CASE REPORT

Atlantoaxial instability and cervical noninfectious spondylodiscitis in a patient with Wegener’s granulomatosis: A case report

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Abstract
A 61-year-old male patient with Wegener’s granulomatosis was admitted due to neck pain and quadriplegia. Clinical evaluation showed severe cervical noninfectious spondylodiscitis, myelopathy, sagittal imbalance, and atlantoaxial instability. A combined anterior and posterior approach was implemented. Postoperative clinical evaluation showed improved neurologic status.

KEYWORDS
atlantoaxial instability, cervical spine myelopathy, noninfectious spondylodiscitis, spine surgery, Wegener granulomatosis

1 | INTRODUCTION

Wegener granulomatosis (WG) or granulomatosis with polyangiitis (GPA) is an autoimmune disease affecting small- to medium-sized vessels in almost all systems through a vicious cycle of humoral cell-mediated immune response. Classic clinical presentations are necrotizing lesions in paranasal sinuses/ pulmonary system, glomerulonephritis, and small–medium vessel wall necrotizing granuloma formation in the presence of positive antineutrophil cytoplasmic antibodies for proteinase 3 (PR3/C-ANCA) antigens.1,2 Compared with other systemic presentations, neurologic manifestations are less reported in WG. Headache, peripheral neuropathy, sensory-motor deficits, pituitary problems, hemorrhagic, and ischemic cerebral events are among the most reported nervous system clinical manifestations.3,4 Spine lesions in WG are even less prevalent, but the majority of the cases are around meningeal thickening or mass effect of granulomatous lesion on neural elements.3,5

Spondylodiscitis (SD) is the inflammation, destruction, and deformation of vertebrae and disk materials due to a pyogenic or noninfectious process. Noninfectious spondylodiscitis (NISD) is very similar to pyogenic spine osteodiscitis, but a distinct etiology cannot be identified in most of these cases. However, slow-growing bacterial and
fungal species have previously been reported; the exact correlation is still not proven. Spondyloarthropathy is a medical condition that immune mediators affect joints and disk–vertebra interface. At the same time, the disc tissue can be inflamed on the margins of the vertebra end plate–annulus fibrosus, but the disk structure is not destroyed. Despite the considerable body of evidence in autoimmune spondyloarthropathies, the exact correlation between systemic inflammatory disease and NISD is not reported yet.

Atlantoaxial instability (AAI) is a harmful pathological event. Trauma is the leading cause of AAI worldwide, but degenerative diseases, congenital and developmental errors, and systemic medical comorbidities are the most common predisposing factors associated with AAI. Rheumatoid arthritis (RA) is the most commonly reported systemic inflammatory disease causing AAI, but the association between other autoimmune diseases and AAI is less understood. Mechanical instability or neurological deterioration in patients with spondylodiscitis /AAI is a logical indication for surgical spine stabilization.

We describe a known case of WG presented with cervical noninfectious spondylodiscitis with severe kyphotic deformity, AAI, and cervical spine myelopathy treated with a combined anterior and posterior cervical spine surgery.

2 | CASE PRESENTATION

A 61-year-old male known case of Wegener granulomatosis (WG) was referred to our department for neck pain and quadriparesis. His general appearance was a gloomy and malnourished old male looking into the floor instead of a horizontal plane. He complained about his neck pain since long time ago, which was exacerbated within the last weeks, and was not amenable to conventional doses of analgesics. He also complained about his recent fear of holding up his head and looking in a horizontal plane. “I feel that if I keep my head up, I fall from behind, and my head will drop into my chest” he described his fear to maintain the horizontal plane’s line of sight. He also complained that his limbs, especially his hands, are not strong as before, and there is a tingling sensation in his hands more than legs. He was routinely appointed his trusted rheumatologist, and his WG was in control in recent years. He also had controlled diabetes mellitus (DM), hypertension (HTN), chronic kidney disease (CKD, due to WG), and coronary artery disease (CAD) that required coronary artery bypass grafting (CABG) surgery in previous years. He was on steroids, antirheumatic drugs, and multiple drugs for his CDK, DM, HTN, and CAD.

