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A review on the pathophysiology and management of Anterior Spinal Artery Syndrome

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Abstract

As an uncommon cause of spinal cord infarction, anterior spinal cord syndrome can manifest with motor paralysis, loss of pain, and temperature sensation distal to the site of the lesion. The main pathogenesis of this syndrome is the disruption of blood flow in the anterior spinal artery. Mortality and morbidity differ with the etiology of the syndrome. So knowing the etiology of blood flow disruption is essential for patient management. This review article highlights the important clinical manifestation of Anterior spinal artery syndrome. Also describes etiology, pathogenesis, diagnosis, prognosis, possible management, and complications.

Keywords:

Anterior spinal artery syndrome, Spinal cord Infarction, Aortic insufficiency, Aortic surgery, Spinal shock, Quadriplegia, Bowel-bladder incontinence

Introduction

Anterior spinal artery syndrome (ASAS) is an infrequent cause of acute ischemic spinal cord infarction. It results from occlusion or hypoperfusion of the anterior spinal artery supplying the anterior two-thirds of the spinal cord. The anterior spinal artery formed by the union of the two branches from the intracranial vertebral arteries at the level of the foramen magnum and descends to the conus medullaris along the anterior median sulcus of the spinal cord. Along its course, ASA supplies the ventral medulla and the anterior two-thirds of the spinal cord. The caliber of the artery is variable with the narrowest segment in the thoracic region, considering a vulnerable zone for ischemia. The paired posterior spinal arteries most frequently arise directly from vertebral arteries and run through the posterolateral sulcus of the spinal cord. Throughout the course, they give off penetrating branches to supply the posterior columns and dorsal gray matter. Anterior and posterior spinal arteries join to form an anastomotic loop at the conus medullaris. The radiculomedullary arteries, which arise from certain segmental vessels, traverse through the intervertebral foramina and run along ventral and dorsal roots to reinforce the longitudinal spinal

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arteries. The spinal cord receives an unevenly distributed blood supply along the different regions. Larger diameter and numerous radicular arteries made the upper cervicothoracic and thoracolumbosacral segments richly vascularized. Where a single anterior radicular artery (artery of Adamkiewicz) between T4 and T8 made the intermediate or mid-thoracic portion poorly vascularized.[1] This fewer anastomosis between ASA and the artery of Adamkiewicz makes ASA effectively an end artery. In contrast, PSA has numerous posterior radicular arteries at this level, providing an extensive collateral system. Therefore, PSA occlusion usually does not cause significant clinical dysfunction. This Regional variation explains the vulnerability of the T4-T8 region of the spinal cord to ischemia, particularly with hypoperfusion due to hypotension. Anterior spinal artery syndrome is a rare cause of acute ischemic spinal cord infarction, which occurs due to complete occlusion or hypoperfusion of the anterior spinal artery. Clinical features include motor paralysis, bowel-bladder incontinence, bilateral loss of pain and temperature sensation, with intact proprioception and sense of vibrations due to the sparing of the posterior column.[2][3][4][5]

Epidemiology

Spinal cord infarction and its subtype anterior spinal artery syndrome is not a typical spinal lesion. Only secondary and incomplete data are available regarding incidence or prevalence. In the USA, one of the extensive studies has shown only 9 of 3784 autopsies demonstrated spinal cord infarction, thus the occurrence at the death of 0.23%. On the other hand, spinal stroke is about 1.2% of total strokes; therefore, the overall annual incidence can be estimated as of 12 in 100,000.

