# 14 Tuberculosis of the Craniovertebral Junction

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## Introduction

Tuberculosis (TB) of the craniovertebral junction (CVJ) is rare, accounting for no more than 0.3 to 1% of all cases of spinal TB.[1–6] If not detected early and treated appropriately, CVJ tuberculosis may result in progressive osteoligamentous destruction of the most mobile segment of the spine. This may lead to atlantoaxial dislocation (AAD), basilar invagination (BI), rotatory dislocation, cervical canal impingement, and compression of vital cervicomedullary structures.[7–10] In this chapter, a brief management protocol for CVJ TB is presented.

## Pathogenesis

TB of the CVJ is usually secondary to either contiguous spread from the cervical, retropharyngeal, or mediastinal lymph nodes to the retropharyngeal space, or due to hematogenous spread from other organs to the metaphysis of the involved vertebrae. Progressive infection results in increasing destruction of the ligaments and bone, that may culminate with involvement of transverse and alar ligaments and osteolytic destruction of odontoid or C1 leading to AAD.[11] Destruction of the bone eventually involves the anterior and posterior arches of C1, lateral mass of C2, and body of C2 along with occipital condyles, which may lead to marked instability of the region.[12] The destruction of the occipital condyles and C1–C2 facet joints may lead to BI.

## Clinical Manifestations

Severe neck pain (with or without suboccipital headache), restricted neck movements, and/or torticollis are the characteristic features of CVJ TB. The severity of neck pain, feeling of instability at the upper cervical spine, and restricted neck movements often force these patients to support their chin constantly with their hands.[7] They support their head with the hands even when getting up from the lying down to the sitting position. Difficulty in swallowing or mechanical obstruction of the airway can occur due to a large retropharyngeal abscess. Ninth and tenth cranial nerve paresis can manifest as dysphagia, nasal regurgitation, and hoarseness of voice. Neurological deficits are seen only with advanced disease due to direct compression of the spinal cord/cervicomedullary junction by pus or granulation tissue or because of AAD or BI. Myelopathy, with weakness and spasticity, due to spinothalamic tract and posterior column dysfunction may occur in the later stages.[7] Patients may also complain of hesitancy of micturition and a sensation of incomplete evacuation. The respiratory status of these patients may be compromised. It can be evaluated at the bed side by a single breath count (SBC) and a breath holding time (BHT). A SBC of less than 10 or a BHT less than 10 seconds is considered abnormal.[7,13,14] Systemic symptoms of TB (fever, night sweats, and weight loss) are encountered in 40 to 65% of the patients.[7,15,16] Cervical lymphadenopathy, abscesses within the posterior triangle of the neck, and discharging sinuses may also be encountered.

These patients often live in an area endemic for tuberculous infection and may have a history of contact with a patient of TB, or may be having past history of partially or even completely treated TB. TB at other sites is infrequently encountered. As neurological deficits in CVJ TB are a result of both cervicomedullary compression and instability, prognosticating the progression of neurological deficits on the basis of the extent of cervicomedullary compression may lead to a false sense of security.[4,5,7,9] Even patients with minimal demonstrable compression may, therefore, present with severe neurological deficits. A sudden death associated with CVJ TB may also occur due to an unrecognized instability at the CVJ.[17]

## Diagnosis of Tuberculosis

Hematological tests have a limited role in the definitive diagnosis of spinal tuberculosis. Elevated erythrocyte sedimentation rate (ESR) and C-reactive proteins (CRPs), although sensitive, are nonspecific for tuberculosis. Serial reduction in ESR and CRP is seen with disease healing. Cytokine assays like QuantiFERON-TB test can be used for the diagnosis of latent tuberculous infection in a population with low-to-moderate risk.[18] Other vital tests include serological testing for human immunodeficiency virus 1 (HIV 1) and HIV 2.

The Mantoux skin test is of limited value in the diagnosis of active TB because of its low sensitivity and specificity. False-negative reactions are common in immunosuppressed patients and in those with overwhelming TB. Positive reactions are obtained when patients have been infected with *Mycobacterium tuberculosis* but do not have active disease, and when persons have been sensitized by nontuberculous mycobacteria or bacillus Calmette–Guérin (BCG) vaccination.

Definitive diagnosis of tuberculosis can only be made with isolation of *Mycobacterium tuberculosis* on culture or demonstration of a caseating epithelioid granuloma, Langerhans' giant cells, lymphocytes, and plasma cells on histopathology of a tissue specimen.[4,5,18] It takes 4 to 8 weeks to grow the mycobacteria on the solid egg-based media. However, the newer liquid media may be able to achieve this within 10 days to 2 weeks. Drug sensitivity testing must be done on the positive cultures to detect drug resistance.

