**Charcot spinal arthropathy**

**ABSTRACT**

Charcot spinal arthropathy (CSA) is a rare progressive disorder of vertebral joint degeneration that occurs in the setting of any condition characterized by decreased afferent innervation with loss of deep pain and proprioceptive sensation in the vertebral column. While surgical circumferential arthrodesis remains the most effective treatment modality, it is associated with multiple complications, including hardware construct failure. This manuscript represents an up-to-date narrative review of the treatment of CSA, its associated complications, and complication prevention.

**Keywords:** Bone morphogenetic protein, Charcot spinal arthropathy, circumferential arthrodesis, spinal cord injury

**INTRODUCTION**

Charcot spinal arthropathy (CSA), also commonly known as spinal neuroarthropathy or neuropathic spinal arthropathy, is a rare progressive disorder of vertebral joint degeneration that occurs in the setting of any condition characterized by decreased afferent innervation, involving loss of deep pain and proprioceptive sensation in the vertebral column. Neuropathic arthropathy was first described by Jean-Martin Charcot in 1868.[1] The first case of CSA was reported in 1884 by Kronig in a patient with tabes dorsalis secondary to tertiary syphilis.[2] Historically, CSA was most commonly reported in the setting of tertiary syphilis. Nowadays, as a consequence of low incidence of syphilis due to improved antibiotic therapy, CSA presents almost exclusively in patients who have suffered traumatic spinal cord injury (SCI).[3‑7] CSA secondary to traumatic SCI occurs 17 years after injury on average.[8] Less commonly, CSA may present secondary to syringomyelia, meningocele, myelomeningocele, diabetes mellitus, peripheral neuropathies, anesthetic leprosy, congenital analgesia, Parkinson’s disease, arachnoiditis, transverse myelitis, and others.[8‑12] In cases of CSA involving gross spinal instability and absence of medical comorbidities that would otherwise cause contraindication, surgery has become the preferred treatment modality. While posterior-only reconstruction has been indicated in mild cases with minimal bony involvement, the majority of CSA today is treated by circumferential arthrodesis. In addition to improvements in surgical technique, the application of a multimodal treatment model, including the use of bone morphogenetic protein (BMP), has reduced the rates of treatment failure.[13] We present a comprehensive narrative review describing the treatments for CSA, associated complications, and their prevention, with an emphasis on circumferential arthrodesis involving both an anterior and posterior-column construct.

**PATHOPHYSIOLOGY/DIAGNOSTICS**

While the etiology of CSA remains disputed, two theories have gained prominence. The neurotraumatic theory hypothesizes that abnormal motion and spinal instability secondary to loss of deep pain and proprioceptive sensation in the posttraumatic SCI vertebral column results in

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repetitive microtrauma that leads to inflammation of the subchondral bone and articular cartilage.\textsuperscript{[9,14]} Ultimately, the chronic inflammation results in facet joint destruction, intervertebral disc degeneration, progressive deformity, and spinal instability [Figure 1].\textsuperscript{[13]} Conversely, the neurovascular theory describes the onset of hypervascular regions in the subchondral bone due to underlying systemic pathologies such as diabetic neuropathology or autonomic dysfunction. This leads to increased osteoclastic resorption, causing microfractures that ultimately result in facet joint destruction and gross instability.\textsuperscript{[8]}

CSA can be divided into atrophic and hypertrophic subcategories, with the atrophic form presenting predominantly with bone resorption, and the hypertrophic form presenting with extensive bone formation and osteophytosis.\textsuperscript{[6]}

To this day, CSA remains a difficult pathology to diagnose due to the nonspecific characteristics of its symptoms, as well as its prolonged progressive destructive nature. The thoracolumbar spine is typically the most commonly affected region.\textsuperscript{[14]} Bone erosion, osteophytosis, stenosis of the intervertebral space, and presence of paravertebral masses are all common to CSA but are nonspecific. Spasticity, hyperreflexia, asthenia, and leg pain may also be present.\textsuperscript{[15]} Loss of lower extremity spasticity is thought to be suggestive of CSA secondary to SCI.\textsuperscript{[16]} On average, the mean time interval between traumatic SCI/neurological impairment and diagnosis of CSA is 17.3 years.\textsuperscript{[8]} close to our reported mean of 15.1 years; however, we included data of CSA secondary to nontraumatic SCI. Of note, Aebl et al. found that CSA secondary to traumatic SCI presented roughly 14 years sooner than CSA secondary to nontraumatic SCI.\textsuperscript{[17]} Radiological testing may feature extensive intervertebral disc degeneration, significant erosion of the vertebral body, hypertrophic paravertebral osteophytosis with pseudotumoral appearance, and early facet destruction.\textsuperscript{[8,13,17,18]} Radiographic involvement of all three columns of the spine allows for differentiation from infectious or degenerative etiologies. Computed tomography (CT) is valuable in the assessment of the severity of vertebral bone destruction and paravertebral bone formation. Magnetic resonance imaging may provide increased resolution of adjacent soft tissue.

Although of rare occurrence, CSA may present as a paravertebral pseudotumoral mass, resembling a spinal tumor.\textsuperscript{[18]} Care must be taken during the differential diagnosis to rule out other progressive destructive spinal pathology, spondylodiscitis, pyogenic spondylitis, metastatic spinal tumor (including both solid and hematological), osteomyelitis, discitis, and Paget’s disease.\textsuperscript{[9,13,14,18,19]} The presence of gas within the disc spaces, also known as the “vacuum phenomenon,” is a diagnostic criteria and can help differentiate CSA from spondylodiscitis.\textsuperscript{[20]} Tumors can be ruled out through specific immunohistochemistry markers. Due to the relatively nonspecific nature of its symptoms, CSA is arguably an underdiagnosed pathology.

