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Personal view

Symptomatic lumbar spinal arachnoiditis: fact or fallacy?

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Summary It is generally accepted that chronic adhesive lumbar arachnoiditis is a cause of symptoms, notably back pain and/or pain (of almost any type, not necessarily 'anatomical') in the lower limbs, although there is no clearly defined clinical pattern which is clearly associated with this syndrome. There is no doubt that arachnoiditis occurs as a pathological and radiological entity due to a number of causes. In the view of the present authors, the nexus between the pathology and radiology on the one hand, and the patients' symptoms on the other hand, has not been demonstrated with any degree of scientific rigor. © 2000 Harcourt Publishers Ltd

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INTRODUCTION

Spinal arachnoiditis is a pathological entity with a multiplicity of causes and it is unwise to use the term to describe a clinical syndrome. There are also definable radiological changes and it is assumed by many authors that there is congruence between the clinical and the investigational findings. However, we question the rationale for this assumption. Horsley' is usually credited with priority, but Gowers[2] gave an elegant description of the condition and considered that some cases were due to `prolonged overexertion and sexual excess'! It is clear that most of the early cases were due to either syphilis or tuberculosis, and tuberculosis still accounts for many cases in certain parts of the world, notably South India (for instance, see Wadia & Dastur).[3] Horsley was aware of the association with syphilis when he described 'a relatively large number of such cases, on most of which I have performed laminectomy and subdural mercurial irrigation'. He described a localised encysted fluid collection, most commonly in the mid-thoracic region. Decompression improved a number of his patients, and he was at a loss to explain the pathology in some of his cases. Elkington[4] reviewed the literature in1936, and collected 41 cases which he analysed. He concluded that there is a condition of localised fluid collection around the cord, associated with 'an extremely local change in the pia-arachnoid. He named this meningitis serosa circumscripta spinalis (`spinal arachnoiditis)'. Most experienced neurosurgeons will have anecdotal knowledge of similar cases. French[5] described 13 cases of arachnoiditis among 200 laminectomies for disc. All had preoperative (first) myelograms using 'Pantopaque' confirming the diagnosis of arachnoiditis, and seven had a complete block. The diagnosis was confirmed at operation in all. It is not stated how many of the remaining patients had an intradural exploration ('intradural exploration infrequently performed'). Eight had disc protrusions, and five had a good result; five had no detected disc abnormality, and two of these obtained a good result. The clinical findings were indistinguishable in the two groups. This was probably the first paper to show that lumbar disc prolapse may be associated with localised arachnoiditis. He described a number of clinical, pathological and aetiological factors in spinal arachnoiditis and warned that: - `Failure to recognise these variants has resulted in fathomless confusion.' It would seem that this warning has gone largely unheeded.

Following the introduction of iophendylate ('Pantopaque', 'Myodil') myelography in 1944 by Steinhausen[6] it became recognised that *radiological* changes of arachnoiditis could follow myelography. The likelihood of this occurring was increased both with repeated myelograms, and with myelograms which were associated with the production of subarachnoid bleeding. Initially only 'Myodil' was implicated, but it is now known that all contrast agents have the potential for causing radiological arachnoiditis. Shaw et al.[7] set out the Glasgow experience between 1951 and 1976 when 80 patients were diagnosed with arachnoiditis. During this time they performed 7600 myelograms and 2900 lumbar disc explorations. They describe the changing disease pattern, from mainly

infective (1590 of total) in the earlier part of the series, to mainly lumbar disc disease (649c of total) in the later part. They concluded that usually several factors are involved in the genesis of arachnoiditis and `rarely does contrast medium alone produce spinal arachnoiditis'. Long' in 1992 contributed what was in many ways the definitive paper on the subject, with an extensive literature review, and a personal series totalling 321 cases among over 3500 chronic pain patients. This is by far the largest series in the literature. He commented that: 'Because of the multiplicity of procedures, it was impossible to determine the causative event in most patients.' In only one case did he consider arachnoiditis to be due to a myelogram alone. The diagnosis was confirmed by myelography in all cases. He stated: 'There was no consistent clinical pattern.' Lumbar spinal surgery and mechanical trauma, particularly when it is associated with subarachnoid bleeding, is a recognised cause of *radiologica*l and *pathological* arachnoiditis. Also, intrathecal medication with such agents as antibiotics and steroids have been implicated in the development of *radiological* and *pathological* arachnoiditis, although Wilkinson[9] has recently challenged the role of steroids in this context.