## Radiological Investigations

A chest radiograph is performed primarily to assess for pulmonary TB. Lateral and open-mouth views of the cervical spine may reveal reduction in the normal cervical lordosis, increase in the prevertebral soft tissue shadow (> 7 mm measured at the lower border of axis),[1,6] destruction or erosion of bones, AAD, rotatory subluxation, or BI. Lateral cervical radiographs in flexion and extension may reveal abnormal mobility (atlantodental interval > 3 mm in adults and > 4 mm in children). This may be reducible, partially reducible, or irreducible in nature. If destruction of the anterior arch of atlas and odontoid process by the tuberculous process leads to loss of conventional radiological markers that are used to diagnose AAD, like the anterior arch of atlas and the odontoid, the position of the posterior arch of atlas relative to the spinolaminar line may be utilized on lateral plain X-rays of the CVJ.[7,10,13,19] Radiographs of the cervical spine may often not detect early changes and instability, as angular C1–C2 displacements as well as erosion of the bones lead to the loss of the conventional craniometric parameters. Besides, bony changes require 2 to 6 months to appear on plain radiographs as nearly 50% of the bone needs to be destroyed before the changes are detectable on plain radiographs of the CVJ.[1,6,15]

A CT scan better delineates bony abnormalities like AAD, erosion or destruction of the odontoid, the body of axis, the anterior or posterior arch of atlas, the clivus, occipital condyles or the lateral masses of C1 and C2. An increase in the prevertebral soft tissue shadow can also be appreciated

On magnetic resonance imaging (MRI), especially with gadolinium contrast enhancement, the presence of prevertebral, paravertebral and epidural collections, and/or granulation tissue is assessed. A low signal intensity on T1-weighted (T1W) images and a high signal intensity on T2-weighted images are seen within the affected vertebral bodies, because normal fatty marrow is replaced by edematous tissue infiltration. This is often the first change to appear.[1,20,21] A multiloculated calcified abscess with a thick irregular enhancing rim in the presence of vertebral body fragmentation may occur. Other lesions at CVJ such as rheumatoid arthritis, sarcoidosis, brucellosis, fungal infections, lymphoma, or chordoma may produce a radiological appearance similar to that of CVJ TB. The common features of tuberculosis on MRI include bone marrow edema, pre- and paravertebral intraosseous

and epidural abscesses,[22–24] subligamentous spread of tuberculous abscess and granulation tissue to adjacent vertebral bodies beneath the longitudinal ligament and in the epidural plane with associated thecal sac compression.[25] Fat suppression images with gadolinium enhancement may potentially improve the contrast between the hyperemic osseous and soft tissue involvement and the normal structures.[26]

Follow-up T1-weighted images may be used to prognosticate the response to antitubercular therapy (ATT). When the patient starts responding to ATT, T1-weighted images show a progressive increase in signal intensity within the previously affected vertebrae suggesting fatty marrow replacement. The corresponding T2-weighted MR images show complete resolution of the granulation tissue and pus formation, correlating with clinical improvement.

## Staging of Disease

Lifeso has staged CVJ TB into[12] the following:

- Stage 1: Minimal bony or ligamentous destruction; no AAD.
- Stage 2: Minimal bony or ligamentous destruction; reducible or irreducible AAD present.
- Stage 3: Significant bony or ligamentous destruction evident.

Bhagwati's grading includes the following[27]:

- Grade 1: Inflammatory involvement of bony structures of the CVJ with formation of granulation tissue and destruction of bone.
- Grade II: Formation of a large retropharyngeal abscess with bony changes.
- Grade III: Associated subluxation of the atlantoaxial joint, either by bony destruction and/or laxity of apical and transverse ligaments.
- Grade IV: Formation of epidural abscess and compression of the cervicomedullary junction and the upper cervical cord, with neurological deficits, which may be mild or severe.

## Management of Tuberculosis of the Craniovertebral Junction

### Antituberculous Treatment

Irrespective of whether surgical or conservative treatment is pursued, ATT is the mainstay of treatment of CVJ tuberculosis. Tissue diagnosis must be obtained prior to starting ATT. The initial four-drug regimen (rifampicin 10–20 mg/kg/d, isoniazid 5–10 mg/kg/d, ethambutol 15 mg/kg/d in a single daily dose, and pyrazinamide 20–35 mg/kg/d in two divided doses) is recommended for 2 months[7,15,20,28–33] followed by a 2 or 3 drug regimen consisting of isoniazid and rifampicin or isoniazid, rifampicin, and ethambutol for 7 to 10 months. Pyridoxine 20 mg/d is given along with isoniazid to prevent peripheral neuritis. Periodic monitoring is essential to evaluate improvement as well as to detect adverse reactions to the ATT.