**TREATMENT**

Treatment options for CSA typically involve conservative monitoring, nonsurgical immobilization, or surgery.\textsuperscript{[8]} Assuming no contraindications, surgical management has become the ideal treatment modality for CSA, particularly if gross spinal instability is present, to avoid spinal cord compression or cauda equina syndrome. For cases in the elderly who cannot tolerate surgery, or patients with minimal neurological sequelae, it may be appropriate to pursue a conservative or nonsurgical treatment approach. In CSA cases with mild bony involvement, the surgeon may decide to use a posterior column-only construct. This approach should be considered in complete sensorimotor paraplegic patients who are not at risk of neurological aggravation.\textsuperscript{[16]} Otherwise, the majority of surgical procedures for CSA today comprise of a circumferential arthrodesis, involving a combined anterior-posterior vertebral column construct [Figure 2]. Intralesional debridement before fusion is ideal\textsuperscript{[13,19]} and has shown greatest efficacy when applied through an anterior approach.\textsuperscript{[16]} In wheelchair-bound patients, a neutral or slightly flexed sagittal position is ideal, as this facilitates proper bladder function.\textsuperscript{[21]}

![Figure 1: A 56-year-old man with a history of a complete spinal cord injury 23 years ago requiring a T9-L2 fusion presented with 4 months of sitting instability. Sagittal computed tomography (a) and magnetic resonance imaging (b) demonstrated bony destruction affecting L3 and L4 consistent with a Charcot spine](image-url)
METHODS

We performed a systematic PubMed database search using the terms “Charcot spine,” “CSA,” “neuropathic spinal arthropathy,” and “spinal neuroarthropathy.” Studies between 1991 and 2017 were included in our analysis. Our study focuses on patients receiving surgical treatment for CSA; however, nonsurgical treatment is briefly discussed in our narrative review. Inclusion of CSA patient data in our quantitative analysis required a minimum report of CSA surgical treatment levels, surgical approach (posterior-only instrumentation and fusion, anterior-only instrumentation and fusion, or combined anterior-posterior instrumentation and fusion), and presence or absence of postoperative failure of the initial CSA treatment requiring surgical revision (hardware failure, presumed pseudarthrosis, new Charcot joint formation, etc.). While traumatic SCI represented the most common etiology leading to CSA, patients with development of CSA secondary to Parkinson’s disease, congenital insensitivity to pain, and others were included in our study if they underwent surgical treatment, reported surgical approach, and presence or absence of initial CSA treatment failure. Our study analyzes the outcomes of surgical treatment for CSA regardless of the etiology of the initial SCI.

The following data points were extracted from our literature review: Patient age and sex, level of initial SCI (American Spinal Injury Association grade if reported), the number of levels fused during spine surgical treatment of the underlying condition (the “index procedure”), vertebral column level of CSA, the number of years between onset of the underlying condition and diagnosis of CSA, symptom presentation and important medical histories leading to the diagnosis of CSA, vertebral levels undergoing CSA surgical treatment, type of surgical treatment, total follow-up, and presence or absence of failure of the initial CSA treatment. All cases treated by circumferential arthrodesis were included, including single-stage and multi-staged approaches, posterior-only and combined anterior-posterior approaches, and multi-modal treatment paradigms including but not limited to the addition of BMP and four-rod instrumentation. Total follow-up was defined as the length of time in months from initial CSA surgical treatment to the last outpatient clinic follow-up [Table 1].

RESULTS

Twenty-seven articles from our literature search were included for data analysis [Table 1]. A total of 84 patients with 86 Charcot joints were included. Two patients presented with multiple Charcot joint involvement. The largest case series presented comprised of 23 patients. The mean age at presentation was 43.2 years (range 11–74 years). Gender was reported for 71 patients, resulting in 54 males (76.1%) and 17 females (23.9%). The level of initial SCI was reported in 72 patients. T10 was the most common site of initial injury (15 patients), with T12 (11 patients) and T8 (8 patients) being the second and third most common sites of initial injury, respectively. Index procedure fusion levels were reported for 58 patients. Patients developing CSA had long initial fusion constructs spanning a mean of 8.0 vertebral levels.

Pain (53.6%), deformity/loss of sitting tolerance (47.6%), and an audible click on movement/mechanical transfer (31.0%) were the most common symptoms leading up to a diagnosis of CSA at patient presentation. Other symptoms were present as well, including 10 patients (11.9%) presenting with symptoms of autonomic dysreflexia, 8 patients (9.5%) with concomitant infection, 6 patients (7.1%) with bladder/anal/erectile dysfunction, and 4 patients (4.8%) with skin breakdown. Four patients presented with a history of congenital insensitivity to pain, 1 with congenital insensitivity to pain with anhidrosis (CIPA), and 1 with Parkinson’s disease.

L2 was the most common vertebrae involved in a Charcot joint, with 25 of 86 Charcot joints (29.1%) involving L2. L3 and L1 were the second and third most common vertebrae involved with Charcot joints, at 27.9% and 26.7%, respectively.

