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Severe kyphotic deformity in tuberculosis of the spine

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Summary. *Almost 3% of cases of tuberculosis of the spine develop a severe kyphotic deformity. The patients at risk are those who developed the disease under the age of 10 years, who had involvement of three or more vertebral bodies and had lesions between C7 to L1. A severe kyphosis is more than a cosmetic disfigurement because nearly all such patients develop cardiopulmonary dysfunction, painful impingement between ribs and pelvis and compression of the spinal cord with paraplegia at an average of 10 years after the onset of the disease. Correction of the established deformity is difficult and dangerous. Anterior transposition of the cord does not always result in permanent neurological recovery, so it is imperative to diagnose and treat the condition either before bony destruction has occurred or when it is in an early phase. Those patients who are at risk of developing a severe deformity should be treated by posterior fusion of the spine.*

Résumé. *Près de 3% des tuberculoses du rachis s'accompagnent d'une cyphose sévère. Les patients à risques sont ceux dont la maladie a commencé dans la petite enfance (avant l'âge de 10 ans), qui ont une atteinte de 3 corps vertébraux ou plus avec une localisation entre C7 et L1. La cyphose importante ne doit pas être considérée seulement comme un trouble esthétique, puisque presque tous les patients vont développer dans un délai de 5 à 20 ans après le début de la déformation, des troubles cardio-pulmonaires, des douleurs par conflit costo-pelvien et des troubles neurologiques allant jusqu'à la paraplégie. La correction d'une déformation établie est difficile et dangereuse, la transposition antérieure de la moelle et des racines ne donne pas toujours un bon résultat sur les troubles neurologiques, aussi est-il impératif que le diagnostic et le traitement soient faits à une phase précoce de la maladie. Il faut traiter par arthrodèse rachidienne postérieure, les patients présentant des lésions à risques qui peuvent conduire à une cyphose importante.*

Introduction

Thirty million people throughout the world suffer from tuberculous disease with 8 million new cases occurring each year. The downward trend of the disease in affluent countries has been reversed because of the large number of people with autoimmune diseases. At present, over 3 million are infected with both HIV and tuberculosis.

Tuberculosis of the vertebral column constitutes nearly 50% of all lesions of osteoarticular tuberculosis, the commonest site for the disease being in the paradiscal region. Before modern imaging techniques became available, the clinician depended on conventional radiographs for making the diagnosis. The classical lesion was narrowing of the disc space with erosion and fuzziness of the paradiscal margins of the vertebral body and varying degrees of localised osteoporosis with

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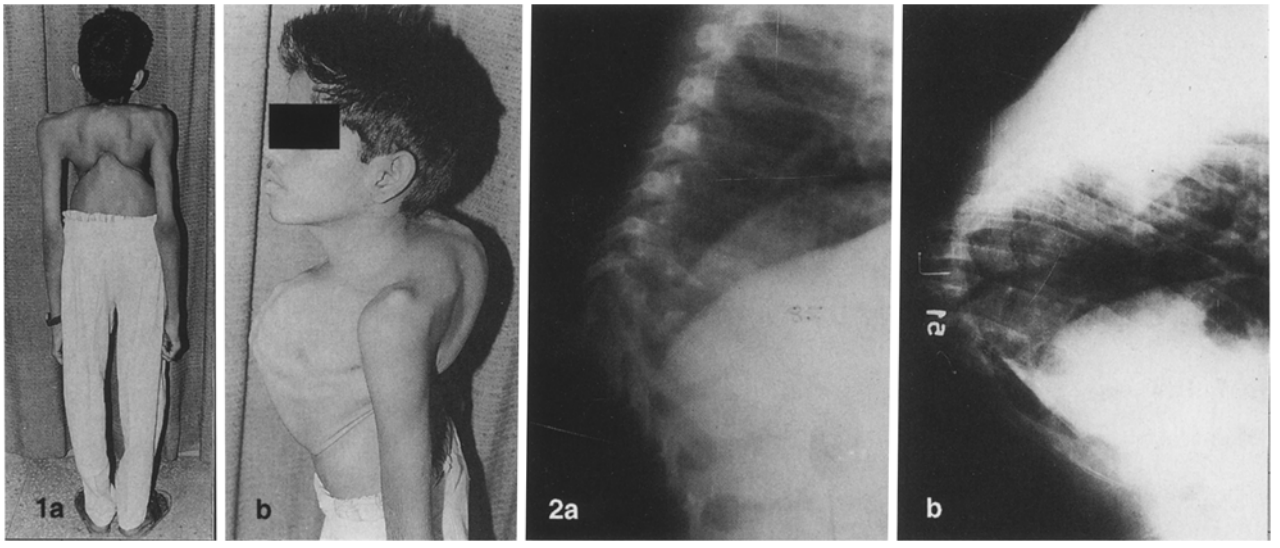