### Paraspinal Abscess

Tuberculous abscess may or may not be accompanied by bony and ligamentous destruction (**Fig. 14.1, Fig. 14.2a, b**). A *small abscess* usually resolves with antituberculous medication alone. In case of a *large abscess* that is showing a subligamentous spread into the subaxial spine resulting in respiratory distress, dysphagia, dysphonia, or swelling in the neck due to its mass effect, surgical intervention may be required. This may be in the form of a minimally invasive ultrasound or CT-guided aspiration of the abscess, or an open surgical drainage. An anteriorly based abscess at the C1–C2 region may be drained either utilizing the transoral approach[34] or the anterior retropharyngeal approach,[35] while a posterior or posterolaterally located abscess may be accessed using the midline suboccipital approach through the midline raphe. After abscess drainage, a rigid orthosis must be applied because incipient instability due to ligamentous affliction always accompanies the abscess at the C1–C2 level (**Fig. 14.2a, b**).

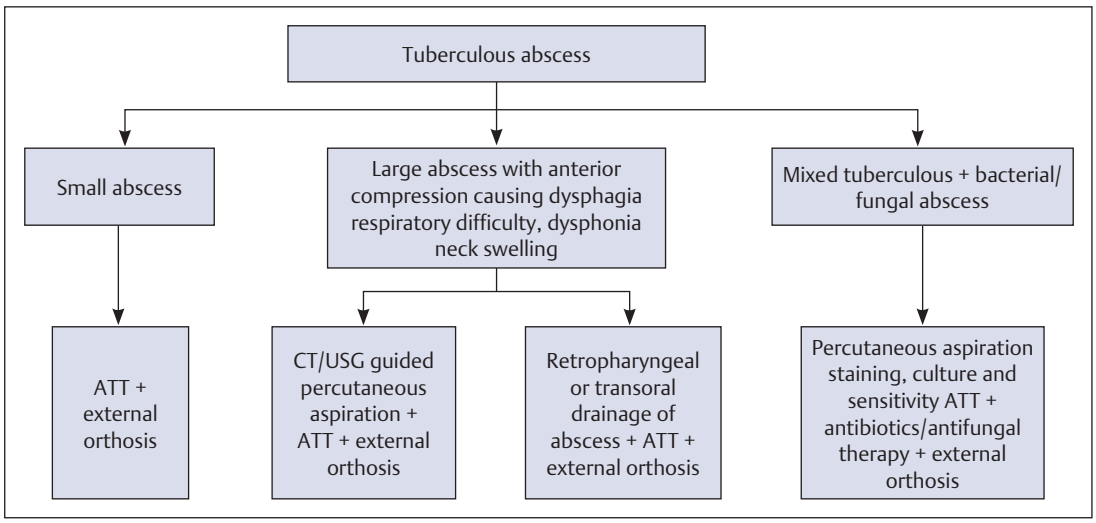


**Fig. 14.1** Algorithm for the management of craniovertebral junction (CVJ) tuberculous abscess. ATT, antitubercular therapy.

## Occipito-C1–C2 Instability

- *If the only evidence of tuberculous affliction is bony enhancement or granulation tissue without bony destruction or any evidence of atlantoaxial instability,* then ATT with an external orthosis may be adequate.
- *In case the tuberculous granulation tissue has caused occipital condylar and/or C1–C2 facetal and ligamentous destruction, or, is accompanied by irreducible or reducible atlantoaxial dislocation, but the patient has only neck pain, or only minor neurological deficit and is completely independent for all his or her activities of daily living* (**Fig. 14.3**), then a rigid orthosis like a halo brace or a Minerva jacket or strict bed rest (including not going to the toilet and not getting up for even eating) for at least 3 months must accompany the administration of ATT (**Fig. 14.4a–c**). The usual response to ATT occurs in 6 weeks to 3 months and includes a resolution of neck pain, improvement in the restriction of neck movements or neck deformity, absence of clicking sound on neck movements, and improvement in neurological deficits and in systemic symptoms like fever, arthralgia, night sweating, and loss of weight. The radiological improvement is in the form of resolution of soft tissue granulation, abscess, and edema; improvement of hypointensity on T1-weighted images in the vertebral bodies due to conversion of tuberculous granulation to normal bone marrow (signifying the presence of fat); decrease of abnormal bony hyperintensity on T2-weighted images (signifying the decrease of edema); and, absence of enhancement within the bone and in the soft tissues on MRI-enhanced images.[7,23,24] In case there is a clinicoradiological response to ATT, the patient must be reassessed with both dynamic (in flexion–extension) transtable lateral radiographs or sagittal reconstructed bone windows of computed tomographic images of the CVJ at the 3-month follow-up. If there is no AAD or a reducible AAD, and the patient has had neurological improvement, or at least no neurological deterioration, from his or her preoperative status of having "minor deficits but being functionally independent," then the patient is continued on a hard cervical collar and ATT is continued.
- *The rare instances when surgery is indicated in a patient with either no deficits or minor deficits include the following:*
  - ➢ *The presence of reducible AAD causing significant cervicomedullary compression with or without neurological*