The interval in years between onset of the underlying condition and CSA diagnosis was reported for 76 patients, with a mean of 15.1 years (range 1–41 years).
Table 1: Surgical treatment of Charcot spinal arthropathy

| Study                        | Case number | Age (years), sex | Level of SCI (American Spinal Injury Association grade) | Levels of initial fusion for SCI | CSA location | Interval between SCI and (years) | Symptoms                                                                 | CSA surgical treatment levels | Approach | Total FU (months) | Failure of initial CSA treatment? |
|------------------------------|-------------|------------------|--------------------------------------------------------|---------------------------------|--------------|---------------------------------|----------------------------------------------------------------------|-------------------------------|----------|------------------|-------------------------------|
| Staudt et al., 2017[22]      | 1           | 28, female       | None                                                   | None                            | L5           | N/A                             | Deformity, loss of DTR, history of congenital insensitivity to pain  | L2-ilium                      | Combined | 18               | No                            |
|                              | 2           | 32, male         | None                                                   | L1-I3 (SLS)                     | L4           | N/A                             | Lower extremity weakness, history of congenital insensitivity to pain | T11-ilium                     | Combined | 60               | No                            |
| Zyck et al., 2016[23]        | 1           | 60, male         | C6 (A)                                                 | N/A                             | L1-L2        | 41                              | Autonomic dysreflexia                                                  | T8-L5 FRC                     | Combined | 6                | No                            |
| Ravindra et al., 2015[24]    | 1           | 68, male         | T4                                                     | T3-T6                           | T10-T11      | 22                              | Deformity, increased lower extremity spasticity due to baclofen catheter fracture | T6-L2                         | Combined | 17               | No                            |
| Aebli et al., 2014[17]       | 1           | 47, male         | T8                                                     | T9-T12                          | L2-L3        | 20                              | Pain, deformity, autonomic dysreflexia                                 | T8-S1                         | Combined | 36               | No                            |
| Loriiat et al., 2014[16]     | 1           | 72, male         | None                                                   | None                            | L2-L3        | N/A                             | Pain, history of Parkinson's disease                                    | L1-L4                         | Combined | 60               | No                            |
| Moreau et al., 2014[25]      | 8           | N/A              | T9 (B)                                                 | T7-L3                           | L5-S1        | 30                              | Pain, audible click, spasticity                                       | Extension ilium               | Posterior only | N/A               | No                            |
|                              | 9           | N/A              | C6-C7 (A)                                              | T12-S1                          | T10-T11      | 25                              | Pain, deformity, autonomic dysreflexia                                 | T4-L4                         | Combined | N/A               | No                            |
|                              | 10          | N/A              | N/A (A)                                                | T2-S1                           | L5-S1        | 15                              | Pain, deformity                                                        | Extension ilium               | Combined | N/A               | No                            |
|                              | 11          | N/A              | N/A                                                    | None                            | L4-L5        | 5                               | Bladder/anal dysfunction                                              | Extension ilium               | Combined | N/A               | Yes                           |
|                              | 12          | N/A              | T8-T9 (A)                                             | T5-L1                           | L4-L5        | 11                              | Spasticity                                                            | Extension ilium               | Combined | N/A               | No                            |
| Kim et al., 2013[19]         | 1           | 57, male         | T8                                                     | N/A                             | T11-T12      | 26                              | Audible click, deformity                                              | T8-L3                         | Combined | 24               | No                            |
| Quan and Wilde, 2013[21]     | 1           | 42, male         | T6                                                     | T3-L3                           | L3-L4        | 21                              | Autonomic dysreflexia, deformity, bladder dysfunction                   | Extension S1                  | Combined | 2.5              | Yes                           |
| Jacobs et al., 2012[13]      | 1           | 36, male         | T9 (A)                                                 | T10-L3                          | T9           | 25                              | Pain                                                                   | T6-T12                        | Combined | 33*              | Yes                           |
|                              | 2           | 54, female       | T12 (B)                                                | T10-L2                          | T11-L2       | 40                              | Pain, audible click                                                    | T11-S1                        | Combined | 6*               | No                            |
|                              | 3           | 57, female       | T10 (A)                                                | T7-L1                           | L1           | 2                               | Deformity, loss of spasticity                                          | T7-ilium                      | Combined | 78               | No                            |
|                              | 4           | 22, male         | T6 (A)                                                 | T5-L4                           | L4           | 9                               | Pain                                                                   | T5-ilium                      | Posterior only | 19               | Yes                           |
|                              | 5           | 37, female       | T12 (A)                                                | T7-L3                           | L3           | 8                               | Deformity, pain                                                        | T12-ilium                     | Combined | 62               | Yes                           |
|                              | 6           | 40, female       | T4 (A)                                                 | None                            | T12          | 20                              | Pain, audible click                                                    | T8-L3                         | Combined | 7*               | No                            |
|                              | 7           | 42, male         | T1 (A)                                                 | T10-L2                          | T10          | 29                              | Deformity, pain                                                        | T7-L2                         | Combined | 0*               | No                            |
|                              | 8           | 47, male         | T12 (A)                                                | T10-L2                          | L2           | 25                              | Deformity                                                              | T10-ilium                     | Posterior only | 58               | No                            |
|                              | 9           | 48, male         | T8 (A)                                                 | T7-11                           | L1           | 30                              | Audible click                                                          | T9-ilium FRC                  | Combined | 0*               | No                            |
|                              | 10          | 46, male         | T10 (A)                                                | T5-L4                           | L5           | 22                              | Pain                                                                   | L1-ilium FRC                  | Combined | 17               | No                            |
|                              | 11          | 43, male         | T4 (A)                                                 | T2-T8                           | T8           | 4                               | Deformity                                                              | T2-L3                         | Combined | 1*               | No                            |

