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INTRODUCTION

Those neurologists who perform medicolegal work are unlikely to have overlooked the intense debate that has raged in recent years in legal circles regarding the possible association between multiple sclerosis and trauma. Some high profile legal cases have taken place and reports on these have, from time-to-time, found their way into the national UK press. It might be assumed that the decisions made in law courts in the various parts of the British Isles would be able to draw on a wealth of previous cases that have addressed this issue. Rather surprisingly, there is relatively limited established legal data addressing this issue and the same few cases crop up frequently in discussion. Neurologists might be interested to hear a little of the nature of the cases that provide the background for current legal judgements.

THE ISSUES

I would probably agree with our colleagues in the legal profession that it is inadequate simply to pose the question: ‘does trauma cause multiple sclerosis?’ Whilst this question would be an interesting point at which to start a discussion, it is clear that a far more complex set of questions needs to be considered, both by lawyers and by the expert witnesses who advise them.

The question can readily be expanded to ask: ‘can any type of trauma cause multiple sclerosis in any individual?’, or is it just the case that ‘any type of trauma may cause multiple sclerosis in a person who has some pre-existing tendency to the development of the disease?’ In recent legal judgements, the concept of an individual with some pre-existing tendency to multiple sclerosis has been expanded and such individuals are referred to as ‘potential demyelinators’.

Lawyers will consider these questions yet further, almost certainly wishing to pose some follow-on questions: ‘if it should be found that diffuse trauma to a body cannot give rise to multiple sclerosis, then is it possible instead that trauma directly to the central nervous system may cause multiple sclerosis?’, ‘if it should be considered that no sort of trauma can initiate multiple sclerosis as a new entity, then is it possible instead that trauma may cause the aggravation of an established diagnosis of multiple sclerosis, or can it accelerate the progress of MS in a given individual?’ Many other subquestions arise: in particular, simple queries such as, ‘how severe should the trauma be’, and ‘how soon after the experience of trauma would it be expected that an individual develop multiple sclerosis if the trauma were itself the cause?’

None of these questions is easy to answer. Such questions might be answered more readily if we better understood the cause of multiple sclerosis, but in the absence of a comprehensive understanding of the pathological sequence of events that gives rise to multiple sclerosis in a sufferer, then it becomes extraordinarily difficult to provide legal colleagues with unequivocal answers to their questions. Even when we are reminded by members of the legal profession that, in their eyes, to establish ‘proof’ - at least in a civil case - it is only necessary to show that something is ‘more likely than not on the balance of probability’, i.e. if it can be agreed that something is 51% likely then that may be determined to be ‘proof’, many of us would still feel that we lack adequate data to answer these questions robustly.
THE CASES
The lawyers who were charged with dealing with the recent high profile case of ‘Dingley v Chief Constable, Strathclyde Police’ (1998 S.C. 548) (discussed in greater detail below), had very little in the way of previous legal cases to assist them in their decision.

The O’Leary case
There was a case heard in Ireland (Niall O’Leary v Cork Corporation) in 1993. O’Leary suffered a works accident in mid-1985 and by mid-1986 had been diagnosed as suffering from multiple sclerosis. It was suggested that his accident had given rise to his MS and, writing in judgement, it was held that although it was possible for an accident to trigger multiple sclerosis, the judge considered that O’Leary had not adequately proved his case that his MS was the direct result of that accident.

This case went to Appeal in Ireland, the judgement being delivered during 1997, and the Appeal Court correctly separated two key issues as follows: first, whether there is, or may possibly be, a relationship between trauma and the onset or aggravation of symptoms of MS; and, secondly, whether the injuries sustained by the victim in this case were the causative factor in his developing MS. The Appeal Court had the advantage of the work by Sibley and others (Sibley et al. 1991), which was a prospective study of physical trauma and multiple sclerosis, and felt that the original judgement should be reviewed in the light of that scientific paper. The Supreme Court in Ireland therefore held that the original decision was correct in determining that the nature of O’Leary’s trauma was not the likely cause for his development of MS.

The Dingley case
The Dingley case (Dingley v The Chief Constable, Strathclyde Police) (1998 S.C. 548) concerned a young police officer who was involved in a road traffic accident when a police van overturned near Glasgow. Some months later Dingley developed multiple sclerosis. He sued the Strathclyde Police Force asserting that were it not for the road accident, which took place in a police vehicle, he would not have developed multiple sclerosis.

The case was heard in the Court of Session in Edinburgh before Lord Dawson, the judge of the first instance. He found, on the balance of probability, that Dingley’s multiple sclerosis had been caused by the road accident and very generous damages were awarded. This decision caused some surprise both in neurological and in legal circles and it was considered that an Appeal was justified on the grounds that Lord Dawson had not adequately analysed and weighed the expert evidence that was offered. Three Scottish Appeal Judges then reconsidered the case and reached three slightly different views on the subject, albeit with the same eventual outcome. The Lord President determined that in his view trauma could never trigger multiple sclerosis even in individuals that might be somehow predisposed. The Lord Prosser took the same view but with the proviso that if the evidence which was considered had shown that MS could be triggered by direct trauma to the neck, then it might have been reasonable in this case to assume that Dingley’s neck trauma had given rise to his MS. But the evidence had not shown that, so the second Law Lord found no link. The Lord Caplan determined that trauma might trigger multiple sclerosis in individuals with some form of susceptibility, but it did not do so in the case of Dingley.