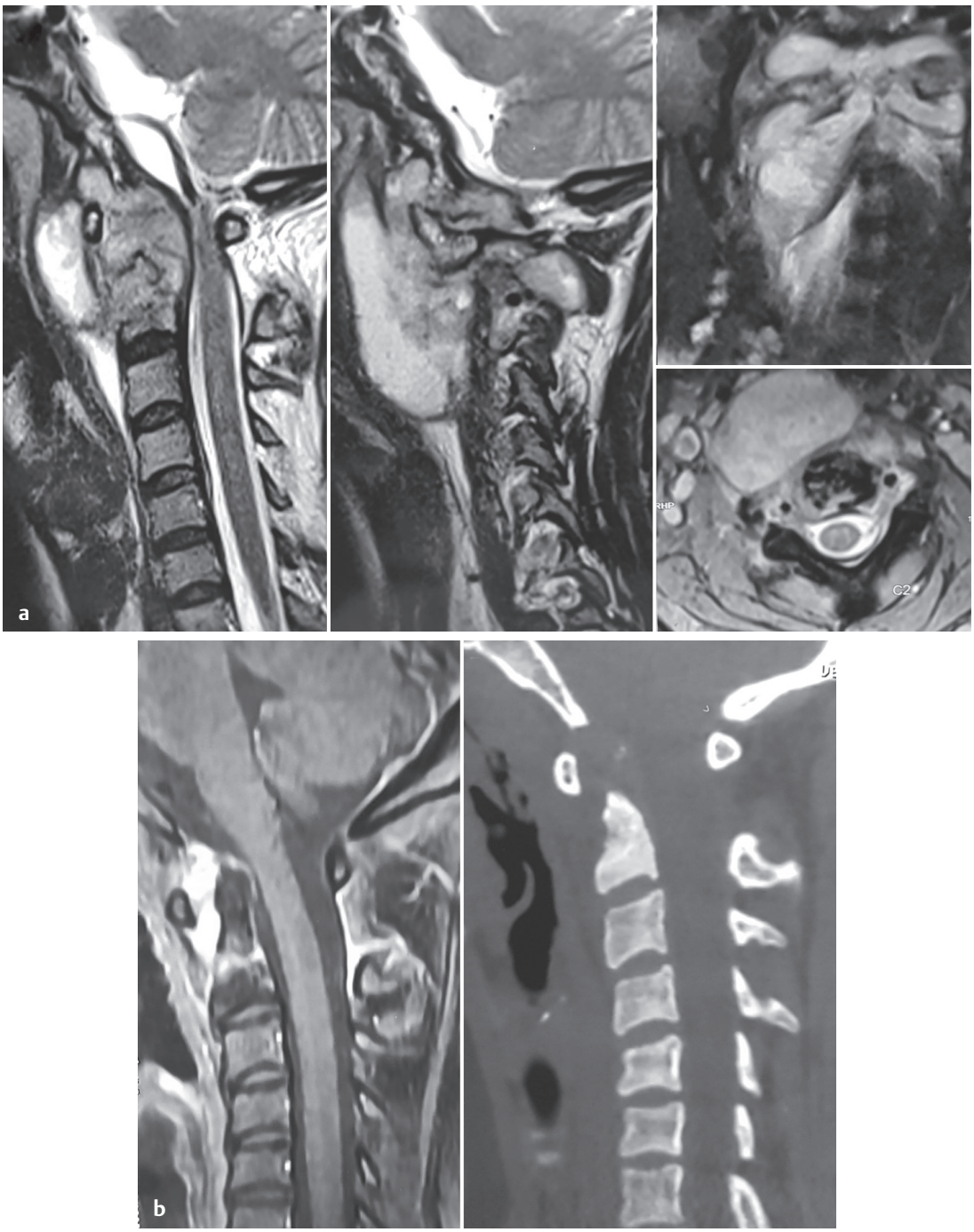


**Fig. 14.2 (a)** Patient 1: T2-weighted MR images: showing a large anterolateral tuberculous abscess, granulation tissue involving odontoid and atlantoaxial dislocation (AAD). As the patient had minimal deficits, a percutaneous drainage of the abscess under cover of antitubercular therapy (ATT) along with the application of an external orthosis was adopted. **(b)** T1-weighted sagittal contrast MR image after 1.5 years of ATT, showing significant resolution of the granulation tissue and abscess as well as soft tissue edema. A small amount of enhancing granulation tissue is still seen anterior to the odontoid; the sagittal CT reconstructed CT image, however, shows AAD with canal compromise.