Contd...
Table 1: Contd...

| Study | Case number | Age (years), sex | Level of SCI (American Spinal Injury Association grade) | Levels of initial fusion for SCI | CSA location | Interval between SCI and (years) | Symptoms | CSA surgical treatment levels | Approach | Total FU (months) | Failure of initial CSA treatment? |
|-------|-------------|------------------|----------------------------------------------------------|----------------------------------|--------------|---------------------------------|----------|-------------------------------|----------|----------------------|----------------------------------|
|       | 12          | 35, male         | T10 (A)                                                  | T8-L3                            | L3           | 16                              | Deformity, loss of spasticity | T10-ilium FRC | Combined           | 4                   | No                   |
|       | 13          | 43, male         | T4 (A)                                                   | T4-L4                            | L4           | 26                              | Deformity                       | L1-ilium | Combined         | 95                  | Yes                  |
|       | 14          | 38, male         | T4 (A)                                                   | None                             | T9           | 11                              | Pain                            | T7-L2   | Combined          | 0*                  | No                   |
|       | 15          | 64, male         | T5 (A)                                                   | T3-10                            | L2           | 15                              | Deformity                        | T9-ilium | Combined         | 12                  | No                   |
|       | 16          | 39, male         | T5 (A)                                                   | T4-L4                            | L3           | 34                              | Deformity, audible click         | T10-ilium FRC | Combined       | 12                  | No                   |
|       | 17          | 50, female       | T12 (A)                                                  | T10-L2                           | L4           | 34                              | Audible click                    | T10-ilium | Posterior only | 40                  | Yes                  |
|       | 18          | 30, female       | T10 (A)                                                  | T5-S1                            | T10          | 30                              | Deformity, pain                  | T6-ilium | Combined         | 9                   | No                   |
|       | 19          | 46, male         | T8 (A)                                                   | None                             | L3           | 4                               | Deformity                        | T10-S1   | Combined         | 22*                 | Yes                  |
|       | 20          | 50, male         | T10 (A)                                                  | T7-L4                            | L5           | 29                              | Deformity, pain                  | L2-ilium | Combined         | 94                  | Yes                  |
|       | 21          | 39, male         | T8 (A)                                                   | T4-L4                            | L5           | 1                               | Pain                            | T10-S1   | Combined         | 99                  | Yes                  |
|       | 22          | 34, male         | T12 (A)                                                  | T10-L2                           | L3           | 15                              | Pain                            | T9-ilium | Combined         | 91                  | No                   |
| Bishop et al., 2010[21] | 1 | 33, male | T10 | T8-T12 | T12-L3 | 5 | Increasing abdominal girth, discomfort | T3-ilium | Combined | 12                  | No |
| David et al., 2010[21] | 1 | 44, female | T9 | T4-L2 | L3-L4 | 12 | Pain | T12-ilium | Combined | 24                  | No |
| Haus et al., 2010[22] | 1 | 46 | T10 (A) | T9-L3 | L3-L4 | 1 | Pain, skin breakdown, Deformity, audible click | T9-sacrum | Combined | 84                  | No |
|       | 2 | 46 | T5 | T2-T9 | L4-L5 | 28 | Pain, deformity, dysreflexia, Suicidal ideation | T5-ilium | Combined | 60                  | Yes |
|       | 3 | 41 | T12 | T9-L3 | T12-L1 | 1 | Skin breakdown, hardware exposure | T10-L4 | Posterior only | 48                  | Yes |
|       | 4 | 47 | T10 | T9-sacrum | L2-L4 | 13 | Pain, audible click, sensory loss | T9-sacrum | Combined | 60                  | No |
|       | 5 | 17 | T1 | T5-L3 | L2-L3 | 8 | Pain, skin breakdown, orthostatic hypotension, autonomic dysreflexia | L1-L5 | Posterior only | 252                 | Yes |
|       | 6 | 17 | T10 | T4-S1 | T11-T12 | 15 | Pain, audible click | T4-S1 | Combined | 348                 | Yes |
|       | 7 | 12 | T10 | T4-L4 | L1-L2 | 7 | Pain | T4-ilium | Combined | 216                 | Yes |
|       | 8 | 25 | C6-C7 | T4-ilium | T11-T12 | 25 | Pain, autonomic dysreflexia | T4-ilium | Combined | 180                 | Yes |
| Jameson et al., 2010[21] | 1 | 47, female | T10 | None | L2-L3/L4-L5 | 4.5 | Deformity | T10-S1 | Posterior only | 65                  | Yes |
| Morita et al., 2010[21] | 1 | 50, male | C8 | C6-T1 | L2-L3 | 30 | Autonomic dysreflexia, audible click | L1-L5 | Combined | 72                  | No |
| Proietti et al., 2010[21] | 1 | 33, male | T10 | T6-L2 | L2 | 10 | Pain, deformity, audible click, skin breakdown | T12-L5 | Combined | 3*                  | No |
| Hong et al., 2009[21] | 1 | 29, male | T5 (A) | T2-T11 | T11-T12 | 6 | Pain, audible click, deformity | T5-L2 | Combined | 6                   | Yes |
|       | 2 | 45, male | T6 | C4-S1 T6-T12/L1-L4 | L5-S1 | 10 | Pain, lumbar nodule | T5-S1 | Combined | 46                  | Yes |
## Table 1: Contd...