Etiology and pathophysiology

Disrupted flow in the anterior spinal artery may result from a variety of clinical contexts; those can be post-surgical, traumatic, or even idiopathic. Clear concepts of exact etiology and in detail knowledge of the surrounding anatomy are crucial in the management planning of anterior spinal artery syndrome. Since the branches of aorta ultimately supply the anterior spinal artery, the most common causes of anterior spinal cord artery syndrome are insufficiencies within the aorta and its branches. These include aortic dissections, direct trauma to the aorta during surgery or accident, aortic aneurysms, vasculitis and atherosclerosis. Aortic surgery may contribute to anterior spinal artery syndrome directly by causing intraoperative hypotension or embolization. Damage to the spinal column can contribute to anterior spinal cord syndrome by disrupting blood flow through vertebral artery or spinal artery. Examples of such contributors are acute disc herniation from trauma or pathological weakening from vitamin D or calcium deficiency, cervical spondylosis, untreated kyphoscoliosis, atlanto-occipital dislocation. Neoplasia around this area can result in ischemia due to occlusion of the vertebral or spinal artery. Vasculitis, polycythemia, sickle cell disease may cause disruption of blood flow due to hyperviscosity or occlusion of arteries, which may present as an anterior spinal cord artery syndrome. Patent foramen ovale can be complicated with anterior spinal artery syndrome due to the formation and travel of embolus to the anterior spinal artery. Another feared condition is embolism or thrombus formation in the largest feeder vessels (artery of Adamkiewicz). This condition rarely happens during the process of bronchial artery embolization for the management of massive hemoptysis due to any etiology. Another rare condition is cocaine abuse, which causes vasoconstriction, ischemia, and finally causes anterior spinal artery syndrome. Down syndrome can increase the risk of anterior spinal cord artery syndrome in pediatric patients due to its association

with heart defects. Certain infectious diseases (syphilis, tuberculosis, schistosomiasis, and Neisseria meningitidis) are also reported to be the contributor of anterior spinal cord syndrome. [6]

Anterior spinal artery syndrome results from blood flow occlusive or nonocclusive disruption in either an anterior spinal artery or its reinforcing artery (artery of Adamkiewicz) or both resulting spinal cord ischemia in ASA distribution. The most common associations with related pathogenesis are discussed below.

Aortic surgery: thoracic and thoracoabdominal aortic aneurysms surgery is the most common cause of spinal cord infarction. Open and endovascular both approaches are associated with spinal cord ischemia, where risks are lower with an endovascular repair. Spinal cord ischemia usually manifests immediately after thoracic aortic surgery or following an interval of normal neurologic functioning. A delayed presentation even 27 days after surgery has also been reported. Factors responsible for this complication include systemic hypotension, aortic cross-clamping, and occlusion of collateral circulation (e.g., the artery of Adamkiewicz or other intercostal arteries) by ligation, resection, or embolization.[7][8] Risk of spinal cord ischemia following aneurysm repair is higher in patients with advanced age, aortic rupture, prior aortic surgery, extensive aortic disease, postoperative bleeding, longer cross-clamping, intraoperative or postoperative hypotension, sacrificing intercostal vessels and presence of comorbidities like atrial fibrillation, renal insufficiency, cerebrovascular disease.[9][10][11][12][13]

Non-aortic surgeries: Many other non-aortic operations are also associated with spinal cord ischemia. Among these, spine surgery is the commonest. However, hepatectomy, bowel resection, hip and prostate surgery, and many other open procedures also increase the risk. Surgical injury, direct injury, or vasospasm by epidural anesthesia to a radicular feeding artery, intraoperative or perioperative hypotension, are considered as the contributing factors. [14][15][16][17][16]

Aortic dissection: Survivors of the acute catastrophic descending aortic dissection often contend with complications resulting from occlusion of branch arteries that may include radicular artery supplying the spinal cord. The incidence of spinal cord infarction following aortic dissection is 4 percent but with a more unusual presentation. Mid to lower thoracic segments typically involved in this setting. Atherosclerotic disease, chronic hypertension, and Marfan syndrome are the potential risk factors for aortic dissection. 5-15% of cases of aortic dissection are painless, which delays the diagnosis; therefore, it can increase the risk of complications. [18][19][20][21][20][19]

Cervical spondylosis and thoracic disc herniation: Only a few case reports have described ASA syndrome associated with cervical spondylosis and thoracic disc herniation, where most of these were based on clinical manifestation and MRI findings. However, in three cases of ASA syndrome with cervical spondylosis diagnosed with ASA visualizing by spinal angiography images confirmed ASA compression due to a herniated disc may result in ASA syndrome. Patients usually present with acute, painful myelopathy. Diagnosis is based on neuro examination, MRI findings, and ASA visualization for confirmation. Anterior spinal cord decompression and fusion is the mainstay of treatment. The timing of the intervention is the most critical determinant of the neurologic outcome. [22][23][24][23]

Spinal trauma: In the setting of spinal trauma, ASA syndrome can occur due to direct injury to the anterior spinal artery either by disc retropulsion or fragments of bone from vertebral burst fracture and also from traumatic rupture or thrombosis of the anterior spinal artery. An immediate MRI should be obtained to assess the injury site. Emergency decompressive surgery is necessary to regain neurological function.