Physical examination showed severe limitation in range of motion (ROM) in his neck on voluntary and cautious passive movements. Manual muscle strength testing (MMT) of his limbs showed 3/5 in his upper limbs, which was worse in distal motor units (2/5 in hands) and 3/5 in his lower limbs, which equally inflicted all proximal and distal muscles. The strength and stamina were slightly lower in the right deltoid muscle (MMT = 2/5). His deep tendon reflexes (DTR) were absent (0) in upper limbs and exaggerated (3+) in lower limbs. Plantar reflexes were diminished with negative Babinski's sign. Muscle bulk was decreased in all four limbs, which was more prominent in both hands. He could not walk on his feet more than a few steps. Other physical examinations had no remarkable new findings regarding his existing medical comorbidities. Laboratory data showed baseline Cr = 2.6 mg/dl, and other laboratory results were in the acceptable range regarding his WG and underlying diseases.

Neuroaxis multidetector computerized scan (MDCT) showed severe degenerative cervical spine kyphotic deformity with increased atlantodental interval (Figure 1, ADI = 5.8 mm). The rest of the neuroaxis showed no remarkable findings. A cervical magnetic resonance imaging (MRI) was taken, which showed severe cervical spondylodiscitis, degenerative cervical kyphotic deformity with concurrent myelopathy (Figure 2). Myelography sequences showed severe cerebrospinal fluid (CSF) blockage. He was consulted with a rheumatologist and nephrologist as preoperative anesthesia consultation was requested. Informed medical consent was taken from the patient and his first-degree relatives. The neuro spine committee reviewed the case and suggested combined anterior and posterior surgical approaches.

To restore the optimal cervical alignment, considering the severity of the disease and poor bone profile of the patient, we planned to perform an extensive posterior laminectomy and cervical fusion(PCF) combined with augmentation of the anterior–middle column of cervical spine by anterior cervical disectomy, corpectomy, and using intervertebral grafts fusion with plate fixation(ACDFP). Compared with single-staged procedures, this combined surgery carries higher perioperative risks, but it reconstructs the whole cervical spine and restores the best cervical alignment. After final reviews, the patient was scheduled for cervical spine surgery.