| Study                                      | Case number | Age (years), sex | Level of SCI (American Spinal Injury Association grade) | Levels of initial fusion for SCI | CSA location | Interval between SCI and (years) | Symptoms                                                                 | CSA surgical treatment levels | Approach | Total FU (months) | Failure of initial CSA treatment? |
|--------------------------------------------|-------------|------------------|--------------------------------------------------------|--------------------------------|--------------|-----------------------------------|-------------------------------------------------------------------------|--------------------------------|----------|------------------|----------------------------------|
| Cassidy and Shaffer, 2008[30]              | 1           | 23, male         | None                                                   | None                           | L1-L2        | N/A                               | Numbness, unsteady on feet (postoperative CIPA diagnosis)               | T12-L3                         | Anterior only | 24               | Yes                              |
| Morita et al., 2008[16]                    | 1           | 50, male         | C8                                                     | C6-T1                          | L2-L3        | 30                                | Autonomic dysreflexia, audible click                                   | L1-L5                          | Combined   | 60               | No                               |
|                                            | 2           | 62, female       | T5                                                     | T5 laminectomy                 | L2-L3        | 23                                | Pain, deformity, audible click                                         | T12-L5                         | Combined   | 60               | No                               |
|                                            | 3           | 55, male         | T7                                                     | None                           | L1-L3        | 17                                | Deformity, audible click                                                | T11-L6                         | Combined   | 84               | No                               |
|                                            | 5           | 55, male         | T12                                                    | None                           | L1-L3        | 36                                | Pain, deformity, audible click, infection                               | T11-L5                         | Combined   | 108              | No                               |
|                                            | 6           | 25, male         | T10                                                    | None                           | L4-L5        | 2                                 | Pain, audible click                                                     | L4-L5                          | Combined   | 120              | Yes                              |
|                                            | 7           | 73, male         | L1                                                     | None                           | L5-S1        | 33                                | Pain, deformity, audible click, infection                               | L2-sacrum                      | Combined   | 96               | No                               |
|                                            | 9           | 48, male         | T10                                                    | T9-T10 laminectomy             | T12-L1       | 11                                | Deformity, audible click                                                | T10-L3                         | Combined   | 48               | No                               |
| Suda et al., 2007[3]                       | 1           | 55, female       | None                                                   | None                           | T10-T11      | N/A                               | Audible click (prior laminectomy T7-L2)                                 | T5-L3                          | Combined   | N/A              | Yes                              |
|                                            | 2           | 42, female       | T12                                                    | T12-L1                         | L4-L5        | 2                                 | N/A                                                                     | L4-S1                          | Combined   | N/A              | Yes                              |
|                                            | 3           | 58, male         | N/A                                                    | None                           | T10-T11      | 1.5                               | Pain                                                                    | T7-L2                          | Combined   | N/A              | No                               |
|                                            | 4           | 48, male         | N/A                                                    | L1-L3                          | L3-L4        | 26                                | Pain, audible click                                                     | L1-S1                          | Posterior only | 12               | No                               |
| Rose et al., 2006[31]                      | 1           | 37, male         | T6                                                     | None                           | T10-T12      | 10                                | Pain, deformity, audible click, instability                              | T3-L4                          | Combined   | 1.5              | No                               |
| Mohit et al., 2005[32]                     | 1           | 50, male         | C8                                                     | N/A                            | T11-T12      | 20                                | Autonomic dysreflexia, audible click                                    | T8-L3                          | Combined   | 18               | No                               |
| Tsirikos et al., 2004[33]                  | 1           | 11, male         | None                                                   | None                           | L1-L2        | N/A                               | Lower extremity tingling, history of congenital insensitivity to pain   | T12-L3                         | Combined   | 84               | Yes                              |
| Standaert et al., 1997[6]                  | 1           | 29, male         | T9                                                     | T8-T11                         | T12-L1       | 6                                 | Pain, audible click, loss of spasticity, bladder/erectile dysfunction  | T8-L3                          | Combined   | 12               | No                               |
|                                            | 2           | 56, male         | T12                                                    | T12-L4                         | L4-L5        | 31                                | Pain                                                                    | T5-ilium                       | Combined   | 14               | No                               |
|                                            | 3           | 38, male         | T10                                                    | T7-T12                         | L1-L2        | 6                                 | Infection, loss of spasticity, bladder dysfunction                     | T5-ilium                       | Combined   | 7                | Yes                              |
|                                            | 5           | 30, female       | T7                                                     | T4-T12                         | L1           | 9                                 | Deformity, audible click                                                | T4-L4                          | Posterior only | 2                | Yes                              |
| Arnold et al., 1995[5]                     | 1           | 55, male         | T8                                                     | N/A                            | L1-L3        | N/A                               | Deformity                                                              | T12-L4                         | Combined   | 15               | No                               |
| Heggeness, 1994[34]                        | 2           | 39, male         | T9                                                     | T7-L2                          | L1           | 10                                | Pain                                                                    | T12-L2                         | Combined   | 0.75             | No                               |
|                                            | 1           | 17, female       | None                                                   | None                           | L3-L4        | N/A                               | History of congenital insensitivity to pain, lower extremity weakness   | L1-L5                          | Combined   | 12               | No                               |
| Pritchard and Coscia, 1993[35]             | 1           | 30, male         | C6                                                     | T2-T9                          | T12-L1       | 13                                | Deformity, infection                                                   | T11-L2                         | Posterior only | 12               | No                               |

Contd...
Table 1: Contd...

