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# Rehabilitative Management of the Anterior Spinal Artery Syndrome (ASAS) Patient

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## Abstract

Anterior Spinal Artery Syndrome (ASAS) is a rare pathology affecting the anterior 2/3 of the spinal column, most commonly developing iatrogenically following aortic surgery. Due to the high degree of permanent disability and dysfunction, optimized medical treatment is vital for disease management. The purpose of this literature review is to provide the first comprehensive management guide for the rehabilitation of ASAS. One of the most important means of ASAS management is immediate reversal of the offending etiology once identified. A patient-centric interdisciplinary approach should be employed in physical rehabilitation of this pathology's sequelae. Recent studies have shown that in addition to medical management, physical therapy is crucial for flaccid paraplegia. Additionally, nerve transfer surgery may prove beneficial in denervated musculature. Management of pain and spasticity should include NSAIDs, tricyclic antidepressants, muscle-relaxants, baclofen, and botulinum toxin. Botulinum toxin may also be employed in cases of neurogenic bladder. Spinal cord stimulation has shown some promise in management of pain and spasticity. To ensure whole-person treatment, providers should also endeavor to address less commonly discussed sequelae such as sexual function and psychological distress. ASAS is a challenging condition to manage long term, but an interdisciplinary team that takes a patient-centered approach to managing the diverse symptomatic manifestations discussed in this review can foster improved quality of life for patients.

**Keywords:** Anterior Spinal Artery Syndrome; Spinal Cord Injury; Neurorehabilitation; Multidisciplinary Rehabilitation

## Introduction

Anterior Spinal Artery Syndrome (ASAS), also called anterior cord syndrome or ventral cord syndrome, is a pathology impacting the anterior two-thirds of the spinal cord. ASAS is caused by occlusion of the anterior spinal artery (ASA) and leads to neurological complications [1]. The purpose of this review is to compile the symptomatic presentations of anterior spinal artery syndrome and act as a reference for the most up to date management methods for rehabilitation of an ASAS patient.

### Presentation

The clinical findings of ASAS are associated with the spinal cord tracts affected-notably the corticospinal and spinothalamic tracts, the ventral horns and their respective lower motor neurons. These varied origins mean a patient with ASAS can present with a variety of clinical manifestations, making it exceptionally difficult to manage. Symptoms may include neuropathic pain,

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autonomic dysfunction, bowel and bladder dysfunction, and sexual dysfunction [1]. These symptoms are often severe in nature. Patients who suffer from ASAS are often left wheelchair bound and may never regain full bladder or bowel control [2]. Proprioception, vibratory sense, and touch sensations are preserved, since blood supply to the dorsal columns remains intact.

### Etiology

There are numerous potential causes of ASAS. Iatrogenic aortic surgery is the most common cause. Risk factors such as hypotension, increased spinal cord pressure, and occlusion of radiculomedullary arteries can increase the likelihood of developing ASAS [2,3]. Other etiologies include thoracic disc herniation, especially those secondary to degenerative spine disease [4].

Additionally, atherosclerotic disease and hypercoagulable diseases, vertebral fractures, severe hypotension from shock, and vasculitis can affect ASA blood flow [5].

As examples, two articles discussed patients who developed ASAS due to interruption of blood flow during surgery. These include a 2010 case study of ASAS as a complication of head and neck surgery and a 2002 report of eight patients who experienced it following infrarenal abdominal aortic surgery [6,7].

### Pathophysiology

The reduction of blood flow through the ASA initiates the neuronal destruction pathway.

This pathway generally begins with microglia and astrocytes becoming activated, leading to disruption of the blood brain barrier [8]. Subsequent failure of ionic pumps and cellular depolarization due to the lack of blood flow causes glutamate and calcium buildup in the cells. This, in turn, interrupts mitochondrial activity of the cells of the spinal cord and results in neuronal cell death. Finally, this cell death yields the symptomatic presentation of ASAS [9].