This case was then taken to the House of Lords and the verdict of their Lordships was exactly in keeping with the decision made in the Scottish Appeal (2000 S.C. 77). Accordingly, it was determined that Constable Dingley’s multiple sclerosis was not caused by the trauma of the road accident and the damages awarded were reduced very dramatically.

The Nixon case
The most recent case that has considered this issue is that of Nixon v F.J. Morris Contracting Ltd (2000 All ER (D) 2418). In 1992 Nixon had the misfortune to be driving near Milton Keynes when contractors allowed a lamp standard to fall on to his car. Following the accident he complained of dizziness and dysequilibrium. More than two years later he was referred to a neurologist who made the diagnosis of multiple sclerosis. In this case, much of the evidence given by expert witnesses focused on whether trauma at particular sites within the central nervous system might give rise to the development of multiple sclerosis. In particular, there was lengthy discussion as to whether a severe whiplash injury might cause damage to the blood brain barrier in the region of the cervical spine, and if it could then be shown that initial demyelination took place within the cervical spinal cord, whether this might mean that the MS was indeed due to the trauma. This attempt to correlate the anatomical site of injury with local damage to the blood brain barrier, and
subsequent localized development of demyelination, was a feature not considered in such great detail in the previous cases.

Writing in judgement, Mr Justice Garland concluded that he was ‘satisfied on balance of probability that trauma to the spinal cord can, if it causes an alteration of the blood brain barrier, also provoke the symptoms of multiple sclerosis, both in those who were previously asymptomatic and in those who had symptoms (of MS) in the past, whether or not these were in remission at the time of the trauma’. This interesting comment was to a great extent at variance with the decision made by the House of Lords a few years previously. Perhaps frustratingly for Mr Nixon, however, the judge determined that from his understanding of the nature and forces involved in this particular road accident, a whiplash injury had not been sustained and, accordingly, the case failed.

This case is likely to be the subject of further discussion, as it is the first occasion on which a judge has determined that trauma can provoke multiple sclerosis, albeit in rare and exceptional cases but that such cases might be found when the trauma has been directly to the ‘more vulnerable’ cervical spinal cord. Mr Justice Garland accepted that his judgement might well provoke reconsideration of this issue at a higher level in the future.

THE NEUROLOGISTS’ VIEW

Putting to one side the deliberations of the legal profession, we have to accept that neurologists are divided on this issue. Some well-known names in the field of multiple sclerosis have very strongly held, almost polarized, views, whereas others keep an open mind. The advocates of trauma being a cause for multiple sclerosis argue that relatively minor trauma to the central nervous system may cause disruption of the blood brain barrier and therefore invite the onset of an unwanted immunological cascade that will give rise to the demyelination of multiple sclerosis. Reference is often made to the observations of Gonsette and others (Gonsette et al. 1966) who observed that after needle thalamotomies on patients with multiple sclerosis being done therapeutically to control tremor, new areas of demyelination formed adjacent to the needle tract at several levels. The advocates in favour of the trauma and MS argument suggest that there is robust clinical, pathological and neuroradiological evidence to back-up their assertion (Poser 2000).

In contrast, those neurologists who are unpersuaded that there is a causal link between trauma and multiple sclerosis, stress the need for appropriate prospective studies and draw on the only high-quality evidence available to us, i.e. the study by Sibley and colleagues that observed 170 patients with MS for up to eight years (Sibley et al. 1991). No significant correlation was found between mechanical trauma and MS exacerbations or progression. They make the point that the explanation for the development of, or the deterioration of MS after trauma, is more likely to be explained by coincidence, or by the physical and psychological effects of trauma rather than any direct neuro-pathological link (Cook 2000). This side of the argument has been given strong support in a recent consensus paper, written by North American colleagues (Goodin et al. 1999).

CONCLUSION

As clinical neurologists we have a responsibility to look critically at evidence presented on both sides of this argument. We also need to consider the quality of that evidence. We are often reminded that the ‘plural of anecdote is not data’. Until the advocates of the ‘trauma causes MS’ theory produce adequate data to balance their assertions, then we would be well advised to place more weight on the best epidemiological data currently available to us which have, up to this point, failed to demonstrate any link between trauma and the development or exacerbation of multiple sclerosis.

REFERENCES

2000 S.C. 77
Niall O’Leary v. Cork Corporation (Murphy, J, 19 May 1993).
Poser CM (2000) Trauma to the central nervous system may result in formation or enlargement of multiple sclerosis plaques. Archives of Neurology, 57, 1074–6.