Intermittent claudication and lateral lumbar disc protrusions

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The term 'intermittent claudication', which really means intermittent limping, is usually applied to a clinical condition due to vascular insufficiency in the lower limbs. There have been reports of lesions of the spinal cord or of the cauda equina causing similar symptoms. Déjerine (1911) described two cases of lesions in the spinal cord producing pain in the legs and limping, and he called the condition intermittent claudication of the spinal cord. Blau and Logue (1961) reported the case histories of six patients who experienced similar disturbances which were found to be due to central protrusions of lumbar intervertebral discs or spondylotic bars. They called this syndrome intermittent claudication of the cauda equina. Brish, Lerner, and Braham (1964) described a similar clinical syndrome associated with narrowing of the lumbar spinal canal following spondylotic changes.

Although it is well known that patients with lateral lumbar disc protrusions may experience an increase in crural pain after walking, limping and pain in one leg after walking a constant distance as the presenting or predominant symptom of a disc protrusion is very rare. We have been unable to find any reports in the literature. The purpose of this paper is to present three such cases and to discuss the mechanism for the syndrome.

CASE REPORTS

CASE 1 Mr. L.E., aged 57, for six months had been experiencing discomfort in the right foot after standing for a few minutes. Over the same period he noticed that after walking a quarter of a mile an aching pain would develop in the right calf and he then found himself limping. He had to stand still for five minutes for this pain to wear off before he could resume his walk. Gradually the distance he was able to walk before the pain came on and forced him to stop was reduced to a hundred yards. At this stage he was referred to a vascular surgeon who could find no evidence of vascular insufficiency in the lower limbs. Aorto-arteriography was performed and no abnormality was demonstrated. When he was referred to the neurosurgical unit he could only

walk about 50 yards before developing pain and a limp. He never had pain in the leg when at rest. Coughing and sneezing caused no discomfort. In the history taking it was now elicited for the first time that four months before the onset of his symptoms he had had a fall, which was followed by intermittent low back pain which he had ignored.

Examination In the spine there was only minimal loss of the lumbar lordosis. On bending forwards his fingers were 6 in. from his toes. Straight leg raising was 90° on

FIG. 1. Myelogram showing small disc protrusion at the L5-S1 interval on the right side.

the left, and 85° on the right. There was a very slight degree of hypotonia of the right glutei and hamstring muscles, and a slight reduction of the right ankle jerk. There was no sensory loss. The patient was then asked to walk as far as he could. After covering 80 yards he complained of pain in his leg and developed a limp; 20 yards further on the pain was so severe he had to stop. It was now noted that straight leg raising was reduced to 70° in the right leg. The right glutei had become quite atonic and the patient was now unable to contract them. The right ankle jerk had disappeared. Five minutes later when the pain had gone, tone returned to the right glutei, and the ankle jerk reappeared.

Investigations Plain radiographs of the lumbar spine showed no abnormality. The cerebrospinal fluid obtained at lumbar puncture contained 160 mg. of protein per 100 ml. Myelography revealed the presence of a small laterally placed disc protrusion at the L5-S1 interval on the right side (Fig. 1).

Treatment An operation was performed and the last intervertebral interval explored on the right side. There was a laterally placed disc protrusion under the first sacral root, which however, was neither tense nor displaced. It was found that the summit of the protrusion, which was rather less than 5 mm. in height, was soft and it could be partially flattened with an instrument. The protrusion was excised and the interval cleared of as much disc tissue as possible. This was of a softer consistency than usual, and rather granular. The weight of the tissue excised was 3 g. At microscopy it showed features of soft mucoid cartilage.

Progress The patient was discharged home two and a half weeks after operation, able to touch his toes and to walk any distance. He remains symptom free 24 months later.

CASE 2 Mr. J.W., aged 59, six months previously, while dressing, suddenly developed pain in the bottom of the spine, which spread down the back of the right leg. After two weeks all the pain disappeared, and he returned to work. A few weeks later he noticed that after walking for 200 yards he would develop an ache in the right calf which caused him to limp. If he reduced the speed of walking the ache would be less severe, and if he stopped still the ache would go completely after about three minutes. Running would bring on the pain after covering a much shorter distance. When the calf ached, his right ankle felt weak.

Examination The patient was a fairly obese man. There was no spinal deformity, but forward flexion was restricted so that his fingers could approach no closer than 15 in. from his toes. Straight leg raising was 90° on the left, and 85° on the right side. There was slight loss of tone in the right glutei and hamstrings. No weakness was detected in any part of the right leg, and there was no sensory loss. The right ankle jerk was absent. The pulses in the lower limbs were present and equal. Oscillometry was carried out and showed no evidence of obliterative vascular disease. After the patient walked 200 yards to bring on the pain the only change noted in the physical signs was a slight decrease in straight leg raising. No weakness was detected at the ankle.

Investigations Radiographs of the lumbar spine showed narrowing at the L5-S1 interval, and myelography the presence of a small disc protrusion at this level. The protein content of the cerebrospinal fluid was 60 mg. per 100 ml.