### Diagnosis

Diagnosis of ASAS can be made with the use of MRI imaging. A major MRI finding on T2-weighted imaging associated with ASAS is hyperintensity present at the anterior horns, often referred to as "Owl's Eyes" for their appearance when viewed in the axial plane [10]. On a sagittal view of a T2-weighted image, a fine "pencil-like" hyperintensity is often seen, usually running down the spinal cord. Conversely, on T1-weighted imaging, ASAS typically presents with hypointensity at the area of the ischemia [11]. If necessary, magnetic resonance imaging angiography can further confirm the pathology. Analysis of blood and urine samples, along with a lumbar puncture for CSF collection, can rule out differential diagnoses of infections or drug abuse that may yield a similar clinical presentation [10].

## Management of the ASAS Patient

Immediate treatment of the underlying cause of the anterior spinal artery syndrome is first-line for preventing long term neurological complications [12]. For example, in the case of iatrogenic endovascular aortic surgery, the healthcare team could augment blood pressure and use a lumbar drain to reduce cerebrospinal fluid pressure. The appropriate intervention often fails to result in full neurological recovery. In a review of 20 childhood cases, only 7 were mobile, but all requiring assistance to walk [13]. In a series of 44 adult patients, at follow-up of varying durations, only 11% were walking independently and 27% were walking with support [14]. Therefore, intense rehabilitation efforts are necessary. Rehabilitation should be interdisciplinary and include physical medicine & rehabilitation specialists; in fact, it has been shown that the involvement of physiatrists leads to better prognoses for patients with acute traumatic spinal cord injuries [15]. The challenge of the rehabilitation lies in the breadth of symptoms a patient with ASAS can present with, depending on the location and severity of the occlusion.

### Paralysis

Dysfunction of the lower motor neurons (LMNs) in the ventral horns and upper motor neurons (UMNs) in the corticospinal tract due to ischemia in ASAS can result in paralysis [16]. LMN lesioning presents with flaccid paralysis or weakness, hyporeflexia, hypotonic musculature, fasciculations and wasting atrophy of musculature in a region innervated by a LMN from a single spinal cord level [17]. UMN lesioning presents with spastic paralysis or weakness with usually more voluntary movement than LMN, hyperreflexia, clonus and disuse atrophy occurring in the regions below the site of spinal cord lesion [18].

Electromyogram and nerve conduction studies, voluntary muscle control loss, muscular atrophy, hyporeflexic deep tendon reflexes at the level of the ASAS, and even nerve biopsies, can all help confirm that the ASAS has affected LMNs [19]. After confirming LMN lesioning, check for combined UMN lesioning by looking for hyperreflexic deep tendon reflexes anywhere below the level of the lesion and a positive Babinski's Sign. Assessing signs of UMN lesioning in a limb already affected by severe LMN deficit is appreciably difficult [20].

Loss of the ability to contract or control musculature results in dramatic lifestyle changes for the patient, necessitating interdisciplinary orchestration of physical therapy, occupational therapy and possibly assistive device fitting [21].

Personalized physical therapy and exercise programs for the ASAS patient experiencing flaccid paraplegia are a vital part of rehabilitation [22]. Even if the LMN lesioned musculature is never expected to regain strength, the patient

must strengthen musculature adjacent to the affected areas in order to facilitate new means of ambulation. Aside from traditional exercise, newer interventions such as transcranial magnetic stimulation, functional electrical stimulation and robot-assisted treadmill training have all effectively rehabilitated function in patients [23,24]. Consistent exercise has even been shown to promote neuroprotection and regeneration in a post-spinal cord injury (SCI) patient [25]. Lastly, structured and planned exercise is essential to prevent health complications and metabolic syndromes secondary to neurogenic obesity [26,27]. The term applied to the weight gain common among recent paraplegics due to reduced energy expenditure and increased adiposity near viscera, skeletal muscle, and bone marrow [28,29].

Occupational therapy should be utilized to help maximize independence and quality of life. Some tasks to be practiced may include dressing, grooming, bathing, bed mobility, balance if ambulatory, and wheelchair maneuverability if paraplegic or tetraplegic [30].