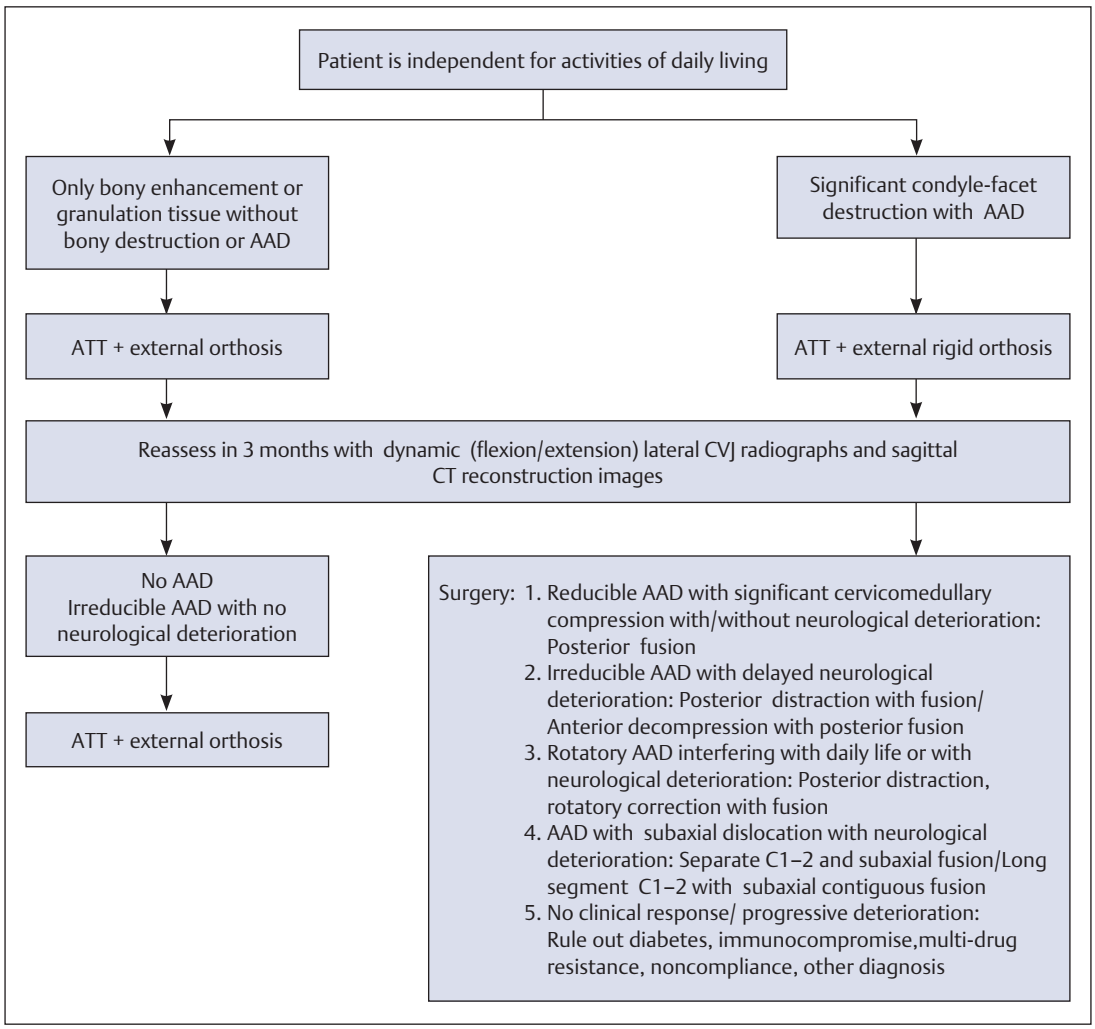


**Fig. 14.3** Algorithm for the management of craniovertebral junction tuberculosis (CVJ TB) in patients with either no deficits or minor deficits (those who are independent for their daily needs). AAD, atlantoaxial dislocation; ATT, antitubercular therapy.

*deterioration:* In this situation, a posterior stabilization procedure may be carried out either utilizing the occipitocervical plate and rod technique, the Ransford contoured rod technique, the C1–C2 transarticular screw, or the C1–C2 plate and rod technique.

