) patients but only one 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).

### Table 4: Outcome of the patients in relation to socio-demographic criteria of the patients.

| Parameters | The studied patients |
|------------|----------------------|
|            | N = 67               |
|            | Walk N = 34          |
|            | Plegic N = 11        |
|            | Death N = 21         |
| Age (years)| 57.46±11.78          |
| Mean ±SD   | 60.82±10.53          |
| Range      | 24 – 85              |
|            | 45 – 78              |
|            | 46 – 92              |
| P value    | 0.63²                |
|            | 0.045²               |
|            | 0.37³                |

Citation: Hosna Elshony, Abdelrahman Idris, Alaa Ahmed, Murouj Almaghrabi, Walaa Ahmed, Shouq Fallatah. Spinal Cord Ischemia Secondary to Aortic Dissection: Case Report with Literature Review for Different Clinical Presentations, Risk Factors, Radiological Findings, Therapeutic Modalities and Outcome. Arch Neurol Neurosci. 10(2): 2021. ANN.MS.ID:000734. DOI: 10.33552/ANN.2021.11.000734.
The overall spinal cord infarction mortality rate is estimated to be between 9-23% [110,111]. Most deaths occurred early after the SCI. In a study by Robertson et al 2012, older age, severe neurologic impairment, and peripheral vascular disease were independently associated with increased mortality [10]. In our review death occur in 21/67 (31.3%) patients, 12/21 (57.14%) of defined dissection type were A and 8/21 (38%) were B, one of them died of sepsis, one of 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., 2012 on long-term outcome in 115 SCI patients, among survivors, 37 (42%) were using a wheelchair, 23 (26%) were using a gait aid (cane or walker), 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 highest level in the thoracic region were associated with wheelchair and catheter use at 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 [10]. In a study by Nedeltchev et al., 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 [2]. 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 Table 4 and 5.

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/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            | Symmetrical          | Asymmetrical |
|                     | 25 (73.5)            | 9 (26.5) | 10 (90.9) | 1 (9.1) | 20 (95.2) | 1 (4.8) |
|                     |                      |        |         |        |         |         | 0.09 |