Depending upon which spinal cord level the ASAS occurred, the patient may be paraplegic, necessitating transition to wheelchair. Performing some sort of physical exercise designed for wheelchair users is crucial to improving function, depression and general health, with no notable downsides [31-33]. One trial even showed that weight-bearing exercises improved patients' ability to complete activities of daily life and safely transfer into and out of wheelchairs [34].

Although rarer, a lesion in the cervical spinal region may cause the patient to become tetraplegic, necessitating more complex and expensive assistive devices. Some newer and novel devices include those controlled by brain-machine interface or motion of the tongue [35-37].

Tendon and nerve transfer procedures offer innovative surgical rehabilitative opportunities. Both these transfer surgeries are well-established and proven to restore notable levels of function [38,39]. However, despite proven improved functional independence and quality of life, only a few of the estimated 65-75% of patients with cervical SCI that would benefit from these procedures undergo them [40]. Nerve transfer surgery is a more recent development in which, instead of transferring a new tendon to the denervated muscle, the disconnected musculature is directly reanimated from an intact spinal cord level. Whereas tendon transfers can be done later in the rehabilitation process, nerve transfer must be done before the targeted muscle sustains significant atrophy [41]. Studies showed that this early nerve transfer surgery, especially when combined with tendon transfers, maximizes functional benefits in tetraplegic patients [42].

## Pain and Spasticity

Pain management of a spinal cord injury (SCI) as severe as ASAS is a complicated matter. Pain can be neuropathic or nociceptive, and possibly visceral or referred. Since UMNs are damaged, muscle spasticity may accompany this pain and be a major point of discomfort for the patient.

Noninvasive, low risk techniques to address musculoskeletal pain, increase muscle tone and limit spasticity include physical therapy and osteopathic manipulative treatment [43]. If the patient wants to explore alternative pain management routes, such as acupuncture or psychological treatments, there is typically little to no risk of complications [44].

However, pharmacological intervention and other more interventional pain management methods tend to be necessary. For nociceptive pain and inflammation, non-steroidal anti-inflammatory drugs (NSAIDs) are an effective, convenient and affordable option. They provide relief but have a marked side effect profile, most notably for stomach ulcers, especially when used chronically. In addition, NSAIDs may be contraindicated for some patients, such as those with aspirin hypersensitivity or renal disease [45-47].

For more severe and resistant musculoskeletal pain and even acute neuropathic pain, opiate treatment can be considered. However, such treatment requires great caution due to its addictive potential and side effects. Additionally, although highly effective as an analgesic, some studies have shown that morphine can undesirably attenuate locomotor recovery in the post-SCI patient [48,49].

For neuropathic pain, several drugs typically used for other conditions have proven effective. One such drug is gabapentin, traditionally used an anti-seizure medication. Others include antidepressants such as venlafaxine, a serotonin norepinephrine reuptake inhibitor (SNRI), and amitriptyline, a tricyclic antidepressant (TCA) [50,51]. These drug classes have shown the greatest efficacy among non-opioid pharmacological interventions [52].

To help with spasticity due to corticospinal tract and upper motor neuron lesioning, muscle relaxants and anti-spasticity drugs include baclofen, diazepam, tizanidine, dantrolene, and clonazepam [53-57] In cases of severe spasticity, baclofen may be delivered continuously via intrathecal pumps [58-60]. Chemodenervation through the administration of botulinum neurotoxin is also highly effective in the treatment of severe spasticity [61,62].

Several minimally invasive techniques also offer the potential for pain relief. Spinal cord stimulation (SCS) involves an implanted device that transmits low levels of electrical current directly into the spinal cord [63]. This technique has proven effective in reducing chronic pain and spasticity, thereby reducing the need for dangerous pain medication [64]. A recently published report on a trial of a transcutaneous iteration of the traditional epidural implant showed promising results, allowing for similar benefits with

even less invasiveness [65]. Another option is radiofrequency neurotomy, a type of radiofrequency ablation. This procedure uses alternating current to ablate select nerve endings with heat in order to provide longer-lasting pain relief for the patient [66].