- *If there is irreducible AAD with delayed neurological deterioration:* In this situation, if there is significant anterior osteoligamentous granulation and destruction causing anterior cervicomedullary compression, then a single-stage transoral or retropharyngeal odontoidectomy with C1–C2 (or occipitocervical, if needed) posterior stabilization is performed, and the patient is continued on ATT and a hard cervical collar for at least 3 months after surgery to ensure bony union of the posterior construct. In case the irreducible AAD is as a result of facetal instability, then either posterior distraction with stabilization using either C1–joint spacers or bone graft, or anterior decompression accompanied by posterior stabilization may be performed, and the patient may be continued on a hard cervical collar under the cover of ATT.

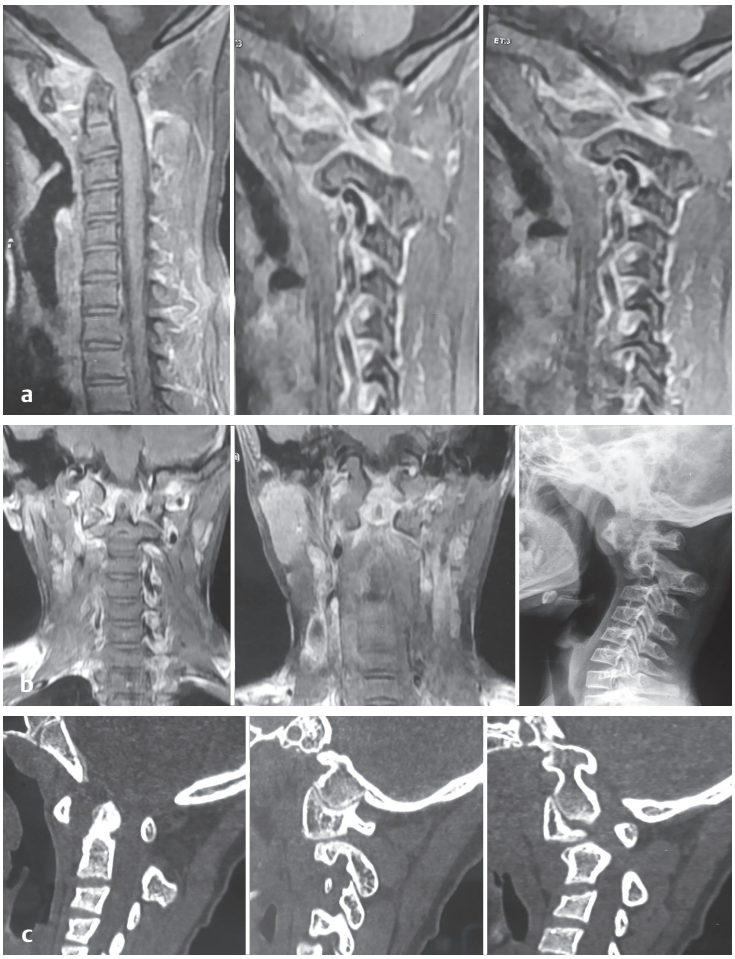


**Fig. 14.4 (a)** Patient 2: contrast-enhanced sagittal T1-weighted MR image showing extensive granulation tissue involving the craniovertebral junction (CVJ) with atlantoaxial dislocation (AAD). **(b)** Patient 2: contrast-enhanced coronal MR images showing extensive tuberculous granulation tissue involving the C1–C2 facet joints and the paravertebral ligamentous and muscle planes; the lateral radiograph of the craniovertebral junction showing AAD with canal compromise. **(c)** Patient 2: sagittal CT reconstructed images showing AAD with a stable right C1–C2 facet joint and a displaced left C1–C2 joint.

- *If there is rotatory C1–C2 instability that is progressive, is associated with significant neck rotation that is interfering with normal life style of the patient, and/or is accompanied by progressive neurological deficits:* The C1–C2 joint is opened posteriorly, distracted, and the rotation is gently corrected followed by stabilization using either C1–joint spacers or bone graft.
- *If the C1–C2 instability is accompanied by a contiguous or a remote subaxial instability with progressive neurological deterioration:* In case the subaxial affliction level is contiguous or in close proximity to the C1–C2 level, then the subaxial levels should also be included in the segments undergoing posterior fusion to prevent subsequent instability from occurring at the level. If the atlantoaxial and the subaxial levels are far from each other, it is also possible to carry out a simultaneous posterior stabilization procedure at two levels along with the institution of ATT and the hard cervical collar for at least 3 months.
- If there is no clinicoradiological response and/or progressive neurological deterioration in spite of 3 months of ATT, then the patient must be reassessed for being immunocompromised, having uncontrolled diabetes mellitus, being a multidrug ATT-resistant individual, being noncompliant to ATT, or having an etiology other than tuberculosis at the CVJ (**Fig. 14.3**).