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Vascular malformation: Most commonly present with progressive, stepwise myelopathy with an abrupt or stroke-like presentation.

Vasculitis: Vasculitis, either due to infection or an autoimmune disease like systemic lupus erythematosus, polyarteritis nodosa, and giant cell arteritis, can be attributed to spinal cord infarction as well as ASA syndrome when involved anterior spinal artery.

Embolic cause: cardiogenic emboli from artificial valves, vegetations, mural thrombus have been reported as the cause of spinal cord infarction. Embolic cause: cardiogenic emboli from artificial valves, vegetations, mural thrombus have been reported as the cause of spinal cord infarction. The atherothrombotic disease is presumed at least in some of these cases having vascular risk factors with the absence of other identifiable etiology. Paradoxical embolism through a patent foramen ovale or other cardiac defects can also be a possible cause of embolic ASA occlusion.

Hypercoagulable conditions: Inherited or acquired hypercoagulability as well as sickle cell disease, polycythemia appeared to be responsible for some cases of spinal cord infarction. By theory, all of these can also occlude the anterior spinal artery resulting in ASA syndrome.

Vertebral artery disease: Vertebral artery atheroma and dissection are also associated with rostral cervical cord Infarction. Depending on the site of involvement, these conditions can selectively cause ASA dysfunction.

Umbilical artery catheters: Umbilical artery catheterization in newborns can rarely occlude the artery of Adamkiewicz, resulting in spinal cord ischemia.

Cocaine-related vasospasm: it has been thought to responsible for spinal cord infarction in a few patients

Occlusion or hypoperfusion of unpair ASA produces abrupt and bilateral clinical findings that correspond to the territory of distribution of the artery. Bilateral lower extremity paralysis is the most common presentation. Initial LMN signs (flaccid paralysis, areflexia) are due to spinal shock. Subsequently, LMN signs are replaced with UMN signs (spasticity and hyperreflexia) over days to weeks as SC regains its function. Moreover, the motor exam demonstrates LMN (alpha and gamma motor neuron cell bodies in the ventral horn) lesion signs in muscles at the level of ischemic cord segments. Whereas UMN (descending tracts in the white matter) lesion signs manifests in muscles innervated by segments below the lesion. In contrast to the motor findings, sensory disturbances remain similar throughout the course. Bilateral loss of pain and temperature sensations (conveyed in the anterolateral system) below the level with the preservation of dorsal column sensation (proprioception, vibration, and discriminative touch) are characteristic for ASA syndrome.

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Histopathology

Cellular events are almost similar in spinal cord infarction and cerebral infarction. The infarct can be either complete or incomplete, depending on the severity of tissue damage. Complete infarction results in the death of every cellular element, where few elements survive in incomplete forms, such as blood vessels, astrocytes, and the damage can also be confined to only neurons. The earliest change of infarction may become evident after 6 hours as a pale and swollen area. Ischemic changes of the neuron or eosinophilic cytoplasm become prominent in the central portion of the infarct. Non-neuronal cells like astrocytes, oligodendroglia, and microglia, along with myelinated axons, disintegrate and contribute to a granular appearance to the neuropil. All of these changes are followed by complex neovascularization, with endothelial hyperplasia and tissue fragmentation. In the subsequent 2 to 3 weeks, phagocytes invade the infarcted area and liquefy the necrotic tissue with eventual cavitation. Finally, the astrocytes proliferate and for the glial scar at the edges of the cavitation.[25]

History and Physical

Spinal infarction due to ASA syndrome is acute, and often apoplectic onset evolves over minutes. This scenario is potentially essential to differentiate the spinal infarction from other confounding diagnoses with an abrupt onset but slower evolution comparison to vascular lesions. The most frequent presentation is sudden severe back pain, which may radiate caudally. Patients may present without pain, but over 80% of spinal infarcts are painful, which made an exciting and unexplained difference from painless cerebral infarction. Besides this pain, almost all patients usually have other neurological deficits stem from the lesion of the spinal cord tracts located in the anterior two thirds, including bilateral weakness, paresthesia, and sensory loss. Loss of sphincter control with difficulties in bowel and bladder evacuation becomes evident within a few hours. Depending on the level of the spinal cord lesion motor weakness may vary from bilateral leg weakness to quadriplegia.