## Neurogenic Bladder

A notable complication of ASAS is neurogenic bladder, a lower urinary tract dysfunction in which patients lack bladder control. In spinal shock, patients generally experience urinary retention but can later develop neurogenic bladder. Neurogenic bladder and associated incontinence can pose a significant detriment to patients' quality of life [67]. With a spinal cord injury such as ASAS, the neurogenic bladder typically manifests as detrusor overactivity and detrusor-sphincter dyssynergia. Detrusor overactivity symptoms present with urinary urgency, frequency, nocturia, and incontinence whereas detrusor-sphincter dyssynergia presents with urinary hesitancy, an interrupted urinary stream, sensation of incomplete bladder emptying and double voiding [68,69]. A neurogenic bladder can also lead to increased urinary tract infections (UTIs) and can cause upper urinary tract damage in the forms of vesicoureteral reflux, hydronephrosis and renal impairment [70].

The primary evaluation tool for a neurogenic bladder is history and physical examination along with basic urinalysis. The provider can also use ultrasound to determine post-void residual volume (PVR), which helps establish whether there is a voiding dysfunction. Another noninvasive test is uroflowmetry to assess PVR and other voiding functions. More invasive testing options such as combined cytometry and pressure-flow study are available, but there is debate over their usage and effectiveness [71].

Proper management of a neurogenic bladder is crucial in maintaining quality of life, preventing UTIs and preserving upper urinary tract function. For patients with urinary storage dysfunction, antimuscarinics to induce detrusor relaxation and lower intravesical pressures are a primary treatment option. Another approved agent is mirabegron, a beta-3-adrenergic agonist.

Further, injections of onabotulinumtoxinA into the bladder wall can cause relaxation of the detrusor muscle and has been shown to be efficacious, safe and well tolerated [72]. Neuromodulation is another promising treatment modality for improving overactive bladder symptoms by electrically stimulating the tibial or other lower extremity nerve [73].

Management is quite different for patients with a voiding dysfunction, mainly consisting of catheterization. The primary forms of long-term catheterization include transurethral indwelling catheters, intermittent self-catheterization, suprapubic catheterization and condom catheters. The type used is largely based on patient preference [74]. Some patients are comfortable inserting an intermittent catheter and able to maintain the cleanliness to avoid UTIs; others may prefer a more permanent alternative such as a suprapubic catheter [75]. Many physicians and rehabilitation teams endorse intermittent self-catheterization for their patients. However, conversion to indwelling catheters may be recommended in late stage rehabilitation (>10 years) due to additional comorbidities and difficulties in self-care [74]. With regard to outcomes and patient reported data, studies suggest that an indwelling catheter and surgical options provide the best symptom relief for a neurogenic bladder as well as a higher quality of life [76]. While there is evidence to suggest that indwelling catheters or surgical options are ideal, it is important to consider the cost of the surgical insertion procedure and the maintenance it will require. Catheterization is a major life change and providers should be aware of each patient's concerns.

## Neurogenic Bowel

A neurogenic bowel is a significant complication for ASAS patients that can lead to complications in patient autonomy, dignity, and infection. In a neurogenic bowel due to ASAS, high pressure and lost coordination in rectal contractions result in a failure to contract the anal sphincter [77]. This form of bowel dysfunction is an upper motor neuron dysfunction. The primary goal of neurogenic bowel management is to improve quality of life by reducing time spent on defecation, decreasing incontinence, and increasing functional independence [78].

The first consideration is to determine the level of assistance a patient needs. Studies show that higher spinal cord injuries correlate with an increased level of assistance [79]. In one study, those who were fully dependent at discharge tended to remain fully dependent, while those who were less dependent generally transitioned to increased functional independence, suggesting that correct management can promote improved quality of life [79].

Diet constitutes one of the most important management tools. Researchers have proposed the Mediterranean diet, consisting primarily of dairy, whole grains, fruits, vegetables, and lean meat, as optimal diet for bowel management. The diet also limits many spices, caffeine products and processed foods to inhibit bowel inflammation [80]. Exercise is also recommended, as it promotes a healthy bowel. The motive behind these lifestyle changes is to promote a bowel schedule that allows the patient to plan their day accordingly [80].