- If the patient has severe and progressive neurological deficits and is bedridden and dependent on others partially or completely for his or her daily needs, then rather than subjecting him/her to the risk of pressure sores, pneumonia, or deep vein thrombosis due to the prolonged and mandatory recumbent position required during the conservative treatment, a more aggressive operative approach may be adopted.
- *If the patient suffers from significant anterior compression due to granulation tissue or irreducible AAD*, then a transoral/retropharyngeal decompression with simultaneous posterior stabilization, or, only a posterior stabilization with C1–C2 joint distraction using intrajoint spacer or bone graft may be done. *The presence of a reducible AAD* would warrant only a posterior stabilization with or without a C1–C2 joint distraction with intrajoint spacer or bone graft.

On the other hand, a conservative approach in the form of ATT along with neck immobilization utilizing a rigid external orthosis has also often proved to be beneficial in these patients, thus avoiding surgical intervention in these patients who often present with advanced myelopathy and respiratory embarrassment (**Fig. 14.5**).[7]

## Craniovertebral Junction Tuberculosis with Intramedullary Tuberculoma, Arachnoiditis, or Syringomyelia

An intramedullary tuberculous abscess, granuloma, arachnoiditis, vasculitis, obliterative endarteritis, radiculomyelitis, and syringomyelia due to the inflammatory exudate surrounding the spinal cord may be observed. An intramedullary tuberculoma may also occur. ATT forms the mainstay of treatment often leading to resolution of the tuberculous process. In case of rapid neurological deterioration, a short-term (2–3 week) course of steroids may be initially added in conjunction with ATT.

Surgery is rarely indicated to do the following:

- Drain a loculated intramedullary or subarachnoid abscess.
- To obtain a culture and sensitivity sample for institution of appropriate antibiotics.
- To remove a tuberculoma (leaving its capsule attached to the cord, which often does not have a well-defined cleavage) that is causing mass effect on the cord segment.

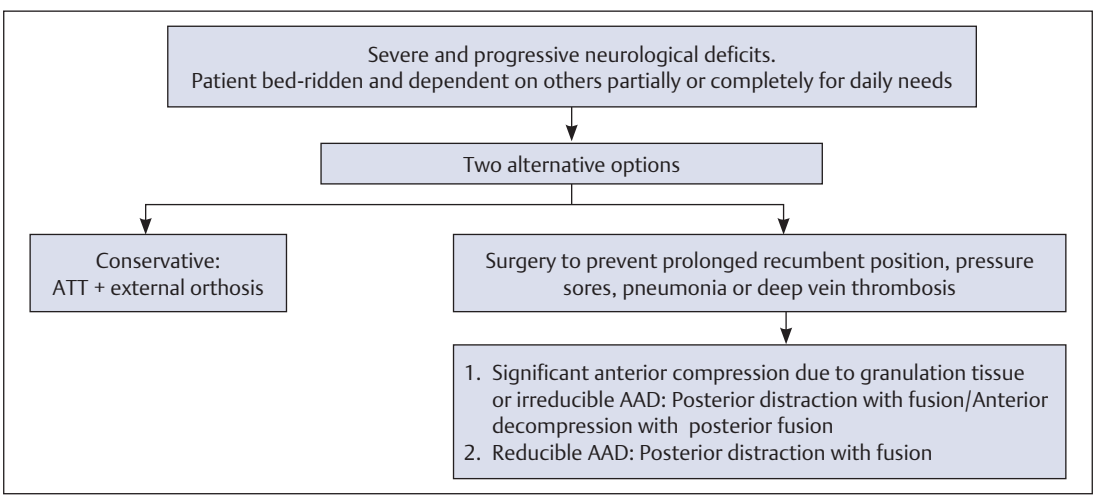


**Fig. 14.5** Algorithm for the management of craniovertebral junction (CVJ) tuberculous affliction in patients with significant deficits who are partially or completely dependent on others for their daily needs. AAD, atlantoaxial dislocation; ATT, antitubercular therapy.

