

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. Horsley¹ is usually credited with priority, but Gowers² 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. Elkington⁴ reviewed the literature in 1936, 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. French⁵ 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 Steinhäuser,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 (15% of total) in the earlier part of the series, to mainly lumbar disc disease (64% 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. Longs⁸ 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 radiological 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 Wilkinson⁹ has recently challenged the role of steroids in this context.

CLINICAL SYNDROME

In modern clinical practice we can now recognise and treat both tuberculous and syphilitic arachnoiditis. Similarly, the rare

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condition of meningitis serosa circumscripta spinalis of Elkington can be satisfactorily diagnosed and managed, even though we still do not understand its pathogenesis. Beyond these specific diagnoses there exists a significant set of patients variously labelled as 'failed back syndrome', 'chronic low back pain', 'chronic pain syndrome (lumbar)', or any permutation of the words 'chronic - lumbar - spinal - adhesive - arachnoiditis'. These are overlapping and not mutually exclusive labels - they should not be dignified with the epithet *diagnosis*. All authors are agreed that there is no specific *clinical syndrome* within this group to which the *pathological diagnosis* of arachnoiditis can be associated.

The clinical picture in any of this set of patients is almost always the presentation following a back injury leading to back and/or leg pain. Investigation including myelography (often multiple) is followed by laminectomy (often multiple), and sometimes fusion of one sort or another. They either improve transiently, or fail to improve at all. These patients place a heavy diagnostic and therapeutic burden on the treating practitioner. Their myelograms, and more recently their magnetic resonance (NIR) images will commonly show changes consistent with the *radiological diagnosis* of arachnoiditis. It is, therefore, very tempting to *label* these patients as *diagnosed* with 'arachnoiditis' (which they have from the radiological point of view), and thus imply that there is clinical arachnoiditis which is the cause of their continuing symptoms.

RADIOLOGICAL APPEARANCES OF CHRONIC ADHESIVE ARACHNOIDITIS

Apart from direct inspection at surgery, radiology has been the means of providing objective evidence of chronic adhesive arachnoiditis in the living since the condition was described almost a century ago. Over this period radiological modalities have evolved providing ever increasing detail of the spinal meninges and adjacent structures. Plain radiography led to myelography in the 1920s, followed by computed tomography (CT) in the 1970s and MRI in the 1980s. Although arachnoiditis calcificans and ossificans have been described as end results of arachnoiditis in some, generally this is not detected by plain radiography. Myelography allowed for assessment of the contours of the subarachnoid space, the roots of the cauda equina to some extent and abnormal distribution and flow of contrast. Cross-sectional imaging with CT, particularly following myelography, and MRI provided more detail of the thecal sac, the root sleeves, and the components of the cauda equina. For radiological changes to be evident in patients with arachnoiditis it is necessary for the fibrinous exudate to result in adhesion between the subarachnoid membrane and contained nerve roots or between the roots themselves, or for fibrosis and scarring to result in a detectable degree of distortion of anatomical structures. From the accumulated literature it seems that a range of appearances may be seen in chronic adhesive arachnoiditis according to the severity of the condition.

No demonstrable radiological' changes

Irritation of the arachnoid results in an inflammatory response which may extend to adjacent tissues. If this process resolves without anatomical distortion radiological appearances will remain within normal limits.

Minor radiological' changes

Water soluble myelography contrast media have the advantage of outlining the extent of the lumbosacral nerve root sleeves to a much greater degree than iophendylate, which is more viscous.IRSTAM et al." performed repeat myelography on 48 patients who

had undergone initial myelography using one of two of the earlier water soluble media, namely, Methiodal (Kontrast U) and methylglutamine iothalamate (Conray 60). They found that about one third of the patients showed minor changes consistent with adhesive arachnoiditis and these changes involved the nerve root sleeves or the lumbosacral cul de sac of the spinal theca. In these cases they found that root sleeves and any associated root sleeve cysts filled to a lesser extent than initially and this was the only difference in appearances between the two examinations for the majority. A small number showed, in addition, some reduction in length and width of the lumbosacral arachnoid cul de sac. The distribution of the changes was consistent with those sites being exposed to the highest concentration of the water soluble medium which was absorbed relatively quickly.