Bowel and bladder complications following ASA syndrome also differ on the course of lesion. Immediately after injury, patients usually experience urinary retention due to loss of micturition from spinal shock. In this phase, incontinence results from the over distended bladder and manifests as continuous dribbling. As the spinal cord regains its function, the micturition reflex becomes independent without interruption from the higher center. This uncontrolled micturition reflex contributes to overactive bladder and the urge incontinence. In this phase, patients usually have intermittent urinary loss rather than continuous dribbling, which results from disrupted inhibitory neurons to the spinal micturition center.

Occlusion or hypoperfusion of unpair ASA produces abrupt and bilateral clinical findings that correspond to the territory of distribution of the artery. The acute phase is characterized by "spinal shock," which manifests as flaccid muscle tone, absent Babinski reflexes, and the loss of all spinal reflexes include micturition reflex, deep tendon reflex, and bulbocavernosus reflex for a while. Bilateral lower extremity flaccid paralysis is the most common finding. Initial lower motor neuron lesion signs (flaccid paralysis and areflexia) are due to spinal shock, which eventually replaced with upper motor neuron lesion (UMNL) signs (spasticity and hyperreflexia) over days to weeks. As the spinal cord regains its function, the motor exam demonstrates LMNL signs in muscles at the level of ischemic cord segments (lesion of ventral horn neuron) and UMNL signs (lesion of corticospinal tract) in muscles innervated by segments below the injury.

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Evaluation

Spinal imaging is the crucial first-line investigation to detect spinal ischemia, identifying etiology, and exclude other possible diagnoses. A spinal MRI is sensitive enough to identify or eliminate intra-axial or extra-axial mass that may or may not compromise spinal blood circulation. Spinal X-ray on the other hand has very little value to detect cord lesion; however, at least can detect burst vertebral fracture. MRI DWI and T2 sequences are reliable and sensitive means of evaluating spinal cord ischemia, thus ASAS. SCI can be detected in DWI as early as 3h after the onset, while it takes around 24 h for T2-weighted imaging to diagnose SCI. T2-weighted imaging demonstrates abnormal T2 hyperintensity and “owl’s eye” appearance in the axial view. Spinal angiography (arteriography) can detect spinal arteriovenous malformation and can confirm ASA compression. However, Spinal MRI has enough sensitivity and reliability to diagnose spinal AVM. Visualization of ASA by CT angiogram (CTA) illustrates spinal cord blood supply in the healthy spinal cord and, to some extent, spinal cord ischemia. CT myelography, MR angiography are modern imaging techniques to diagnose, localize, and classify spinal vascular lesions. The advantages of CT include visualization of the cord and bone anatomy, display of the exact site of ASA flow disruption, where disadvantages are ionizing radiation exposure and administration of nephrotoxic contrast agents.

Laboratory studies in a case of ASAS should be directed to identify vascular risk factors (such as hyperlipidemia), possible underlying systemic diseases, and to exclude differentials. Lab studies may include but not limited to a full blood count, ESR, fasting blood sugar, fasting lipid profile. Serologic tests for syphilis, tuberculosis, schistosomiasis, and Neisseria meningitidis can also be done if those are considered as a potential cause. Rarely hypokalemia or hyperkalemia may present with flaccid quadriparesis and can mimic ASAS thus serum electrolytes are ordered to rule this out.

Besides those; a CSF study can be done to exclude the traumatic etiology of anterior spinal artery syndrome by detecting red blood cells in CSF. A coagulation profile when to determine whether any underlying coagulative disease is responsible as certain autoimmune diseases such as Systemic lupus erythematosus, antiphospholipid antibody syndrome may contribute to the anterior spinal artery syndrome by promoting hypercoagulability. Therefore, these conditions should be screened for better management of the patients. Antinuclear antibody, anticardiolipin antibody, anti-double-stranded DNA antibody, Anti histone antibody assay should give a proper screening and rules in disease as well in case of a positive screen test. Urine/Blood Toxicology report is indicated in rare cases where cocaine abuser presents with anterior spinal artery syndrome. So, when there is suspicion of such a condition, a toxicology report may become essential.

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Treatment / Management

The level and severity of spinal cord ischemia determine the risk of having several systemic as well as neurologic complications. The goal of early intervention is to avoid and ameliorate many of these potentially life-threatening complications. Patients with high thoracic or cervical cord involvement should be managed in an intensive care unit with close monitoring.