- To obtain tissue for histopathological diagnosis, to divide a focal arachnoidal band that may be causing focal cord compression, and consequently, resulting in syringomyelia and to perform a laminoplasty with duraplasty to provide adequate space to the edematous cord. A syringosotomy or syringoperitoneal shunt for secondary syringomyelia seldom leads to neurological improvement as multiple arachnoidal adhesions within the subarachnoid space and adhesions within the syrinx cavity prevent the collapse of the secondary syrinx. An inadequate pressure head between the subarachnoid space/syrinx cavity and the peritoneal cavity also leads to the blockage of the installed shunt system.[36–38]

## Long-Term Spinal Deformity

Often, torticollis at the C1–C2 level produces the "cocked robin deformity" with rotation of the head in one direction and tilt in the other. At the subaxial spine, there is reversal of the cervical lordosis/kyphosis.[39] AAD and unilateral or bilateral restricted axial rotation may also result.[39,40] Primary and secondary scoliosis of the cervical spine may also occur. Following starting of ATT, if osseous fusion occurs, kyphosis does not progress significantly. In case fibrous or fibro-osseous healing occurs, this kyphoscoliosis may progress further requiring treatment.[39]

A mild nonprogressive deformity following institution of ATT may be stabilized using an external orthosis and strict bed rest until bony union at the C1–C2 joints is demonstrable.

In the case of significant progression of kyphoscoliosis, at the C1–C2 level, a C1–C2 joint drilling, with joint distraction and C1–C2 or occipitocervical posterior distraction with stabilization may be required.[40,41] In case of the occipital bone condylar or the subaxial spine vertebral elements being also involved, a long-segment occipitocervical fusion may be performed.[40-43] In the subaxial spine, anterior decompression of the granulation tissue; anterior corpectomy with fusion using bone grafts and cages; posterior column shortening; and, anterior and posterior simultaneous cervical lateral mass fusion with bone grafting are the recommended operative techniques.[39]

## Role of Cervical Traction

Cervical traction may be utilized during several stages of management of CVJ TB:

- It may be utilized to stabilize the unstable atlantoaxial and subaxial joints prior to performing definitive surgery.
- It helps to convert an irreducible AAD to a reducible one, thus requiring only a single posterior stabilization procedure.
- It helps to distract the odontoid from the foramen magnum in patients with basilar impression secondary to C1–C2 facet or condylar erosion.
- It helps to orient the position of a posteriorly directed odontoid causing thecal compression, to a more vertical one, thus relieving pressure on the cervicomedullary junction, in patients who are having a rapid neurological deterioration.
- It helps to stabilize the subaxial spine and correct its curvature from a kyphotic to a lordotic one.
- It helps to stabilize the unstable cervical spine during surgery.[10]

## Follow-Up Assessment and Prognostication

A long-term follow-up of these patients with repeated imaging is recommended to evaluate the long-term sustenance of clinicoradiological improvement of these patients; to prevent a delayed deterioration either due to development of delayed instability, malunion or recrudescence of the disease; and, to assess the effectiveness of the posterior construct in bringing about a good posterior fusion. In CVJ TB, there are no specific radiological markers to determine outcome and even patients with an advanced myelopathy and with MR cord intensity changes at the cervicomedullary junction (that may be indicative of myelomalacia,

edema, ischemia, gliosis, or demyelination) may recover significantly following a combination of ATT, decompression, and stabilization at the C1–C2 level.

## Summary

- Tuberculous affliction of the craniovertebral junction, though uncommon, can lead to disastrous consequences including instability, deformity, neurological deficit, and even death. The management of most patients with early disease is simply ATT and rigid bracing after obtaining a tissue sample. In patients with significant instability, deformity, and severe neurological deficit, surgery is recommended to decompress the spinal cord, correct the deformity, provide stability, and fuse the pathological segments. Transoral surgery is restricted to obtaining a tissue sample for biopsy, draining a retropharyngeal abscess in patients with dysphagia or respiratory difficulty, and excising the anterior arch of atlas and the odontoid to decompress the spinal cord in patents with irreducible AAD or BI. Transoral surgery is usually followed by a posterior spinal stabilization. In patients with reducible AAD or BI, posterior instrumented fusion is the surgery of choice.[44]

## Key Points

- Tuberculosis of the CVJ presents early with neck pain, suboccipital headaches, restricted neck movements, and inability to hold the head. Some patients present with difficulty in swallowing or even respiratory difficulties.
- Myelopathy, variable deficits of the lower cranial nerves or the cervical nerve roots may be seen in the later stages.
- MRI helps detect early infection and visualize soft tissue extension and nerve compression. CT scans help to quantify the amount of bony destruction, while dynamic X-rays may reveal latent spinal instability.
- Transoral aspiration of pus from the retropharyngeal space helps in obtaining tissue for histopathological and microbiological diagnosis.
- Management is nonoperative with ATT and bracing in early cases without instability or neurological deficit.
- Posterior instrumented fusion is the preferred approach especially in patients with reducible AAD/BI.
- Transoral odontoidectomy and decompression followed by posterior stabilization is indicated in patients with significant anterior compression due to an irreducible AAD, anterior epidural pus, or granulation tissue.

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