2.1 | Operation

Under general anesthesia, using neuromonitoring guide, Mayfield® skull clamp was fixed, and the patient was set in prone position. Table 1 summarizes the complex surgical steps and approaches in a step-wise fashion. Prep
and drape were followed in sterile fashion, and the midline skin was incised and dissected from occiput-to-C6. Paravertebral muscles were stripped off, and the facets were freed from any connective tissues. Using a high-speed drill, all facets and zygapophyseal joint were drilled off and released. For early decompression and higher index of safety, using high-speed drill and under neuromonitoring control, an extensive laminectomy from C1 to C6 was performed. Posterior dural layer was normal appearing and covered with hypertrophied ligamentum flavum. Under close neuromonitoring supervision, soft tissues were removed as much as possible. After laminectomy completion, the thecal sac bulged out of laminectomy window. Observing this event warranted the surgical team about a more severe canal stenosis and significant compression on neural structures. Using C-arm navigation, C1, C3, C4, C5, and C6 lateral mass screws were inserted. For better maneuverability during alignment correction C2, instrumentation was skipped. The rods were not fixed. Due to several osteophytes, auto fusion, deformation, and sclerosis in anterior and middle column, the lordosis could not be obtained through a single stage posterior approach. So, the muscles, fascia, and skin were approximated with running sutures and a water-tight dressing was placed on incision line. The patient was rotated and was rested in supine position. Through a right-sided inferior-extended retropharyngeal approach, an oblique-longitudinal skin incision originating from submandibular region extending to supra-sternal notch was made medial to sternocleidomastoid muscle (SCM). Vital structure (vessels, trachea, esophagus, lymphatics, and recurrent laryngeal nerve) was dissected meticulously but generously to prevertebral fascia from C2 to C7. The ventral aspect of cervical spine was kyphotic and fused. There were multiple osteophytes in medial and paramedial borders of ventral surface. Using C-arm navigation, C3–C4–C5–C6 disc spaces were confirmed. Assisting high-speed drill and microsurgical techniques, aforementioned disc spaces were freed and all the discs, osteophytes and soft tissues, and then, cautious C3, C4, and C5 partial corpectomy were performed. Multiple series of bone-disk samples were sent for pathology, polymerase chain reaction (PCR) testing for slow growing organisms, and microbiologic culture and smear. The spine became completely mobile and maneuverable. We used 2× conventional auto-lock stand-alone ceramic intervertebral cervical cages for C3–C4 and C5–C6 disk spaces and an expandable titanium mesh cage for C4–C5 disc space. All the cages were filled with a mixture of autograft-allograft bones chips with vancomycin powder. After cage placement, using intraoperative maneuvers, near optimal lordosis was obtained and a prevertebral plate was bent and fixed onto vertebral bodies just over ventral midline surface of the cervical spine. A submuscular drain was inserted, and musculature and skin
were repaired in anatomic layers. A Philadelphia hard collar was fixed immediately, the patient was set in prone position again, the prior PCF incision was opened, and the optimal cervical lordosis was obtained. Thecal sac was relaxed and decompressed perfectly, the rods were fixed in lateral mass screws, and C1–C2 subluxation was reduced. The neuromonitoring showed no acute neurologic problems during operation. A generous mixture of allo/autograft bone chips was placed on fusion construct, and a submuscular drain was inserted and fixed. The surgical field was irrigated with copious amount of vancomycin-solved sterile normal saline. Paravertebral muscles, fascia, and skin were repaired in anatomic layers. The wounds were dressed, hard collar was re-fixed, and the patient rotated to normal supine position. Neurological status was checked and confirmed by final neuromonitoring checkup, and the patient was transferred to intensive care unit (ICU).

### 2.2 Postoperative period

After ICU admission, he received routine postoperative spine surgery medical orders including Dexamethasone 8 mg every 8 h plus Ceftazidime and Vancomycin intravenously (IV) for 3 days with proper renal adjustment. The patient regained full consciousness but remained intubated for better airway protection until the next day. Postoperative neurologic examination showed increased motor power (MMT = 4/5 in both lower and upper limbs, hands MMT = 3/5). Postoperative examination in the right shoulder showed persistence of mild C5 palsy (MMT = 3/5) in his shoulder abduction compared with the contralateral side. Sensory deficits had no subjective changes. A routine cervical MDCT was taken, and the alignment and screws position were checked to be desirable (Figure 3). Following 24-h postoperation, to prevent venous thromboembolism (VTE), unfractionated heparin (UFH) 5000 IU/every 12 h was administered subcutaneously for the whole hospitalization period. The next day the patient was transferred to the neurosurgery general ward and became mobile with assistance, and by the 4th postoperative day, he was able to walk with minimum assistance. He was able to hold his head in a natural position and maintain the horizontal plane’s line of sight. The anterior and posterior drains were removed 48 and 72 h postoperation, respectively. All the intraoperative samples and microbiologic workups were negative for common and rare microorganisms; thus, the diagnosis was confirmed as a true organism-negative NISD. He has discharged 5 days’ postoperation with a rigid Philadelphia collar, oral antibiotics, dexamethasone, acetaminophen, and subcutaneous heparin for the following 2 weeks. At discharge, physical examination showed stable neurologic and medical status.

![FIGURE 2 Preoperative MRI shows severe cervical spine myelopathy, canal stenosis, severe spondylodiscitis, and CSF blockage](image)

| First stage                        | • posterior cervical exposure  |
|-----------------------------------|--------------------------------|
|                                   | • posterior laminectomy, facetectomy |
|                                   | • posterior lateral mass screws insertion without rod fixation |

| Second stage                      | • anterior cervical exposure |
|-----------------------------------|-----------------------------|
|                                   | • anterior discectomy and partial corpectomy |
|                                   | • interbody graft/cage insertion with plate fixation |
|                                   | • suboptimal lordosis |

| Third stage                       | • posterior rod fixation with optimal lordosis and sagittal balance |
|-----------------------------------|---------------------------------------------------------------|
|                                   | • atlantodental reduction and posterior cervical arthrodesis fusion |