Treatment The last intervertebral interval was explored on the right side. The antero-posterior diameter of the spinal canal was thought to be wider than usual. The first sacral nerve root was not tense or compressed, but was slightly elevated over a small disc protrusion which felt quite soft when pressed upon with a pair of forceps. The protrusion was excised and as much disc tissue as possible removed from the interval. The protruded part of the disc and that below the protrusion was degenerate, but tissue of the consistency usually encountered was removed from deeper in the interval. The weight of the disc tissue excised was 1.7 g. and histological examination showed degenerative changes only. The patient made a full recovery and could walk any distance without pain.

CASE 3 Mr. J.J.B., aged 35, for seven years developed pain in the outer aspect of the left calf after walking a quarter of a mile. He would then have to stand still for a few minutes for the pain to pass and before he could resume his walk. When the crural pain appeared he also noticed a slight ache at the bottom of the back. The distance he could walk gradually became shorter, so that at the time we saw him he could only walk 100 yards before he felt the pain and found himself limping. Coughing aggravated his symptoms. He also experienced some discomfort in the calf and buttock after getting out of bed; this lasted for five minutes. He smoked about 60 cigarettes daily.

Examination The patient was a thickset, obese man, rather old for his years. The appearance and temperature of the skin in the lower limbs were normal. The peripheral pulses were of good volume and oscillometry was also found to be normal. There was loss of the lumbar lordosis, with a scoliosis convex towards the left side. Forward flexion was restricted so that his fingers were 10 in. from his toes. Straight leg raising was 75° on the right side and 50° on the left. The left glutei were hypotonic, but there was no muscular weakness. There was no sensory loss and no reflex disturbance. After walking until he felt pain, forward flexion was reduced by a further 10 in., and straight leg raising was reduced to 40° on the left. There was no change in the neurological signs, except that the left glutei were thought to be more hypotonic.

Investigations A radiograph of the lumbar spine confirmed the scoliosis, which was of long standing; in the antero-posterior view there was wedging of the third, fourth, and fifth vertebral bodies. The protein content of the cerebrospinal fluid was normal, and myelography showed incomplete filling of the left first sacral root.

Treatment The last intervertebral interval was explored on the left side. The posterior common ligament was roughened and there were light adhesions between it and the first sacral root sheath. There was a small localized protrusion no more than 2 or 3 mm. in height, in the centre of a fairly broad disc. The protrusion was abnormally yielding to pressure from an instrument and

it consisted of degenerate tissue. More normal disc tissue was removed from further inside the interval. The disc material excised weighed 3 g. and on histological examination was found to consist of mucoid fibrocartilage.

Progress Progress was slow at first but after four weeks the patient was able to walk for long distances without experiencing pain. Forward flexion was still restricted by 6 in., and straight leg raising to 80°. It was thought that the structural changes in the spine seen radiologically prevented the recovery of a full range of movement, although he was now free of pain all the time.

SYMPTOMS

The chief symptoms of the patients whose cases we have described were those of pain in one calf and limping after walking certain distances. The pain became progressively worse the further they walked, until it became so severe that they had to stop and stand still or sit down. The pain would pass in about five minutes. The distance that the patients could walk before experiencing pain lessened during the course of the illness. These features are identical to those of intermittent claudication due to vascular disease in a lower limb. Vascular insufficiency had been suspected as the cause of the symptoms in each of our cases. When sought for, features suggestive of intervertebral disc disease were, however, elicited. One patient (Mr. J.W.) gave a previous history of a typical lumbar disc protrusion but the characteristic symptoms of this condition passed off before he developed intermittent claudication. Mr. L.E. admitted to having had a fall and experiencing slight low backache only on direct questioning. Mr. J.J.B. suffered from pain in the calf and limping as his only symptoms for seven years. Shortly before admission to the Neurosurgical Unit the pain radiated up to the gluteal region and to the low back from the calf after he had been walking only 110 yards; coughing would then aggravate pain.

PHYSICAL SIGNS

The classical signs of lumbar disc protrusions have been classified into three groups by O'Connell (1943). These are spinal, tension, and neurological. The severity of a patient's pain and therefore the indication for operation may be gauged by the degree of spinal deformity and restriction of movement, and the tension signs, which include a marked reduction of straight leg raising. The most conspicuous feature in two of our cases (L.E. and J.W.) was the almost complete absence of any of these signs so that the presence of the disc protrusion could well be overlooked. The third patient (J.J.B.), who did demonstrate signs sufficient to warrant operation without

myelography, also suffered from some slight pain at times other than after walking. One of the patients in whom there were almost no abnormal findings (L.E.) did, however, demonstrate tension and neurological signs perhaps sufficient to warrant exploration but only after he had walked far enough to experience pain and claudication. Mr. J.W. also showed a mild reduction of straight leg raising when he was in pain after walking.