1: Comparing Walk and plegic, 2: comparing walk and death, 3: comparing between plegic and death, SD: standard deviation, N/A: not available, HTN, hypertension, DM: diabetes mellitus
| Affected artery                              | N/A  | Anterior spinal | Adamkiewicz | Anterior and posterior spinal | Femoral arteries | Iliac arteries | Sulcal arteries | Feeding arteries of cauda equine | Renal and iliac arteries |
|--------------------------------------------|------|-----------------|-------------|-------------------------------|------------------|---------------|----------------|------------------------------|------------------------|
| N/A                                        | 19 (55.8) | 4 (36.4) | 12 (57.1) | 2 (5.8) | 6 (17.6) | 2 (5.8) | 1 (2.9) | 1 (2.9) | 1 (2.9) |
| Anterior spinal                            | 2 (5.8) | 0 (0.0) | 5 (23.8) | 0 (0.0) | 5 (45.5) | 0 (0.0) | 1 (9.1) | 0 (0.0) | 0 (0.0) |
| Adamkiewicz                                | 6 (17.6) | 5 (45.5) | 3 (14.3) | 0 (0.0) | 3 (14.3) | 1 (4.8) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Anterior and posterior spinal              | 2 (5.8) | 0 (0.0) | 1 (4.8) | 0 (0.0) | 1 (4.8) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Femoral arteries                           | 1 (2.9) | 1 (9.1) | 0 (0.0) | 1 (2.9) | 1 (2.9) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Iliac arteries                             | 1 (2.9) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Sulcal arteries                             | 1 (2.9) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Feeding arteries of cauda equine           | 1 (2.9) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Renal and iliac arteries                   | 1 (2.9) | 1 (9.1) | 0 (0.0) | 1 (2.9) | 1 (2.9) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Stanford AD type                           | N/A  | A               | 2 (5.8) | 13 (38.2) | 19 (53) | 4 (36.4) | 8 (38.1) |
| N/A                                        | 2 (5.8) | 1 (9.1) | 1 (4.8) | 13 (38.2) | 19 (53) | 4 (36.4) | 8 (38.1) |
| A                                          | 13 (38.2) | 6 (54.5) | 12 (57.1) | 19 (53) | 4 (36.4) | 8 (38.1) |
| B                                          | 19 (53) | 4 (36.4) | 8 (38.1) | 19 (53) | 4 (36.4) | 8 (38.1) |
| Duration                                   | Transient | 5 (14.7) | 0 (0.0) | 2 (9.5) | 19 (90.5) |
| Permanent                                  | 29 (85.3) | 11 (100) | 19 (90.5) | 0.40 |
| Pain                                       | Yes  | 19 (55.9) | 5 (45.5) | 8 (38.1) |
| No                                         | 15 (44.1) | 6 (54.5) | 13 (61.9) | 0.50 |
| Pain location                              | Chest | 11 (57.89) | 3 (60.0) | 4 (50.0) |
| Back                                       | 5 (26.3) | 2 (40.0) | 4 (50.0) | 0.83 |
| Chest and back                             | 2 (10.5) | 0 (0.0) | 0 (0.0) | 0.83 |
| Chest, back & abdomen                      | 1 (5.2) | 0 (0.0) | 0 (0.0) | 0.83 |
| MRI findings                               | N/A  | Normal         | 0 (0.0) | 6 (17.6) | 0 (0.0) | 14 (66.7) |
| N/A                                        | 21 (61.7) | 7 (63.6) | 14 (66.7) | 0.34 |
| Normal                                     | 6 (17.6) | 0 (0.0) | 2 (9.5) | 0.34 |
| Conus                                      | 0 (0.0) | 1 (9.1) | 1 (4.8) | 0.34 |
| Thoracic                                   | 5 (14.7) | 1 (9.1) | 4 (19.0) | 0.34 |
| Thoracic and conus                         | 1 (2.9) | 1 (9.1) | 0 (0.0) | 0.34 |
| Thoraco-lumbar                             | 1 (2.9) | 0 (0.0) | 0 (0.0) | 0.34 |
| Thoraco-lumbar and conus                   | 0 (0.0) | 1 (9.1) | 0 (0.0) | 0.34 |
| Treatment                                  | No treatment | 2 (5.8) | 1 (9.1) | 11 (52.4) | 0.96 |
| Conservative                               | 12 (35.3) | 5 (45.5) | 8 (38.1) | 0.96 |
| CSF drainage                               | 5 (14.7) | 1 (9.1) | 0 (0.0) | 0.007 |
| Endovascular                               | 2 (5.8) | 0 (0.0) | 0 (0.0) | 0.13 |
| Open surgery                               | 13 (38.2) | 4 (36.3) | 2 (9.5) | 0.13 |

N/A: not available, CSF: cerebrospinal fluid
demonstrate the outcome in relation to patient socio-demographic criteria, clinical presentation, radiological findings and therapeutic modality. Age was not indicator of bad prognosis in our cases but initial degree of disability and 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 2/2 (100%) of patients undergone endovascular surgery, 5/6 (83.3%) of patient undergone CSF drain 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 y, 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 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 be should be considered to avoid complications, CSF drainage is a very useful tool in reversing spinal cord ischemia is setting of AD specially if done early with favourable outcome. Only old age associated with increased risk of mortality. Early diagnosis and appropriate management is crucial for better outcome.

Declarations:
- Ethics approval and consent to participate: The study was performed in accordance with the Declaration of Helsinki. Written informed consent to participate was obtained from the patient.
- Consent for publication: written consent to publish was obtained from study participants.
- Availability of data and material: the datasets used and analyzed during the current study are available from the corresponding author on reasonable request.
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  - I.: manuscript preparation, editing
  - A, M. A, W. A. S. F: literature research, manuscript preparation
  - All authors have read and approved the manuscript

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Conflict of Interest
No conflict of interest.

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