| Study            | Case number | Age (years), sex | Level of SCI (American Spinal Injury Association grade) | Levels of initial fusion for SCI | CSA location | Interval between SCI and (years) | Symptoms | CSA surgical treatment levels | Approach | Total FU (months) | Failure of initial CSA treatment? |
|------------------|-------------|------------------|--------------------------------------------------------|---------------------------------|--------------|---------------------------------|----------|-------------------------------|----------|---------------------|----------------------------------|
| Devlin et al., 1991[7] | 1           | 50, male         | T11                                                   | T8-L3                           | L1-L3        | 19.2 (average)                  | Pain, deformity | T9-L4                         | Combined | 48                  | Yes                               |
|                  | 2           | 27, male         | T3                                                    | T1-T4                           | T12-L1       |                                 | Bladder/erectile dysfunction | T9-L4                         | Combined | (average)          | Yes                               |
|                  | 3           | 53, male         | T12                                                   | T12-L3                          | T12-L2       | Pain, deformity                 | T7-L3             | Combined                     | No                                  |
|                  | 4           | 50, male         | C5                                                    | None                            | T9-T10, L4-L5 | Deformity                      | T3-5                  | Posterior only                | No                                  |
|                  | 5           | 56, female       | T12                                                   | None                            | L1-L2        | Pain, deformity                 | T8-L4             | Combined                     | No                                  |
|                  | 6           | 44, female       | T8                                                    | T8-T12                          | T8-T10       | Pain, deformity                 | T5-L3             | Combined                     | No                                  |
|                  | 7           | 74, male         | N/A                                                   | T12-sacrum                      | L3-L4        | Pain, neurogenic claudication  | T10-L5            | Combined                     | No                                  |
|                  | 8           | 53, male         | T11                                                   | T6-sacrum                       | T12-L1       | Deformity                      | T7-L5             | Combined                     | No                                  |
|                  | 9           | 43, male         | T5                                                    | T3-T10                          | L1-L2        | Pain, deformity, erectile dysfunction | T10-L4          | Posterior only                | Yes                                 |
|                  | 10          | 28, male         | T5                                                    | T2-L2                           | L4-L5        | Pain, deformity                 | T12-sacrum Combined | No                              | No                                  |

*Lost to follow-up. SLS - Spondylolisthesis; FRC - Four-rod construct; DTR - Deep tendon reflexes; CIPA - Congenital insensitivity to pain with anhidrosis; SCI - Spinal cord injury; CSA - Charcot spinal arthropathy; FU - Follow-up; ASIA - American Spinal Injury Association

Of the 84 patients included in our analysis, 71 received combined anterior-posterior instrumentation and fusion, 12 received a posterior-only construct, and 1 received an anterior-only construct. Twenty-one of the 71 patients (29.6%) receiving combined treatment had failure of their initial CSA construct, which resulted in requiring revision surgery. Seven of the 12 (58.3%) patients receiving posterior-only treatment along with the patient receiving anterior-only treatment also experienced construct failure, necessitating a revision surgery.

Total follow-up time was reported for 76 patients, for a mean of 42.7 months. Ten patients were lost to follow-up.

**DISCUSSION**

CSA today is most commonly seen in paraplegic patients who have undergone prior spine fusion surgery for traumatic SCI, with most Charcot joints appearing within the first two vertebrae distal to the caudal end of the initial fusion segment. L1-L2 and L2-L3 is the most commonly affected region, along with the thoracolumbar and lumbosacral joints. CSA has also been reported in segments rostral to the instrumentation level, although this phenomenon is rare.[16] Patients most commonly present with symptoms of lower back pain, sitting imbalance, progressive spinal deformity (usually kyphosis), and an audible clicking sound on changing postures. Some studies in the literature report loss of sitting tolerance/deformity as the most common symptom of CSA,[5,17,32] however, our data show back pain has a slightly higher rate of presentation. Diagnostic criteria for CSA must include the presence of a preexisting condition characterized by deterioration of deep pain sensation and proprioception, profuse bone resorption and osteogenesis, and histopathological evidence of nonspecific chronic inflammation, to differentiate CSA from other inflammatory and neoplastic pathology.[18] Radiographically, all CSA cases present with disc and vertebral destruction.

Long-segment instrumentation spanning five or more vertebral segments create lever arms that increase risk for CSA.[17] Patients developing CSA in our study present with long-segment constructs averaging 8.0 vertebral segments. Excessive biomechanical loads at the ends of the construct, specifically lateral bending and torso rotation, increase probability of CSA development. Supplementing this load by physical activities such as weightlifting may exacerbate the supraphysiological forces already experienced by the joint due to the long-segment construct.[21] Iatrogenic instability triggered by laminectomy in previous spinal surgeries may also increase risk of CSA development.[16,17] The majority of CSA develops within the region of instrumentation or laminectomy, or at the caudal end of the region that initially underwent operation; the exception to this being patients who undergo surgery for the cervical or upper thoracic spine.[20] Morita et al. describe the addition of ankylosing spinal hyperostosis (ASH) to SCI as an additional risk factor for CSA development, as 7 of their 9 patients (77.8%) developed CSA.
at the junction between, or at the end of the ASH.\textsuperscript{[16]} ASH is believed to limit mobility in mobile spinal segments and exposes them to biomechanical stress.