Fig. 1a, b. Photographs of a patient with a severe kyphotic deformity resulting from healed tuberculosis which involved a large number of thoracic vertebrae. He had the deformity for the part 10 years, and been able to walk. He was stunted and suffered from respiratory embarrassment, costo-pelvic impingement and bilateral extensor plantar responses when he was examined at the age of 17 years

Fig. 2a, b. Lateral radiographs of a young patient (**a**) in 1985 when the kyphotic angle was 35°, (**b**) in 1994 when the angle had increased to 140°. Such a rapid and severe deterioration is due to continued growth of the posterior elements when the vertebral end plates had been damaged. We advocate early posterior fusion in such cases

collapse of the involved vertebral bodies. By the time a patient reports for specialised treatment in developing countries, 95% show a clinically detectable kyphos or reversal of the normal lordosis.

In spite of prompt treatment with modern antitubercular drugs, with or without operation, some degree of deformity persists after the disease is healed. Analyses of large series shows that the kyphos increased from 0 to 10° in 85%, from 11° to 30° in 12% and to more than 30° in 3% of cases [7, 10, 11, 12, 15, 17, 20, 21, 22].

This paper considers those cases in which the deformity had increased to more than 30°.

Evolution of severe kyphotic deformity

Most of the deformity in adults is the outcome of, and directly proportional to, the diminution in height of the disc space and destruction of the vertebral bodies. If the disease occurred early in childhood, in addition to the destruction of the intervertebral disc and wedge-shaped collapse of the vertebral bodies, the growth plates of the vertebral bodies are also destroyed to a variable extent, resulting in slowing of their growth potential. As with progressive deformities of the limbs, continued growth of the posterior elements, when the anterior growth plate is arrested, leads to progress of the kyphos even after healing of the disease [2, 13, 15, 16, 20, 21].

Once the deformity is more than 45° the posterior spinal muscles are at a mechanical disadvantage which adds to the deforming force. In the thoracic spine, there is a pre-existing kyphotic curve and gravity perpetuates the deformity. The relatively large transverse processes and preexisting lordosis in the cervical spine minimise the kyphotic deformity. In the lumbar spine, the large vertebral bodies and vertical disposition of their posterior articular facets make them more likely to telescope than to angulate.

A formula to predict the kyphotic deformity has been suggested [18]: $Y = a + bx$, where Y is the future angle, a and b are the constants 5.5 and 30.5 respectively, and X is the initial loss of height of the vertebral bodies. However, it is difficult to estimate the initial loss of height of the vertebral body, or bodies, in children from radiographs.

If the paradiscal type of tuberculous spondylitis begins before the age of 10 years and affects 3 or more vertebral bodies with arrest of the same number of growth plates and the disease is between C7 to L1, there is a very high chance that a kyphotic deformity of 45° or more will develop (Figs. 1 and 2). The deformity may progress to almost 145° depending on the growth potential

remaining. We have not encountered deformity greater than this, because at this angle the vertebral bodies of the proximal part almost rest on those distal to the apex of the deformity (Fig. 2).