CT myelography and NIRI provide good differentiation of soft tissue densities and this combined with the multiplanar potential of MRI have added to our knowledge of changes which occur with chronic adhesive arachnoiditis. Delamarter et al." described three categories of NIRI appearances consistent with chronic adhesive arachnoiditis. Groups 1 and 2 were considered mild and consistent with adhesions developing between nerve roots and the parietal arachnoid. In Group 1 the adherent nerve roots were seen centrally within the cerebrospinal fluid and in Group 2 the nerve roots were adherent peripherally to the theca giving an appearance of an empty thecal sac. Correlation with plain myelography and CT myelography showed similar findings, although less well demonstrated than with MRI. Myelography showed nerve root sleeve changes in all of these patients and in Group 1 cases some showed minor contraction or irregularity of the thecal cul de sac, which was not seen in Group 2 patients. Delamarter's Group 3 category was of a more severe nature (see below).

It seems that the earliest change of chronic adhesive arachnoiditis to be seen radiologically is arachnoid adhesion in the nerve root sleeves and within the theca between adjacent roots of the cauda equina or between roots and the parietal arachnoid or a combination of these. Distortion of the lumbosacral cul de sac appears to be the next stage indicating a degree of concentric fibrotic contraction of the theca.

Major radiological changes

Group 3 MRI appearances, as described by Delamarter et al., show a considerably reduced cross-sectional area of the thecal sac which was completely filled by soft tissue consistent with clumped or matted nerve roots. Correlation with myelography confirmed a high level of obstruction at the level of the abnormality with considerable irregularity of the margins of the outlined subarachnoid space extending above and below the level of the Group 3 lesion. This appearance, apart from being consistent with the adhesion of nerve roots, indicates marked concentric contraction in the arachnoid and surrounding dura. These more severe changes of adhesive arachnoiditis were well recognised prior to the introduction of water soluble contrast media, CT and NIRI and were seen at various levels within the spinal canal. Described changes consistent with established chronic adhesive arachnoiditis were irregularity and narrowing of the subarachnoid space on myelography, obliteration of nerve root sleeves, apparent thickening of nerve roots, irregular distribution of introduced contrast medium with loculation and cyst formation and impaired mobility of introduced contrast. Partial or complete blockage and in some at higher levels even spinal cord atrophy were end results of the process.

Associations between radiological chronic adhesive arachnoiditis and degenerative disc disease, previous surgery, and spinal stenosis have been identified and documented. The literature concerning the association with spinal stenosis is

confusing. The association described by Verbiest¹ and later by Epstein et al.² was with developmental lumbar spinal stenosis in which the bony canal was contracted. Jackson and Isherwood³ studying the association of degenerative lumbar spinal disease and chronic adhesive arachnoiditis, and Laitt et al.⁴ 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 cm² and moderate stenosis if the area was between 0.7 and 1 cm². They describe spinal stenosis as being pure (supposedly developmental), discogenic or spondyloarthrotic. 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 arachnoiditis 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 the lumbar region is well documented, but only in respect to disc protrusion. The association of lesser degrees of lumbar disc disease, cross-sectional width of the spinal 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, Long⁵ 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 ventriculography 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 Isherwood³ presented 98 patients followed for a year or more, and Rowland Hill et al.⁶ 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 Morland⁷ 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.⁸ reviewed their experience with both Conray 60 and Nlethiodal (Kontrast U). They found radiological evidence of arachnoiditis in 8 of 19 patients using Kontrast U (no surgery), one of nine using Conray 60 (no surgery), and similar figures if surgery was performed. No clinical correlation was provided. Mooij⁹ reviewed 63 patients with a radiological diagnosis of arachnoiditis and concluded that if 'lumbosacral arachnoiditis is a coincidental finding in the majority of cases, without clinical consequences'.

In summary, lumbar myelography with any agent may cause the radiological changes of arachnoiditis, but no author has demonstrated any causative relationship between the radiological changes and clinical symptoms. Rather it is noted that if both the radiological changes and the symptoms are present, a causal relationship is implied, but not explicitly stated.