Most of the literature recommends a combined anterior-posterior circumferential fusion to reduce hardware failure rates, and there are studies which support this notion. A case of a 23-year-old male with CIPA underwent anterior-only fusion, a choice the authors believed was sufficient unless biopsies showed active infection.\textsuperscript{[38]} The anterior column-only construct failed, a complication the authors believe likely could have been avoided if they used a circumferential fusion instead. The patient suffered minimal consequences as a result of his CIPA. Of note, this patient did not show elevated C-reactive protein levels (0.9 mg/dl), a finding that contradicts most of the literature regarding CSA diagnostics.\textsuperscript{[38]} C-reactive protein levels have been cited as a measure specific to the diagnosis of CSA.\textsuperscript{[13]} However, there are instances such as the Cassidy case, as well as the Aydini case which do not test positive for elevated C-reactive protein levels.\textsuperscript{[30,36]} In general, patients with congenital insensitivity to pain receive CSA diagnoses at a much earlier age (22.3 vs. 46.7 years in the review by Barrey et al.).\textsuperscript{[8]} Patients with congenital insensitivity to pain may be at risk for new CSA development after fusion, so continued monitoring of these patients is paramount.\textsuperscript{[33]}

Circumferential arthrodesis may be achieved through a single-stage or multi-staged approach. A case study by Kim et al. describes a circumferential arthrodesis through a single-staged posterolateral costotransversectomy approach, which was done in an attempt to avoid the morbidity typically associated with a multi-staged combination of anterior and posterior surgery.\textsuperscript{[19]} Suda et al. also suggest a single-staged circumferential arthrodesis in systemically healthy patients. However, for patients with medical comorbidities, a multi-staged circumferential arthrodesis is advised.\textsuperscript{[3]} While there were no postoperative complications or evidence of hardware loosening, follow-up CT imaging showed inadequate preparation of the endplates and incorrect mesh cage placement, an issue the authors attributed to limited visual capabilities from their surgical approach, and difficulties establishing boundaries between bone and disc due to the scar tissue around the Charcot joint.\textsuperscript{[19]} In another case report presented by David et al., a 44-year-old paraplegic woman with a history of multiple anterior and posterior surgeries for scoliosis, developed CSA at L3 and L4, below her prior scoliosis fusion from T4 to L2. Because of her multiple anterior surgeries for scoliosis, the authors concluded an anterior approach would produce significant risk to the vessels adjacent to the Charcot segment, due to inflammatory adhesion.\textsuperscript{[21]} The single-stage, posterior 3-column resection approach used by the authors provides ventral access to the affected vertebral bodies without transecting the thecal sac, through ligation of nonfunctional roots within the Charcot segment.\textsuperscript{[21]} By permitting direct host-to-host bony contact, the procedure eliminates the need for anterior and posterior column struts, while reducing the number of graft-host sites for the bone union. The authors believe the single-stage, posterior 3-column resection with primary shortening approach avoids the potential complications of a long anterior cage or allograft segment.\textsuperscript{[21]} The use of BMP, in this case, must be acknowledged, as this biological agent is known to promote union and decrease treatment failure rates.\textsuperscript{[13]} While these case studies suggest high fusion and low hardware failure rates for single-stage posterior approaches, the results require further investigation due to the nature of their small sample size. It is also important to consider the length of follow-up in these studies, as hardware failure as a complication of surgical treatment of CSA is usually reported within the first 24-month postoperatively.\textsuperscript{[13]} The majority of studies in the literature report low failure rates of surgical CSA treatment; however, these studies are typically limited by the duration of follow-up. When followed for extended periods, surgical CSA failure rates increase significantly, suggesting CSA is a progressive disorder even with successful surgical fusion.\textsuperscript{[26]}

The extent of instrumentation necessary to maintain stability and reduce hardware failure continues to be a controversial topic. Vertebral segments lacking instrumentation adjacent to prior fusion segments are at increased risk for developing new Charcot joints. Adjacent prior fusion levels should be incorporated into the CSA surgical levels to prevent pseudarthrosis.\textsuperscript{[7]} To prevent relapse of CSA, some authors have proposed extension of posterior fusion from the first sacral vertebrae to at least the first level in a sensitive area.\textsuperscript{[27]} Patients with construct extension to the ilium have a decreased risk of development of secondary CSA compared to patients with constructs ending in the lumbar spine.\textsuperscript{[26,29]} Extension of instrumentation to the sacrum or ilium through a four-rod lumbopelvic construct may prevent development of new Charcot joints distal to the construct, as well as prevent hardware failure.\textsuperscript{[13]} Extension of fusion to the ilium is recommended in cases of lumbar CSA.\textsuperscript{[22]} However, the effect of the decreased lumbar spine mobility on patient function involving daily tasks must be considered. Quan and Wilde describe a case study of a 42-year-old Caucasian man presenting with complete T6 paraplegia and symptoms suggestive of autonomic dysreflexia 21 years after a traumatic SCI event. After multiple surgeries to achieve circumferential arthrodesis, the patient experienced bilateral loosening of L5
and S1 screws, resulting in revision surgery and extension of the posterior reconstruction caudally to the ilium. Ten weeks later, the patient sustained an intertrochanteric femoral neck fracture, a result likely caused by the biomechanical transfer of stress from the spinopelvic fusion to the hip joint. While spinopelvic fusion has been shown to decrease hardware failure rates across the lumbosacral junction; it also risks proximal femoral insufficiency fractures.\textsuperscript{[23]} It is critical to consider these risks when assessing whether to extend instrumentation to the ilium. In addition, it is also crucial to assess hip range of motion before surgery, as limited hip mobility may result in increased compensatory lumbar motion in a sitting or supine position, which can impair quality of fusion.\textsuperscript{[4]} Fusion to the ilium in settings of limited hip range of motion can also result in significant loss of daily activity function.\textsuperscript{[26]}