**TABLE 1** Concise presentation of surgical treatment for cervical spine surgery
2.3 | Follow-up

The patient was appointed 3 weeks later for his first postoperative visit. C5 palsy was not improved, and the wound was healed in general appearance. Two days after skin suture removal, he was admitted to the emergency department due to wound dehiscence. Laboratory tests showed no evidence of surgical site infection, and he underwent a revision operation for his wound dehiscence. The wound was clean with no obvious pus. The fascia was not healed. There were extensive allergic-like reactions around the Vicryl® threads used for fascia and muscle closure in the previous operation. Vicryl® sutures were removed, and there was no infectious process in deep or superficial compartments. After proper irrigation with antibiotic-containing sterile normal saline, the fascia and skin were repaired with Nylon® sutures, and the incision was repaired primarily. The next day, his first postoperative MRI was taken. MRI sequences showed adequate spinal cord decompression, optimum C1-C2 reduction, resolution of CSF block, and adequate cervical sagittal balance. No infectious lesions or hematoma were noticeable (Figure 4). The reason for wound dehiscence was assumed to be an allergic reaction to Vicryl® threads in the context of impaired wound healing regarding his multiple comorbidities and drugs. The patient was discharged 3 days later. Dexamethasone was not prescribed, and he was referred to a rheumatologist to maintain the antirheumatic drugs as low as possible, at least for 2 weeks. Next, follow-ups showed complete wound healing and stable medical condition.

3 | DISCUSSION

Microscopic polyangiitis, granulomatosis with polyangiitis (Wegener’s), and eosinophilic granulomatosis with polyangiitis (Churg-Strauss) are all collectively referred to as ANCA-associated vasculitis (AAV), which are a homogenous diversity of autoimmune diseases affecting small- to medium-sized vessels. In general, AAV is positive for anti-neutrophil cytoplasmic antibodies (ANCA). According to specific antigen-antibody immune response, ANCA is furtherly subclassified into two major subtypes,
anti-proteinase 3 (PR3) and myeloperoxidase (MPO), which are also referred to as C-ANCA and P-ANCA, respectively. WG is positive for C-ANCA in over 95% of cases and is diagnosed with a clinical trial of necrotizing sinusitis, bronchitis, and glomerulonephritis.5,13

WG’s renal involvements are very common but, unfortunately, are usually detected when the disease has caused permanent renal damage and is a late clinical finding. Glomerulonephritis is the most common presentation of WG in kidneys. Reduced glomerular filtration rate (GFR), increased serum creatinine level >2 mg/dl, and end-stage renal disease (ESRD) are the subsequent common problems in WG in lower frequencies.14,15

Central and peripheral nervous system (CNS/PNS) manifestations of WG are not uncommon but are less noticed. Headaches, sensory problems, motor impairments, neuropathy, cranial nerve deficits, seizure, neuropsychiatric presentations, intraparenchymal lesions, encephalopathy, and hypophysial disturbances are among the most common CNS/PNS presentations of WG.5,16

Holle et al. conducted a systematic review on WG and nervous system manifestations and reported 10%–45% of the patients with WG had CNS/PNS signs or symptoms.4 De Luna et al. categorized WG nervous system manifestations into vasculitis and granulomatous phenotypes (V or G-WG). The majority of the patients in the V-WG subgroup had headaches, while in the G-WG subgroups, motor deficits were more common.3 Spine presentations in WG are even less common. Pachymeningitis, necrotizing vasculitis in spinal feeding vessels, and granuloma formation in the spinal canal are the most reported spine lesions associated with WG.3 Barreto et al. reported prevertebral mass lesions in a known case of WG, which was presented with back pain, and after tissue biopsy, medical treatments achieved good outcomes.17 Mentzel et al. reported a case of WG presented with neck pain and paralysis. Her MRI showed pachymeningitis of the cervical spine, and she underwent laminectomy to restore her neurologic function.18 In a similar clinical scenario, Roy et al. reported cervical spine dura thickening presented with paralysis. They resected the thickened dura as much as possible and grafted the dural defect with a synthetic patch. They reported postoperative pseudomeningocele but excellent neurologic recovery.19