TREATMENT

First we must decide the aim of treatment, and then what it is that we are treating. Clearly the aim of treatment is the relief of symptoms, that is, pain. Also there may be neurological deficits which it is hoped to improve. But what are we treating? All authors agree that some patients may be relieved of their 'arachnoiditis' symptoms by relieving nerve root or cauda equina compression, or by treating spinal instability. This is treating associated pathology and is often effective, but clearly it is not treating arachnoiditis, and thus these patients were, in retrospect, not suffering from symptomatic arachnoiditis. Long describes his experience with microlysis of adhesions, and concluded that 'the operation was too dangerous to carry out for pain except in the most desperate of circumstances'. Wilkinson reviewed the use of intrathecal DepoMedrol and considered it to be safe, and often effective in relieving symptoms of chronic back pain and/or sciatica. This has been the experience of the present authors also. However, the drug is not recommended for this use by the manufacturers (because of problems with litigation). Patients no longer can have the benefit of this often helpful treatment, because of the threat of litigation hanging over the use of intrathecal steroids. The treatment is, therefore, that of any chronic pain syndrome, once all treatable causes have been resolved or excluded.

DISCUSSION

Many authors (including our own experience) show that radiological arachnoiditis can be present in the absence of symptoms. All recent authors (French, Shaw, Long for example), as well as personal experience show that in a few patients diagnosed with arachnoiditis it is possible to relieve symptoms with appropriate surgical procedures, such as nerve root or cauda equina decompression. It is, therefore, clear in these patients at least, that while they have arachnoiditis, it is not responsible for their symptoms. If we accept these clinical facts, it then becomes difficult to sustain the belief that arachnoiditis is the underlying cause of symptoms in the majority.

Summarising all the above information, we can arrive at the following conclusions:

1. Arachnoiditis is a clearly defined pathological entity.
2. Arachnoiditis is a clearly defined radiological entity.
3. Arachnoiditis is NOT a clearly defined clinical entity.
4. Lumbar disc prolapse may be associated with the pathological and radiological changes of arachnoiditis locally.
5. Lumbar myelography may be followed by the pathological and radiological changes of arachnoiditis.
6. The pathological and radiological changes of arachnoiditis may be present in the absence of symptoms.
7. Cervical myelography is rarely if ever followed by clinical arachnoiditis.
8. Myodil ventriculography has never been shown to be followed by clinical arachnoiditis.
9. Virtually all patients with the diagnosis of lumbar arachnoiditis have several other problems in their lumbar region, and one or more of these problems are usually sufficient to explain their symptoms.

O.A minority of patients with the diagnosis of arachnoiditis may have their symptoms relieved by surgical decompression of nerve roots, suggesting a mechanical cause for the symptoms in those patients.

Many previous authors seem to have accepted without question that lumbar spinal arachnoiditis is a cause of symptoms. When faced with the facts outlined above, which are accepted by these same authors, we have to ask why? There is no clearly defined clinical syndrome, almost all patients have sufficient other explanations for their pain, and patients can be seen with the radiological changes, but no symptoms.

So why has this confusion arisen? Part of the explanation lies in the gradual shift in the pathogenesis over the years. In the early part of the century and in other cultures, syphilitic and tuberculous arachnoiditis were undoubtedly genuine causes of symptoms, usually leading to death from the systemic complications of a spastic tetraparesis. There is 'meningitis serosa circumscripta spinalis (spinal arachnoiditis)' of Elkington. This rare condition does cause symptoms, and continues to puzzle, to this day. Shaw et al. then documented the shift away from infective to lumbar spine degenerative causes in the pattern of patients seen, and assumed that arachnoiditis, if present, was symptomatic in all groups of patients, because it clearly was so with the infective patients. Also French assumed (despite his own evidence) that arachnoiditis was a cause of symptoms in his patients. With these eminent authorities proclaiming this nexus, it is little wonder that subsequent authors have followed suit. The intellectual basis for making the correlation between the clinical and the radiological syndromes is syllogistic, of the form:

- My patient has back pain and radiological arachnoiditis
- Back pain has been associated with clinical arachnoiditis
- Therefore, my patient's back pain must be caused by clinical arachnoiditis.

Which is as logical as:

- All dogs have four legs
- My cat has four legs
- Therefore, my cat is a dog.

The ancient Greek philosophers would turn in their urns!

CONCLUSION

There is no rational basis for the belief that the radiological and pathological changes of lumbar spinal arachnoiditis are correlated with clinical symptoms, except in the most rare of circumstance. Every effort should be made to find some underlying structural or functional cause for the symptoms and treat the patient accordingly, because the diagnosis of 'clinical arachnoiditis' is essentially a diagnosis of despair or a justification for otherwise unsustainable litigation.

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