Prevention of severe deformity

The disease can now be diagnosed before destruction has occurred, provided the public are aware that symptoms should be reported at an early stage. Modern anti-tuberculous drugs, given for an average of 12 months, will result in healing without significant deformity and only slight narrowing of the affected disc space. In difficult areas and in young children, radiographs do not show the extent of the disease clearly, and CT and MRI are of great value. If the risk criteria mentioned above are encountered, usually in children with extensive disease, posterior spinal fusion, extending from a normal vertebra above to a normal vertebra below the diseased area, should be offered to minimise the chances of developing a severe kyphosis. Those presenting with gross destruction, severe kyphosis, abscess or sinus formation and neurological involvement reflect neglect on the part of the patient, his guardians and the attending physicians. There are, however, 7% of patients who fail to respond to modern drugs due to inherent or acquired resistance, atypical mycobacterium, an immune deficient state, or failure to comply with treatment. Complete and lasting control of the disease in such patients is almost impossible, despite using extensive surgery and newer drugs.

Posterior spinal fusion minimises progress of the deformity by arresting the growth of the posterior elements. A concomitant anterior operation of debridement, with or without decompression, is indicated if the disease is not controlled by drugs or if the neurological deficit does not respond to rest. Yau et al. pointed out that in childhood the kyphotic deformity may be unstable and progressive, and believed that the severe deformity associated with active disease was an absolute indication for anterior operation and stabilisation, as late reconstruction was difficult and dangerous [24].

When the disease involves more than two disc spaces, it is unwise to rely solely on anterior grafting to prevent increase in the deformity, although it may be done as part of a panvertebral fusion to improve the prospects of healing with minimal deformity.

Upadhyay et al. reported recently that they found no evidence in their cases to suggest that growth of the posterior elements, in the presence of anterior destruction, was responsible for progressive deformity [23], but they had excluded patients and lesions which have been generally observed to develop severe deformities, such as those with involvement of three or more vertebral bodies.

Correction of severe kyphotic deformity

This is major surgery which requires experience and thorough knowledge of the operative techniques of spinal and thoracic surgery. There may be serious blood loss and a risk of damage to the spinal cord. Most general orthopaedic surgeons discourage the correction of such deformities because of these difficulties and complications. Hodgson developed a method of correcting fixed spinal curves [5, 6] and the best results have been claimed only by the pioneers [11, 12]. Dommisse and Enslin reported their results in 55 patients with permanent paraplegia in 4, death in one and an average loss of spinal correction of 50% [1].

Hodgson used a transpleural anterior approach to the thoracic spine, combined with an extraperitoneal approach for the thoracolumbar region [5, 6]. The anterior area to be wedged open is cleared of all diseased tissue so that healthy bone is reached above and below where autogenous bone grafts are to be inserted. Care must be taken to avoid damage to the cord when working posteriorly. The corresponding spinous processes, laminae, pedicles and transverse processes are exposed and sufficient bone is excised to produce the required wedge. The wedge is then opened anteriorly and closed posteriorly by manual pressure at the convexity and simultaneous use of vertebral spreaders anteriorly. The anterior wedge is held open temporarily by sterile wooden blocks until they are replaced with massive autogenous iliac grafts. The posterior wedge is closed and secured by Harrington compression rods, whose hooks are firmly fixed into the proximal and distal pedicles, before completely opening the anterior wedge.

As the posterior gap is closed and the hooks approximated, the spinal cord is watched carefully and, if there is any pressure on the dura, bone is nibbled away. Posterior parts of the ribs above and below the deformity may have to be excised in order to close the posterior gap. After operation, the patients are nursed on previously prepared anterior and posterior plaster shells or on a Stryker frame for 8 to 12 weeks. The patient is then transferred to an ordinary bed and gradually mobilised wearing a spinal support.