Nontraumatic/tumoral causes of atlantoaxial instability (AAI) are previously reported in patients with achondroplasia, Down syndrome, mucopolysaccharidosis type IV, neurofibromatosis, skeletal dysplasias, rheumatoid arthritis, ankylosing spondylitis, and even systemic lupus erythematosus.20–24 There is no exact pathophysiologic
explanation for AAI in autoimmune diseases. It has been hypothesized that chronic inflammation causes chronic synovitis in the atlas-dense joint capsule, ligamentous laxity, pannus formation, and finally impaired bone metabolism, all together, lead to AAI. Steroids, anti-inflammatory, and antirheumatic drugs in the context of a systemic inflammatory state will develop bone metabolism derangements, leading to osteomalacia, osteoporosis, degenerative deformities, joint-ligament instability, and insufficiency fractures in the spinal column. Noninfectious spondylodiscitis (NISD) is a devastating degenerative spinal column caused by almost every severe inflammatory process affecting the spinal column. Tuberculosis, brucellosis, candidiasis, aspergillosis, blastomycosis, cryptococcosis, sporotrich infection, histoplasmosis, and coccidioidomycosis are the most commonly reported diseases associated with NISD. As a matter of fact, in many patients with NISD, microbiological evaluations lead to bacterial or fungal etiology. However, it seems rational that a true NISD should be defined as immune-mediated destruction of the spinal column in which no specific “microbial” agent could be identified. Inflammation control, precise antimicrobial treatments, and surgical stabilization in selected groups of patients are the mainstay of treatment in spondylodiscitis. Mechanical instability, progressive spinal deformity, acute neurological manifestations, medical treatment failure in the presence of an infectious collection, and progressive clinical deterioration in treatment-refractory patients with unidentifiable etiology are the most rational indications for surgical interventions in spondylodiscitis. Surgical interventions diversify from a simple core needle biopsy to advanced complex spine procedures. Instrumented spine surgeries are unavoidable in severe spinal deformity and mechanical instability. Extensive surgical debridement of necrosis, restoration of sagittal balance, maximum bony fusion, and the best possible instrumented fixation are the treatment endpoints of surgical stabilization in osteodiscitis surgery.

Atlas-axis (C1–C2) junction is stabilized by close interactions of bones, ligaments, and muscular structures, safeguarding the C1–C2 region. Disruption in any of these elements can potentially result in atlantoaxial instability (AAI). C1–C2 region ligaments play a crucial role in atlantoaxial stability. The transverse ligament is one key element that limits excessive posterior migration of the C2 odontoid process and thus prevents brain stem damage. AAI is a problematic situation that could potentially be life-threatening. Compression of odontoid process on brain stem leads to severe disability or death. Multiple radiographic indices measure AAI. Atlantodental interval (ADI) is one of the most practical and easiest ways to measure and report AAI, which has been used for many years globally. ADI > 3 mm in adults is almost a confirmatory indicator for AAI. Nakamura et al. introduced new AAI indices for AAI, C1/4 space available for spinal cord (SAC) ratio, and C1 inclination angle, which both were comparable to ADI. Surgical treatments in AAI include a variety of atlantoaxial instrumented fixation. Du et al. conducted a systematic review and meta-analysis on different pedicular screw-rod-based techniques for C1–C2 stabilization. They found that almost all the techniques had similar results, while C1 lateral mass (C1LM)-C2 transligament screw (C2TL) had slightly lower biomechanical stability in lateral bending forces. Elliott et al. reviewed transarticular screws (TASs) and different screw-rod constructs (SRCs) for posterior C1–C2 fixation and found almost equal efficacy and safety of both techniques. In an exciting series, Chowdhury et al. treated eight patients with Hangman's fractures using C1–C3 lateral mass screws while skipping C2 instrumentation, and their results were promising. This technique becomes much more popular if the anatomical properties of C2 are unfit for instrumentation or the surgeon is not experienced in direct C2 instrumented fixation.