Autonomic dysreflexia and concurrent infection may present with CSA. Patients suffering SCI rostral to T6 are at risk of developing autonomic dysreflexia, due to interruptions of the connections between the brain and the splanchnic vascular bed.\textsuperscript{[11,23]} It is important to note that while the majority of patients with CSA present with symptoms of spinal instability, deformity, lower back pain, and audible clicking sounds on movement, all reported cases of CSA with autonomic dysfunction emphasize the chief complaints of hypertension-induced headaches and sweating on patient presentation.\textsuperscript{[13]} Consistent with the theory that SCI rostral to T6 are at increased risk for the development of autonomic dysreflexia, Selmi \textit{et al.} report two cases of CSA patients developing autonomic dysreflexia with initial SCI at C7-T1 and C5-C6.\textsuperscript{[37]} Lumbar spinal instability can also act as a rare trigger of autonomic dysreflexia, as demonstrated by a patient presented by Zyck \textit{et al.} with L5-S1 instability.\textsuperscript{[23]} While the etiology of CSA-induced autonomic dysreflexia remain unclear, theories in the literature have proposed pressure exerted on the presacral nerve plexus and retroperitoneal viscera secondary to the CSA spinal instability as a possible mechanism.\textsuperscript{[16]} Routine X-rays of the urinary tract in SCI patients can aid in the initial diagnosis.\textsuperscript{[37]}

Infection has been identified as both a risk factor and a complication of CSA. The literature suggests paraplegic patients with recurring infections, particularly urinary tract infections, are at increased risk for development of CSA,\textsuperscript{[38]} as supported by a case of a 57-year-old paraplegic man presenting with CSA and history of multiple urogenital infections and methicillin-resistant \textit{Staphylococcus aureus}.\textsuperscript{[39]} Infection as a complication of CSA is rare, with a rate of 9.5% (8/84) as presented in our study, and 17.4% (4/23) as reported by Jacobs \textit{et al.}\textsuperscript{[13]} Infected CSA lesions while rarely reported in the literature, present a complicated scenario. Fistula formation of subcutaneous cyst in the back or hematogenous dissemination are proposed causes for infected CSA lesions.\textsuperscript{[16]} In their two cases presenting with infected CSA, Morita \textit{et al.} suggest antibiotic treatment or subcutaneous fistula debridement may be insufficient once the infected CSA lesion has broken down, primarily because degeneration of all three spinal columns and severe instability would result in widespread infection and inflammation.\textsuperscript{[16]} While the majority of CSA instrumentation techniques involve internal fixation, Suda \textit{et al.} have described the benefits of percutaneous external spinal fixation in the treatment of infected CSA.\textsuperscript{[40]}

Although the majority of CSA cases today are secondary to traumatic SCI, we believe it is important to briefly discuss some of the rarer causes and symptoms of CSA. Studies in the literature report rare cases of CSA presentation in patients with Parkinson's disease, although no definitive association between the two pathologies has been established.\textsuperscript{[14]} van Eeckhoudt \textit{et al.} reported a case of a 65-year-old woman with a history of both type 1 diabetes mellitus and Parkinson's disease, but were unable to definitively conclude whether the Parkinson’s exacerbated her CSA.\textsuperscript{[9]} Of note, the patients’ C-reactive protein was elevated at 1.4 mg/dl (normal <1 mg/dl), consistent with the literature on C-reactive protein as a specific diagnostic marker for CSA.\textsuperscript{[13]} CSA has also been reported in the setting of vascular lesion, such as spinal arteriovenous malformation.\textsuperscript{[41]} Bishop \textit{et al.} describe a CSA case involving a 38-year-old male presenting with abdominal discomfort and increasing abdominal girth as a result of massive bone destruction and cystic formation with hyperemia in the retroperitoneal space.\textsuperscript{[10]} Regarding rare symptom presentation of CSA, Oni and Dajoay-Mejia report a case of ascending cephalad sensory loss. While nonspecific to the diagnosis of CSA, the authors suggest cephalad sensory loss as a clinical manifestation supporting its diagnosis. Negative tests for infectious and neoplastic etiology ultimately led to the diagnosis of CSA.\textsuperscript{[42]} Finally, progressive deformity can be accelerated by an intrathecal baclofen catheter, increasing risk for CSA formation.\textsuperscript{[24]}

Conservative management may be indicated in elderly patients with medical comorbidities contraindicated to surgery. It also remains an option for early-stage CSA patients; however, because patients with complete SCI can be exposed to recurrent infection from the urinary tract or a sacral decubitus ulcer, the risk of CSA infection must be considered before choosing a modality of treatment.\textsuperscript{[18]} In a study by Aebli \textit{et al.}, 3 of their 7 patients treated without surgery due to
increased anesthesia risk, died within 10 months, suggesting conservative management increases risk of mortality.[17]

The majority of studies in the literature are limited by duration of follow-up. We suggest future studies to analyze CSA surgical outcomes after a prolonged follow-up period. Our literature review also revealed the lack of studies on the relationship between radiographic fusion and functional outcome. As it is important to consider potential functional impact when considering stabilization surgery, we suggest this as a future topic of study.

CONCLUSION

CSA is predominantly a surgical disease that presents most commonly in patients with prior traumatic SCI. Circumferential arthrodesis remains the best option for surgical treatment of CSA. Since surgical intervention is associated with multiple complications including hardware construct failure, the authors recommend long constructs including sacropelvic fixation to avoid high failure rates. In addition, we advocate the use of BMP with surgical treatment to decrease failure rates.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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