Louis et al. advocated correction of the kyphosis by slow traction and corrective plaster casts

before operation [8]. In the thoracic and lumbar spine, correction of the deformity is followed by bone grafting and a plaster bed. A spinal osteotomy being carried out in late cases. Other methods have been used in the attempted correction of severe deformities including halo traction [16] combined with pelvic traction [14], with tibial traction [19] or with sacral bars [4]. The early results hold some hope for correction of severe kyphotic deformities. Louw used vascularised rib grafts after decompression for a neurological deficit and a preoperative kyphosis of 56° was corrected to 27° after operation [9].

We would not recommend operative correction of fixed deformities proximal to L2. It is safer to prevent deformity before it becomes fixed by progressive halo-pelvic or halo-femoral traction before operative fusion. The maximum limit for traction is 13 kg through the skull and 6½ kg through each femur. The traction should be increased gradually and long tract function carefully observed. All the correction takes place within the first 3 to 4 weeks. It is dangerous to add further correction on the operating table and this should not be done unless facilities for spinal cord monitoring are available. Any correction obtained before operation should be of the nonfixed element of the deformity. In the postoperative period, after the loosening effect of anterior surgery, further correction can be obtained using cephalo-caudal traction, especially of severe kyphotic deformities proximal to L1. We attempted correction of severe kyphotic deformity in two children by a panvertebral operation and halo-pelvic traction. One of them, who had a deformity with the apex at T11, had severe permanent deterioration of his neurological state; the other, with a deformity at L2-L3, had lost 50% of the postoperative correction after 2 years.

Neurological complications due to severe kyphotic deformity

Most patients with severe deformity remain able to walk for many years after the deformity has been established (Fig. 1). The cord tolerates a marked degree of angulation because the deformity occurs slowly over a few years. Many patients, however, begin to show neurological symptoms of a late onset paraplegia after 5 to 20 years. Prolonged stretching of the cord over the internal kyphos results in atrophy or gliosis of the spinal tracts [3] or other ischaemic changes in the cord [22].

Laminectomy with anterior transposition of the cord has been suggested by Griffiths et al. [3]; spinal nerve roots are divided to permit rotation and retraction of the theca so that bone can be removed from the front of the vertebral canal. We believe that satisfactory removal of the internal kyphos can be carried out through an anterolateral approach so that the cord lies in a relaxed anterior position without producing instability of the vertebral column. If necessary, the intercostal nerves on the opposite side can be divided by making another paravertebral incision. Only intercostal nerves can be sacrificed to allow the cord to shift, other spinal nerves cannot be divided without significant loss of function. When there is a severe kyphosis we feel that anterior decompression and transposition of the cord can only be accomplished by an anterolateral approach. An anterior approach is impossible in practice because of the difficulty in working so deeply at an acute angle.

Between 1965 and 1970, we carried out operative decompression for every case of severe kyphosis with neurological involvement.

Unfortunately, some of those who could walk were no longer able to do so after the anterior transposition procedure. Since 1971, our philosophy has been not rush to operate on patients with grade I or II paralysis. They are, however, kept under close observation. Deterioration is slow, but the moment a patient is unable to walk, anterior decompression and transposition is carried out through an anterolateral approach. Antituberculous drugs are given before and after the operation. The neurological recovery in 33 patients with a deformity of 45° or more was complete in 37%, partial in 15%, sensory improvement only occurred in 9%, no recovery in 27% and deterioration in 12%. The prognosis is worse the more severe the deformity, and a 60° kyphos seems to be the watershed. Those who do not recover after adequate surgery and those who deteriorate after a few years should be rehabilitated as paraplegics.