Anterior cervical approaches, including intervertebral disectomy and cage insertion for interbody fusion with or without plate placement (ACDFP or ACDF) or vertebral body corpectomy and intervertebral cage/bone graft placement, provide excellent access to disc space removal of vertebral body's pathologic tissues. Ideal cervical lordosis can be achievable through an anterior cervical approach. Posterior cervical approaches include laminectomy, facetectomy, and instrumented fixation (posterior lateral mass/pedicular/ laminar screws, hook systems, and wiring methods). Posterior techniques provide excellent decompression on neural elements and stabilize the axial and subaxial cervical spine in the best way possible while maintaining cervical sagittal balance. These techniques are specialized for each zone specific to their anatomic-pathologic properties.

Putting all together and because some patients have two or more existing cervical spine lesions, complex surgical approaches become more necessary for optimal results. In the literature review, cervical spine noninfectious spondylodiscitis, cervical kyphosis, and atlantoaxial instability are not reported in WG. Myelopathy in WG is being reported in previous publications, but AAI and NISD are not described before this case. Although this is the first report with this feature, still we cannot neither prove nor reject the association between WG and these findings.

In this novel case, the authors assumed that patient had an existing NISD, which was relapsing/remitting over the years. Newly emerged AAI and progressive CSM lead to acute neurologic deterioration. The surgical approach was based on previous surgical experiences and studies...
in similar reports, considering the patient’s underlying diseases, life expectancy, and performance score. In this operation, multilevel partial corpectomy, long segmented ACDFP in conjunction with multilevel hybrid cage selection (stand-alone ceramic prosthesis and titanium expandable mesh cage) provided excellent lordosis and sagittal balance restoration through an anterior approach. Posterior facet release, laminectomy, and axial–subaxial lateral mass screw fixation provided maximal neural decompression in the context of an excellent fusion bed. The posterior stage also reduced the ADI = 2.3 mm and entirely resolved C1–C2 instability. Due to the undeniable need for excessive facetectomy, C5 palsy was predictable, but the results even in the right shoulder with C5 palsy had better scores than preoperative poor neurologic function. Long operation duration, antirheumatic drugs, steroid therapy, poor nutritional status, and multiple medical comorbidities resulted in wound healing impairment and dehiscence, but the final outcomes remained excellent.

4 | CONCLUSION
Wegener’s granulomatosis can cause atlantoaxial instability and noninfectious spondylodiscitis with insidious clinical presentations. A combined anterior and posterior approach can result in best surgical outcomes.

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CONFLICT OF INTEREST
The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS
MR contributed to the conception of the work, data search, patient treatment, manuscript preparation, manuscript revision, final approval of the manuscript, and agreed to be accountable for all aspects of the work. M. Sabouri, BA, HT, MM, PR, SF, and SBM contributed to patient treatment, manuscript revision, final approval of the manuscript, and agreed to be accountable for all aspects of the work. M. Shafiei contributed to manuscript revision, final approval of the manuscript, and agreed to be accountable for all aspects of the work. AS contributed to the conception of the work, data search, data gathering, patient treatment, manuscript preparation, manuscript revision, final approval of the manuscript and agreed to be accountable for all aspects of the work. He is the corresponding author of the manuscript.

ETHICAL APPROVAL
All procedures performed were under the institutional and/or national research committee’s ethical standards and the 1964 Helsinki declaration and later amendments or comparable ethical standards. Isfahan University neurosurgery department board members supervised and approved this report on behalf of the Ethical Committee of Isfahan University of medical sciences.

CONSENT
Informed consent was obtained from all individual participants included in the study.

CODE AVAILABILITY
Not applicable.

DATA AVAILABILITY STATEMENT
Data and original images in the current study are available from the corresponding author on reasonable request. Authors can confirm that all relevant data are included in the article and/or its supplementary information files.

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