Rod breakage due to complete resorption of bone graft in a patient with occipito-cervial fusion.

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ABSTRACT

Implant failure after occipitocervical fusion (OCF) is rare. A 47 year old male presented with neck pain and features of cervical myelopathy. Imaging of cervical spine showed instability at C2/C3 and compressive myelopathy due to tubercular infection. He underwent OCF with bone graft. Post operatively he improved clinically. After 7 months of initial operation, there was reemergence of symptoms suggesting rod breakage. Revision operation was planned. Per operative finding was complete resorption of the bone graft. At 2 months follow-up, the hardware and bone graft was found to be in good position with significant improvement in symptoms. We concluded that complete resorption of bone graft from first surgery led to poor post-operative fusion and rod breakage.

KEYWORDS: Occipitocervical fusion, revision surgery, rod breakage

INTRODUCTION

Occipitocervical fusion is used to treat instability between skull and spine.¹Instability results from trauma, congenital diseases, tumor, iatrogenic injuries, infection (eg, tuberculosis) or inflammation (eg. rheumatoid arthritis).¹ Stabilization is challenging due to increased mobility at occipito-cervical junction.²Joints are stabilized by a strong ligaments protecting spinal cord integrity.³

Tuberculosis of occipital cervical junction accounts for 0.3- 1% of all cases of Pott's disease.⁴ Clinical features includes cervical pain, neurological deficit, dysphagia or dyspnea.⁵ Dislocation at C2/C3 level could results in mortality or complete quadriplegia. Global incidence of symptomatic rod breakage is 6.8% in adult patients who underwent deformity correction.⁶ Implant failure after posterior cervical spine fusion was found to be 4.2%.⁷ Rod breakage due to lack of fusion secondary to resorption of bone graft in OCF is not reported yet. In this report we present revision surgery for rod breakage after OCF due to graft resorption.

CASE REPORT

We present a case of 47 year old male with C1-C3 tubercular spondylodiscitis, C2/C3 dislocation, cervical myelopathy with Nurick grade 2 and Modified Japanese Orthopedic Association (mJOA) score of 15/18. He developed neck pain progressive for 3 months. He experienced altered sensation over bilateral upper limb and developed unsteady gait. There was no history of trauma, fever, significant weight loss, malaise, night pain, dysphagia, dyspnea and bowel/bladder incontinence. There was no past history of pulmonary tuberculosis, malignancy or family history of the same.

On examination the patient had tenderness over upper cervical region and painful movement of neck. Power over key muscle groups were 5/5 in all four limbs with altered sensation in both upper limbs. Patient had presence of myelopathic signs with exaggerated deep tendon reflexes in all four limbs. Bilateral Plantar reflex was downgoing with no evidence of clonus. Perianal Nepal Orthopaedic Association Journal (NOAJ)

sensation and anal tone was normal.

X-Ray images showed osteolytic destruction of C2/C3 vertebrae and translation of C2 over C3confirmed with CT and MRI images. Prevertebral shadow extending in front of C1-C3 vertebrae was seen compressing against the esophagus. Severe central spinal canal stenosis and myelopathic changes at C2-C3 level with marrow edema in C2-C5 body was seen in MRI.



Figure 1: Radiographic imaging (X-ray, CT scan and MRI) showing pathology at C2/C3 vertebrae.

Patient was operated after necessary preoperative assessment utilizing posterior approach. Gross destruction of lamina and posterior elements of C1-C3 vertebrae was observed. Modular plate was fixed over occipital bone with screws and connected to pedicle screw fixed over C3-C5 at 135 degree angle. Soft tissue and bone were sent for biopsy and Polymerase chain reaction (PCR) test. Bone graft was harvested from posterior superior iliac spine for fusion.

Patient was discharged with SOMI brace and Anti-tubercular medication. He was under regular follow-up with improvement of symptoms. His PCR report was negative for Mycobacterium Tuberculosis and biopsy was negative for granuloma and malignancy. He later developed recurrence of symptoms with no other significant history at 7th post-operative month. Radiographic images showed broken rod with abnormal shift. On examination his reflexes were exaggerated with myelopathic signs. He was admitted with plan of revision surgery.



Figure 2: 7th month follow-up X-ray images showing broken implant

Complete resorption of previous bone graft was observed intra-operatively. Revision surgery was performed with removal of pedicle screw from C3 vertebrae and exchange of rod at 135 degree angle. Cancellous along with cortical bone graft was harvested from PSIS for fusion. Biopsy from the soft tissue was sent.

Patent came for follow-up after 2 months with decrease in his symptoms. Neurological status was unchanged. X-ray showed implant in place along with bone graft. ATT and SOMI brace application was continued. His second biopsy was also negative for malignancy or granuloma.



Figure 3: X -ray images of 2 months after revision surgery.

DISCUSSION

Pilcher⁸ in 1910 was the first to report treatment of an atlantoaxial dislocation by occipitocervical fixation surgery. In 1927, Forrester described atlantoaxial and occipitocervical fusion for an unstable odontoid fracture with a fibular onlay graft, which led to satisfactory clinical results. Complications, like neck stiffness, axial pain, dysfunctional neck movement and dizziness, severe dysphagia, and dyspnea has been reported. ⁹ Loss of lower cervical lordosis and acceleration of degeneration, even the failure of implants, have been suggested in follow-up.⁷

OCF using screw-rod devices is routinely used for the treatment of spinal instability or deformity. There have been limited studies regarding rod breakage in such cases. Factors like posterior bony discontinuity and anterior mechanical imbalance may create a massive concentration of stress, causing fatigue fracture of the implant.¹⁰⁻¹² Our case had osteolytic destruction of bilateral laminae of C2 vertebrae with anterior translation of C2 over C3 vertebrae. These bony defects contributed to the posterior bony discontinuity and anterior mechanical failure. Concentration of stress on the rod may have led to its failure in due course of treatment.

Okamoto et. Al⁷ reported that bone fusion is by far the most important factor in preventing implant failure. Berjanoet al¹⁰ suggested that insufficient correction leads to sagittal imbalance and increased mechanical stress on the posterior implant. Tensile forces through the posterior graft cause bone resorption and reduce the chance of obtaining solid fusion. Total bone graft resorption was demonstrated when the patient was operated second time for revision surgery. It does indicate that the loss of bone graft and eventual poor fusion might have caused fatigue failure of the implant.

It has been reported that non-ideal correction can lead to sagittal imbalance and increased mechanical stress on the posterior implant. On the contrary, stress concentration through the posterior graft can lead to bone resorption and Nepal Orthopaedic Association Journal (NOAJ)

reduce the chance of obtaining solid fusion. Rod breakage, as a consequence, occurred at the point of stress concentration and the area of poor bone graft fusion. In our case we used 135 degree angulation of connecting rod for fixation of occipital plate with underlying pedicle screws.

CONCLUSION

Thus we can conclude that bone graft fusion is an important factor in preventing implant failure. Failure of fusion can lead to concentration of stress over posterior placed implant and cause its failure over time.

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