Aside from lifestyle changes, one study focusing on bowel routines found that most subjects with neurogenic bowel used enemas as their primary management tool [81]. Enemas, also known as transanal irrigation, are a simple and easily teachable method to ensure the emptying of the rectum and descending colon [78]. Furthermore, patients

who regularly incorporate enemas into their routines report reduced complaints of constipation, decreased incontinence and a higher quality of life compared to non-enema users [78].

Overall, the recommendations for a neurogenic bowel include a well-balanced diet and exercise routine, the use of enemas and caregiver assistance if needed. Current studies with non-invasive spinal cord stimulation for neurogenic bowel are also showing promise as an effective method of neurogenic bowel management [73].

## Neurogenic Sexual Dysfunction

Although the notion of sexual encounters post-SCI may seem incongruous, patients may consider it a key aspect of their quality of life. Arousal, ejaculation, and orgasm are significantly affected post ASAS injury in both male and female patients.

In one study of patients with female genitalia, Merghati-Khoei et al. reported as few as 3.2% of patients with a spinal cord injury revealed sexual dysfunction; however, they also reported a lower quality of sexual life compared to controls [82]. Contrarily, another study by Hajiaghababaei et al. revealed that within a population of Iranian women suffering from SCI, around 88% of SCI patients showed at least a one metric of sexual dysfunction, so further studies are necessary [83]. Merghati-Khoei et al. identified focusing on the psychological aspect of sexual interactions as the most important management tool. A spinal cord injury may negatively affect a female's sexual sensuality, which can lead to low self-confidence and interfere with emotional intimacy. The management recommendations include focusing on longer foreplay, powerful stimulation, and positive emotional feedback during sexual experiences. Combining this with attention to personal values, attitudes and expectations regarding sexuality after spinal cord injury may help to create a more intimate and pleasurable experience [82].

Male patients frequently experience sexual dysfunction post-spinal cord injury. Erectile dysfunction, orgasmic limitations, and infrequent sex are some risk factors that contribute to this presentation while a fixed partner, ejaculation, and masturbation have been shown to be protective in men's sexual health [84]. For erectile dysfunction, phosphodiesterase-5 inhibitors are the primary treatment option, including sildenafil, vardenafil, and tadalafil. One study that focused on the efficacy of sildenafil found that spinal cord injury patients who received the drug had a statistically significantly greater ability to achieve and maintain an erection than those who were given a placebo [85]. Additionally, there was a higher percentage of sexual intercourse success. However, it is important to note common side effects include headache and UTIs, [85] which may be a contraindication for those who also have a neurogenic bladder. Second line therapies include intracavernosal injections, vacuum erection devices and erectogenic urethral suppositories [86]. Surgical interventions with penile prosthesis remains an option but is seldom used.

Fertility is generally not a great concern for women, although they may experience amenorrhea that typically resolves within six months. However, men are much more likely to encounter issues with fertility and ejaculatory dysfunction. Studies have shown that semen volume and quality are affected, with semen motility experiencing the largest change [87]. Male fertility treatments include penile vibratory stimulation (PVS) and electroejaculation (EEJ), which stimulate ejaculation to enable sperm retrieval for assisted reproductive use. PVS is the current first line treatment and has been shown to be highly effective in patients with spinal cord injury [87].

The most important idea to convey to patients is that sexual dysfunction is a long and individualized process, requiring time and commitment from the patient and any sexual partners.

## Autonomic Dysreflexia

A cervical or high thoracic level ASAS can interrupt communication of sympathetic branches of the autonomic nervous system, severely impacting viscera function. In fact, up to 90% of cervical or upper-thoracic spinal cord injuries leave patients susceptible to autonomic dysreflexia (AD) [88]. Unusual sympathetic signaling can yield hypersensitive peripheral vasculature and overall dysfunctional hemodynamics [89]. Abnormal heart rhythms should be especially monitored, as abnormal sympathetic reflexes can cause severe bradycardia, tachycardia and arrhythmia [90]. Uncontrolled sweating, anxiety or visual dysfunction may also be present. This autonomic nervous system function can affect bladder and bowel maintenance, renal function and sexual capability as well [90].