Maintaining adequate blood pressure is critical for perfusion to the ischemic, but not infarcted, spinal cord. When thromboembolism is a potential complication, prophylaxis is necessary with low-molecular-weight (LMW) heparin and is considered as the treatment of choice. An acutely increasing respiratory rate and pCO2, declining pO2 and forced vital capacity signals impending respiratory failure. The presence of these signs demands urgent intubation and ventilation support. Atelectasis and pneumonia are possible complications in such a scenario and can be prevented by frequent suctioning and chest physiotherapy. Acute urinary retention can complicate acute spinal cord lesions due to loss of micturition reflex and bladder tone. An immediate indwelling urinary catheter should be placed and be substituted by intermittent catheterization three or four days following the injury. In addition, temperature regulation needs to be monitored as it may be altered due to disrupted autonomic flow.

Long term neurological outcome solely depends on the reversing of the ASA decompression and re-establishment of blood flow. Therefore, the timing between the occlusion and surgical decompression is the most critical factor for long term prognosis. Urgent decompression surgery is the treatment of choice when ASAS results from direct compression to the anterior spinal artery such as spondylosis, thoracic disc herniation, vertebral burst fracture.

Turning the patient in every 1-2 hours, extensor pads, and special mattresses are the essential measures to mitigate the risk of developing pressure sores. An early installment of occupational and physiotherapy is advisable for all patients. Although a minority achieve remarkable functional recovery, a transition to semi-independent living is possible through intense rehabilitation efforts. Long term control of underlying systemic illnesses which are potential causes of ASAS should be addressed, such as sickle cell disease, polycythemia, vasculitis.

Thrombolytic therapy for spinal cord ischemia is still under investigation. Few case reports demonstrate the success of thrombolytic therapy. The potential barrier to thrombolytic therapy in these scenarios is the initial diagnostic uncertainty, delayed diagnosis beyond the treatment window. Concomitant presence of aortic dissection, vascular malformations recent surgery further limits the use of thrombolytic therapy. Systemic corticosteroids have shown improved neurologic outcomes in patients with acute, non-penetrating traumatic spinal cord injury where it has not been studied enough in acute ischemic injury yet. Like acute ischemic brain stroke, systemic corticosteroids are not recommended here. Preclinical studies have shown some benefit with adenosine, prostaglandins, nimodipine, magnesium, naloxone, thiopental sodium, N-methyl-D-aspartate antagonists, but lacking any prospective clinical studies. There is no significant improvement in the clinical outcome that has been observed in a study of SCI patients treated with corticosteroids or anticoagulation, where all patients received antiplatelet therapy for secondary prevention.[26][27]

ASAS can be complicated with persisting spasticity, painful cramps, and spasms & these are treated with oral baclofen, tizanidine, or occasionally diazepam. Intramuscular botulinum and intrathecal baclofen are used for persistent spasticity as well.[28] When an episode results in impotence; oral sildenafil,

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intraurethral or intracavernous injection of alprostadil is useful to attain a successful erection and regain sexual function. After resolving spinal shock, urinary incontinence, and urgency can be a substantial urinary complication. Drugs like Oxybutynin, tolterodine are helpful in the treatment of overactive bladder. If the patient continues to have severe neuropathic and neck pain, then spinal cord stimulation may be another treatment option for the patients in the outpatient setting.[29][30]

Differential Diagnosis

Other causes of Ventral cord syndrome, including mass lesions: have a similar clinical presentation. ASAS is a subtype of ventral cord syndrome. Mass lesions, including spinal cord neoplasms, should have a slow clinical onset, unlike spinal cord ischemia.

Multiple sclerosis: may have similar MRI findings to spinal cord infarctions.

Transverse myelitis: another condition that also may have a similar clinical picture to infarctions; however, the onset is not quite as acute.

Venous congestive myelopathy: although it is a rare entity, it should also be considered as a differential, which most likely presents with prominent and enlarged pial veins and involves the central and peripheral white matter of the spinal cord.