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References

1. Dommisse GE, Enslin TB (1970) Hodgson's circumferential osteotomy in the correction of spinal deformity (Proceedings). J Bone Joint Surg [Br] 52: 778
2. Friedman B (1966) Chemotherapy of tuberculosis of the spine. J Bone Joint Surg [Am] 48: 451–474
3. Griffiths DL, Seddon HL, Roaf R (1956) Pott's Paraplegia. Oxford University Press, London

4. Hardy JH, Grossling HR (1971) Combined halo and sacral bar fixation: a method for immoblisation and early ambulation following extensive spine fusion. Clin Orthop 75: 205–208
5. Hodgson AR (1965) Correction fixed spinal curves (*a* preliminary communication) J Bone Joint Surg [Am] 47: 1221–1227
6. Hodgson AR, Yau ACMC (1969) Anterior surgical approaches to the spinal column in Recent Advances in Orthopaedics. In: AG Apley (ed). Churchill, London, pp 289–323
7. Konstam PG, Blesovsky A (1962) The ambulant treatment of spinal tuberculosis. Br J Surg 50: 26–38
8. Louis R, Conty CR, Pouye I (1970) Operation of Pott's disease with correction of gibbosity (Chirurgie du mal de Pott avec correction des gibbosites). J Chir 99: 401–416
9. Louw JA (1990) Spinal tuberculosis with neurological deficit. J Bone Joint Surg [Br] 72: 686–693
10. Martini MEd (1988) Tuberculosis of the bones and joints. Springer, Berlin Heidelberg New York
11. Medical Research Council Working Party on Tuberculosis of Spine (1978) Five-year assessments of controlled trials of ambulatory treatment, debridement and anterior spinal fusion in the management of tuberculosis of the spine. Studies in Rhodesia and Hong Kong. J Bone Joint Surg [Br] 60: 163–176
12. Medical Research Council Working party on Tuberculosis of Spine (1982) A 10 year assessment of controlled trials comparing debridement and anterior spinal fusion in the management of tuberculosis of the spine in patients on standard chemotherapy in Hong Kong. J Bone Joint Surg [Br] 64: 393–398
13. Moon MS, Kim I, Woo YK, Park YO (1987) Conservative treatment of tuberculosis of the thoracic and lumbar spine in adults and children Int Orthop 11: 315–322
14. O'Brien JP, Yau ACMC, Smith TR, Hodgson AR (1971) Halo pelvic traction. J Bone Joint Surg [Br] 53: 217–229
15. Paus B (1964) Treatment of tuberculosis of the spine. Acta Orthop Scand (Suppl) No 72
16. Pieron AP, Welply WR (1972) Halo traction. Acta Orthop Belg 38: 147–156
17. Puig Guri J (1947) The formation and significance of vertebral ankylosis in tuberculous spines. J Bone Joint Surg 29: 136–148
18. Rajasekaran S, Shanmugasundaram TK (1987) Prediction of the angle of gibbus deformity in tuberculosis of the spine. J Bone Joint Surg [Am] 69: 503–508
19. Schmidt AC (1971) Halo-Tibial traction combined with Milwaukee brace. Clin Orthop 77: 73–83
20. Tuli SM, Kumar S (1971) Early results of treatment of spinal tuberculosis by triple drug therapy. Clin Orthop 81: 56–70
21. Tuli SM (1975) Results of treatment of spinal tuberculosis by "Middle-Path" regime. J Bone Joint Surg [Br] 57: 13–23
22. Tuli SM (1992) Tuberculosis of the skeletal system (bones, joints, spine and bursal sheaths). Jaypee Bros Med, New Delhi
23. Upadhyay SS, Saji MJ, Sell P, Yau AIMC (1994) The effect of age on the change in deformity after radical resection and anterior arthrodesis for tuberculosis of the spine. J Bone Joint Surg [Am] 76: 701–708
24. Yau ACMC, Hsu LCS, O'Brien JP, Hodgson AR (1974) Tuberculous kyphosis: correction with spinal osteotomy, halopelvic distraction and anterior and posterior fusion. J Bone Joint Surg [Am] 56: 1419–1434