CLINICAL SYNDROME

In modern clinical practice we can now recognize and treat both tuberculosus and syphilitic arachnoiditis. Similarly, the rare confusing. The association described by Verbiest[12] and later by Epstein et al[13] was with developmental lumbar spinal stenosis in which the bony canal was contracted. Jackson and Isherwood[14] studying the association of degenerative lumbar spinal disease and chronic adhesive arachnoiditis, and Laitt et al.,[15] when studying patterns of chronic adhesive arachnoiditis following Myodil myelography, used the cross-sectional area of the theca measured on MRI as the criterion for spinal stenosis and made no reference to the dimensions of the bony canal. They described severe spinal stenosis as the theca having a cross-sectional area less than 0.7 cm^2 and moderate stenosis if the area was between 0.7 and 1 cm^2. They describe spinal stenosis as being pure (supposedly developmental), discogenic or spondylolisthetic. They also acknowledge that stenosis may be postoperative. As concentric shrinkage of the theca has been well documented as a manifestation of arachnoiditis, the association of spinal stenosis (using their criteria) with chronic adhesive arachnoiditis is open to challenge in that the spinal stenosis may have been in fact a manifestation of chronic adhesive arachnoiditis rather than an aetiological factor. Furthermore, these workers, using an 0.5 Tesla MRI unit stated that detail was insufficient for them to comment on the status of the nerve root sleeves. This is unfortunate, as patent nerve root sleeves would negate the suggestion that `pure' spinal stenosis was acquired as part of chronic adhesive arachnoiditis. Certainly, this is an important matter in respect to some of the conclusions in these two papers. Jackson and Isherwood, studying the relationship between degenerative disease and arachnoiditis, conclude that arachnoiditis-like changes extending over more than one vertebral level are rare (7%) except in the presence of spinal stenosis at multiple levels (29%). Laitt et al., studying chronic adhesive arachnoiditis patterns following Myodil myelography, conclude that `only a single case of arachnoiditic nerve root patterns was seen in the absence of stenosis or previous surgery. We conclude that chronic adhesive arachnoiditis is significantly related to previous Myodil myelography in the presence of spinal stenosis or previous surgery but that Myodil alone rarely produces these changes'. In each conclusion the question of whether thecal stenosis is acquired rather than developmental is crucial. The addition of contrast enhancement to CT and MRI studies has not added significantly to these techniques in chronic adhesive arachnoiditis. Increased vascularity associated with chronic adhesive arachnoiditis would be expected to result in increased contrast enhancement and that has been the finding.

With the availability of MRI to provide more detail of minor changes in the lumbosacral meninges one is led to the conclusion, supported by the voluminous literature of experimental, surgical and radiological information, that the introduction of virtually any foreign material into the subarachnoid space is likely to result in some degree of arachnoiditis. The radiological findings which follow range from no change, with complete resolution, to minor alterations consistent with adhesions resulting from fibrinous exudation, to more severe anatomical disturbances in some. In patients with more severe radiological changes following Myodil virtually always there is a history of previous surgery or evidence that bleeding occurred into the spinal theca. The radiological definition of spinal thecal stenosis remains unclear as does its relationship to MRI findings of chronic adhesive arachnoiditis. Chronic adhesive arachnoiditis resulting from degenerative disc disease in disc disease in the lumbar region is well documented, but only in respect to disc protrusion. The association with lesser degrees of lumbar disc disease, cross-sectional width of the lumbar theca and chronic adhesive arachnoiditis remains unclear.

CORRELATION OF RADIOLOGICAL EVIDENCE OF CHRONIC ADHESIVE ARACHNOIDITIS WITH SYMPTOMS

The radiological changes seen in chronic adhesive arachnoiditis as a result of fibrous exudation and organisation have shown no consistent correlation with the clinical findings except in those cases where nerve root (or occasionally spinal cord) atrophy has been demonstrated. Certainly the Delamarter patterns do not correlate with the clinical histories. Following his extensive review of the literature, Long summarised the situation: 'There is no doubt that all of the contrast agents that have been and are now employed cause a meningeal inflammatory reaction. What is not known is how significant these reactions are from a clinical standpoint, and if such contrast agent induced reactions are related to the clinical syndrome of chronic adhesive arachnoiditis.'

As regards the correlation of oily contrast media induced lumbosacral chronic adhesive arachnoiditis and clinical symptoms, useful information is available from experience with Myodil ventriculography and cervical myelography. Myodil ventricuiography was a commonly performed procedure until the advent of CT imaging. In this, 3 ml of Myodil were instilled into one lateral ventricle, and then manoeuvred through the third and fourth ventricles. It was then let fall to the lumbar region. never to be retrieved. The total number of such procedures can only be `guesstimated', but it would be of the order of tens of thousands worldwide. Hughes and Isherwood[16] presented 98 patients followed for a year or more, and Rowland Hill et al[17] presented 222 cases. Neither author could demonstrate any clinical case in which this procedure could be blamed for lumbar symptoms. Unfortunately, the opportunity to study with MRI the appearances in the lumbar theca in these patients has probably been lost as the method is now obsolete. Almost certainly the radiological changes of chronic adhesive arachnoiditis would have been found in this asymptomatic group.

Cervical myelography is a commonly performed procedure, and often a larger volume of contrast material is used, compared with lumbar myelography. The contrast, either oily or water soluble, is introduced cisternally or by the lumbar route. At the conclusion contrast gravitates to the lumbosacral region, and in many patients the oily contrast agent was not removed (the water soluble contrast is not usually removed). The present authors have no personal experience of arachnoiditis following cervical myelography. Long states: `There are only a few reports of patients undergoing cervical myelography who subsequently developed lumbar arachnoiditis - this in the face of millions of cervical myelograms' (he cites no references for these 'few reports' and includes no personal cases). A similar lack of correlation between radiological chronic adhesive arachnoiditis and clinical symptoms is seen following water soluble contrast myelography. Dullerud and Morland[18] reviewed 252 patients after Dimer X myelography. Fifteen patients who had previous Dimer X myelogram with DepoMedrol showed radiographic arachnoiditis. There was no correlation between the radiological diagnosis and the presence or absence of clinical symptoms. Irstram et al.[10] reviewed their experience with both Conray 60 and Methiodal (Kontrast U). They found radiological evidence of arachnoiditis in 8 of 19 patients using Kontrast U (no surgery), one of nine using Conray 60 (nosurgery), and similar figures if surgery was performed. No clinical correlation was found.

(Note: Page 389 of this paper is missing and will be inserted here when available)

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