Prognosis

Previous reports showed that spinal cord ischemia following aortic surgery associated with a worse prognosis than that for other infarcts.[14][31][32] Spinal cord ischemia from ASA syndrome is an irreversible tissue injury and generally associated with considerable motor, sensory, and bladder-bowel dysfunction. Patients with high cervical spinal cord involvement and in the setting of aortic rupture or dissection and cardiac arrest have the highest short-term mortality. Most of the survivors recover from functional deficits to some extent. 11-46% can achieve independent gait, while 20-57% remain wheelchair-bounded. Severe impairment at presentation and lack of improvement in the first 24 hours limit the chance of recovery. Other patient-related poor prognostic factors for recovery include female sex and advanced age. Gradual recovery long after hospital discharge has shown in one case series with prolonged follow-up. Patients with residual deficits most often suffered from chronic pain, spasticity, bladder-bowel, and sexual dysfunction. The higher mortality rate in discharged patients is related to the high prevalence of accompanying vascular risk factors.

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Complications

Cardiovascular complications: hemodynamic instability in patients with spinal cord infarction is fairly common either due to the underlying etiology or neurogenic shock. Neurogenic shock characterized by hypotension due to decreased vascular resistance caused by interrupted autonomic pathways in the spinal cord. Bradycardia is usually seen in severe and high cervical (C1 through C5) lesions and may require atropine or external pacing.

Thromboembolism: the risk of deep vein thrombosis and pulmonary embolism is higher due to immobility in a patient with paraparesis after spinal cord infarction.

Respiratory complications: The incidence of pulmonary complications directly correlates with the level of the lesion. The potential complications like respiratory failure, pulmonary edema, pneumonia, and pulmonary embolism highest in higher cervical lesions and thoracic lesions are not rare. The respiratory muscle weakness leads to impaired clearance of secretions, hypoventilation, and atelectasis.

Acute urinary retention: it can complicate acute spinal cord lesions due to loss of micturition reflex and bladder tone.

Loss of temperature control: Patients with a cervical spinal cord involvement may have disrupted autonomic flow with a lack of vasomotor control, which can impair sweating temperature control.

Pressure sores: Denervated skin has a potential risk for pressure necrosis. Pressure sores can complicate an immobilized patient very quickly in particular areas like buttocks and heels.

Discussion

The risk factors of spinal ischemia are almost similar to many common vascular disorders such as stroke, myocardial infarction, and renal failure. Therefore, education of patients who are bearing modifiable risk factors; logically expected to reduce the occurrence of spinal artery thrombosis as well as an anterior spinal artery syndrome. This includes control of diabetes and hypertension, aspirin prophylaxis, and immunomodulatory therapy. Even if any patient develops anterior spinal cord syndrome, they should be evaluated for these risk factors, and proper management should be addressed to minimize these risk factors during the discharge of the patient. So, the chance of development of this condition becomes minimized in the future.

Despite being a rare condition, Anterior spinal artery syndrome may lead to devastating complications, even death of the patient. This serious condition can occur in a wide variety of iatrogenic or non-iatrogenic settings. Any procedure or pathology involving the aorta has the highest possibility of causing anterior spinal artery syndrome. So special attention should be made during thoracic surgery. Patients may present in departments, including emergency or post-operative care following any surgery. Usually, it presents acutely with widespread neurological features and pain. Any patient with acute paraparesis or quadriparesis should be suspected of ASAS. So early evaluation with proper imaging and laboratory techniques must be initiated. The diagnosis of this condition depends on clinical examination and imaging findings. The management of ASAS is nonspecific and varies greatly with the etiology. Besides managing

the acute condition, concurrent risk factors evaluation should be addressed with proper laboratory technique, and confirmed risk factors must be minimized with appropriate medications. The prognosis of anterior spinal artery syndrome depends greatly on the timing of initiation of the treatment. However, even with prompt treatment, residual neurological conditions may persist for a long time. So, suspected patients must be managed by a multidisciplinary team to minimize mortality and morbidity related to this condition. Those patients with ASAS results from acute aortic rupture or dissection, and those associated with high cervical lesions have the highest mortality rate due to concurrent cardiovascular or respiratory complications. These patients should get extra attention with an integrated team approach, including neurocritical care specialists, neurosurgeons, critical care medicine specialists, and neurologists.

Patients who already suffered from ASAS, as well as the caregiver in the family, should be educated about the continuous follow-up, the importance of strict adherence to medications, complications related to this condition, the importance of early initiation, and the continuation of extensive rehabilitation training. Those who become disabled, have the greatest risk of developing embolism, bedsore, and its complications such as sepsis. If any patients develop this condition, they will need emergency care. So, the patient and the caregivers must be educated about these complications, their signs or symptoms, ways to prevent them, and when to call for further assistance. Along with medical and surgical support, extensive psychological support should also be ensured for a majority of ASAS patients.

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