These symptoms of autonomic dysreflexia (AD) —also called autonomic hyperreflexia, sympathetic hyperreflexia or hypertensive autonomic crisis— are triggered by a noxious stimulus occurring anywhere below the spinal cord level of the ASAS injury. As the sympathetic signal tries to travel up the spinal cord, the lesioned level causes a disruption, leading to improper signal messaging. The noxious stimuli are most often bladder related, such as an overfull bladder, a UTI, bladder spasms, or kidney stones. Other less common stimuli include, but are not limited to, overfull bowel, ulcers, sexual activity, pressure ulcers and gallstones [91].

Autonomic dysreflexia is a serious condition, potentially leading to cardiac arrest or hypertensive encephalopathy, but removal of the stimulus resolves most issues. Thus, in an ASAS patient suffering from AD, most preventative measures include proper neurogenic bladder management. In all cases,

the patient and physician should determine the causative stimulus and work to prevent their future occurrence [88,91].

## Diaphragmatic Palsy

Diaphragmatic palsy is a loss of diaphragm muscular strength that can be caused by either muscular weakness or loss of innervation [92]. The symptoms can range from asymptomatic to dyspnea, depending on the cause [92]. In the context of ASAS, injury at a high cervical level can cause damage to the phrenic nerve, resulting in diaphragmatic palsy and respiratory failure that can leave patients reliant on ventilators [93].

Truncal and abdominal support such as girdles and back braces can improve diaphragmatic function in individuals with less severe diaphragmatic palsy [94]. A study by Hart et. al found that their study cohort, 10 patients with spinal cord injuries who did not require ventilation, achieved increased forced vital capacity, inspiratory capacity, and diaphragmatic strength when wearing a customized girdle [94].

Another treatment for diaphragmatic palsy, electrophrenic/ diaphragmatic pacing, requires the phrenic nerve to be intact [95]. In this treatment, a pacemaker installed near the phrenic nerve stimulates the nerve and causes muscles to attract, allowing patients to breathe without a ventilator [96]. This technique is highly beneficial for patients as it greatly improves quality of life, and may enable patients to be weaned off of ventilation [97]. However, its potential risks include damage to the phrenic nerve, lung injury, esophageal injury and vascular injury [96].

Reinnervating the diaphragm with nerve transfers is a novel treatment used in patients who are not candidates for electrophrenic pacing due to lack of phrenic nerve integrity, a frequent result of spinal cord injury in the nerve roots of C3-5 [97]. The treatment involves transplantation of nerves, often taken from the spinal accessory, thoracodorsal or intercostal nerve, as well as implantation of a pacemaker in order to regulate the diaphragm [97]. Kaufman et al. reported that 62% of the patients in their retrospective cohort (n=14) were able to be weaned off ventilators.96 In a study by Krieger et al., they achieved a 100% success rate in their cohort (n=8) [98]. More recently, a 5-year-old female successfully underwent thoracoscopic intercostal to phrenic nerve transfer for diaphragmatic reanimation in 2021 [99].

## Pressure Ulcers

Pressure ulcers are areas of tissue damage that often result from prolonged pressure to one area [100]. They are common in the hospital setting, particularly among those who are immobile for extended periods of time. Individuals with spinal cord injuries are highly susceptible to pressure ulcers, with up to 95% lifetime risk [100]. Pressure ulcers have been shown to poorly impact rehabilitation, increase the risk of future hospitalizations and raise the risk of patient death [100].

The best treatment for pressure ulcers is prevention. The most common method for preventing pressure ulcers in immobile patients is frequent bed/wheelchair repositioning to avoid the prolonged pressure necessary for ulcers to form [101] Previous studies have identified specific areas where pressure ulcers are more likely to develop, such as the trochanters and sacrum.

Special care must be given to avoid their formation in these areas [101]. Another method of pressure ulcer prevention is pressure relief maneuvers, simple weight shifts to promote blood circulation, which can be performed seated or supine. The current recommendation for pressure relief in immobile patients is 30 seconds of relief every 30 minutes [101]. Additionally, some studies have demonstrated that certain cushioning materials can reduce the risk of pressure ulcers, for example bead filled, water filled and high specification foam mattresses [102] Prophylactic dressings and fatty acid creams are believed to reduce the risk of pressure ulcer formation. Proper nutrition may also play a role in preventing skin breakdown [102]. Once a pressure ulcer has formed it can be difficult to treat and often takes considerable time to heal. The most important factor in healing is alleviating pressure from the ulcer.

Cleansing with saline and debriding dead tissue are often advantageous to the healing process [102]. Infection control is highly important, as pressure ulcers raise the risk of osteomyelitis and other infections. Topical treatments such as platelet derived growth factor, phenytoin and platelet-rich plasma may speed healing [102]. Some evidence exists that both cellular and acellular matrices may promote healing, but this has not yet been fully confirmed. Finally, ulcers can be surgically repaired with skin flaps when immediate wound closure is necessary or desired, with the major risk being dehiscence [102].

## Psychiatric

Anterior Spinal Artery Syndrome has severe symptoms that can suddenly and drastically affect the quality of life of an individual. Patients with spinal cord injury are at higher risk for depression, anxiety and reduced quality of life [103-105]. The trauma of spinal cord injury and the reduction of functional independence can lead to mental health difficulty, so psychiatry should be consulted. Some propose that unmanaged depression may even lead to secondary worsening of physical SCI symptoms [106]. It is worth noting that if the patient needs to be medicated for neuropathic pain, treatments that serve a dual function as treatments for pain and depression, such as venlafaxine or amitriptyline, may be worth consideration [50,51].

## Conclusion

In the immediate onset of Anterior Spinal Artery Syndrome, the time between arterial occlusion and surgical decompression is a key factor in prognosis. Rapid restoration of appropriate blood flow is key to minimizing the long term complications due to the ischemia [10]. However, it is important to recognize that while some patients can experience remarkable neurological recovery, the majority of patients need extensive rehabilitation in order to achieve functional independence and satisfactory quality of life [13,14]. Vital rehabilitative considerations include helping the patient adjust to a paraplegic lifestyle, manage pain and spasticity, develop protocols to address neurogenic bladder, bowel and sexual dysfunctions, learn to prevent autonomic dysreflexia episodes, and seek psychiatric assistance to maintain mental health. Even before rehabilitation, acute hospital management of the patient should include close monitoring of motor neuron impairment of the phrenic nerve and preventative measures to avoid pressure ulcers.

Key factors to successful rehabilitation include interdisciplinary support and a patient-centered focus. The surgical and intensivist team transitions care to the rehab led by physiatry, which includes working with neurology, psychiatry, physical and occupational therapy, at-home care providers, and perhaps others, all of which must effectively collaborate and communicate to ensure patients' needs are met[15,107]. Ultimately, ASAS is a challenging condition to manage long term, but an interdisciplinary team that takes a patient-centered approach to managing the diverse symptomatic manifestations discussed in this review can foster improved quality of life for patients.

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## Statement of Authorship

JD was responsible for the study's conception and design, writing primarily sections about paralysis, pain & spasticity, autonomic dysreflexia, psychiatry, and the conclusion, and reviewing and editing the entirety of the manuscript. DP wrote primarily about pathophysiology, presentation, and diagnosis, and assisted in edits. BC wrote primarily about neurogenic bladder, bowel, and sexual dysfunction, and assisted in edits. AP wrote primarily about the introduction and etiology and assisted in edits. JR wrote primarily about diaphragmatic palsy, pressure ulcers, and assisted in edits. BG wrote the abstract and assisted with reviewing and editing the manuscript. GS was the PI and helped oversee the writing process and review the manuscript.

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