INVESTIGATIONS

None of these patients showed clinical evidence of peripheral vascular disease. Oscillometry was carried out in each case and was found to be normal. Aortoarteriography was performed in one patient only, and was also normal. Plain radiographs of the lumbar spine were normal in one case, showed narrowing at the last intervertebral interval in another, and in the third there was a long-standing scoliosis with wedging of the bodies and osteophyte formation at the last three intervals. Myelography was carried out in each case, as the history and physical signs were by no means typical of lateral lumbar disc protrusions. It confirmed the diagnosis in the first two patients and suggested it in the third. The protein level in the cerebrospinal fluid was only found to be significantly raised in one case.

OPERATIVE FINDINGS

The appearances at operation were similar in the three cases. The nerve root at the level of the protrusion was neither stretched, tense, nor compressed. This is quite contrary to the findings in patients operated on for classical disc protrusions where the nerve root is always stretched over the disc and sometimes compressed against the overlying lamina or ligamentum flavum. The affected nerve root in our three patients was just making contact with the summit of the protrusion. There were light vascular adhesions between the nerve root sheath and the posterior common ligament in one case. In each case the posterior common ligament was intact and had not been perforated by the disc. The protrusion was localized and less than 0.5 cm. in height. The conspicuous feature was that the summit could readily be indented by an instrument so the protrusion could almost completely be reduced; when the pressure was removed the disc would protrude again. This state of affairs is never found in patients presenting with the classical clinical picture of lumbar disc protrusions. The protruded and subjacent disc tissue was granular and very degenerate, and this would account for the ease in the 'reduction' of the protrusion when it was compressed with an instru-

ment. The disc material from deeper in the interspace was of more normal consistency. Microscopy showed degenerative changes in each case.

DISCUSSION

In a typical case of sciatica due to a lateral lumbar disc protrusion, the patient usually experiences constant pain in the leg which is often aggravated by walking. In our cases the patients felt no pain in the leg until they had walked certain distances. They then had to rest for a few minutes until the pain passed. At operation the disc protrusion is usually found to be hard and firm and it is not reducible by pressure on its summit. In the three cases we have described the protrusion was fairly small, soft, and partially reducible. It was never large enough when seen at operation to cause tension of the overlying nerve root, and was composed of degenerate and granular material. We believe that when our patients walked, with each weight-bearing step there was a repetitive increase in pressure on the disc, which because of its consistency gradually herniated further into the spinal canal. A critical point was reached when the overlying nerve root became stretched. At this stage nerve root tension would occur and pain in the sciatic distribution be experienced. This would account for the appearance of tension signs, or diminution of straight leg raising, after the patient had walked a specific distance and developed pain in the leg.

With each step taken in walking there is a repetition of Laségue's manoeuvre, or straight leg raising. In a healthy adult the two legs may be separated by as much as 40°. When the Laségue test is performed there is a downward movement of the lumbar extradural roots by several millimetres (O'Connell, 1943, 1946) which will result in increased tension of a nerve root overlying a disc protrusion. Although the degree of straight leg raising occurring with each step in walking is not great, nevertheless the friction from repeated downward movement of a nerve root over a disc protrusion, which may be increasing in size during the course of a walk, could also be responsible for the production of pain.

Blau and Rushworth (1958) have shown in animal experiments that hyperaemia occurs in the nerve roots supplying a limb in response to vigorous exercise. Blau and Logue (1961) described six cases of central lumbar disc protrusions when bilateral crural pain was felt after walking certain specific distances. They attributed the constancy of the distance walked before pain occurred to the physiological vascular congestion in the nerve roots which were at the same time stretched or compressed over the disc protrusion or spondylotic bar. They also

thought that the physiological hyperaemia occurring with exercise may be impaired as a result of constriction of the blood vessels in the nerve root caused by oedema or compression from a disc protrusion. Symptoms of ischaemic neuritis would then follow. It has also been shown by Denny-Brown and Brenner (1944) that compression of a peripheral nerve results in impaired conduction through the segment compressed as a result of ischaemia rather than pressure on the individual nerve fibres. Angulation would further increase the delay in nerve conduction. If a lumbar nerve root is becoming tense and angulated over a disc protrusion increasing in size, then it would be reasonable to accept an ischaemic cause for the transitory neurological signs, particularly in case 1.

In normal walking the energy put out by the appropriate lower limb muscles is relatively small, and is hardly to be compared with the vigorous exercise performed by mice in the experiments of Blau and Rushworth (1958). In these a mouse had to lift a 10 g. weight against gravity with one leg, every two seconds, from a half to two hours. We think it is unlikely that the muscle activity during normal walking will result in such physiological hyperaemia in the roots of the cauda equina as in the animal experiments. It seems therefore unlikely that this vascular factor could be responsible for the production of symptoms whether there is a centrally or laterally placed disc protrusion. We believe that in our patients the following sequence of events would explain the symptoms. Due to the unusually soft consistency of the disc tissue at the diseased level, with each weight-bearing step the protrusion gradually increases in size. A critical point will be reached when the overlying nerve root becomes stretched and tense. As the tension increases, so the patient will experience increasing pain. Also the repeated movement of the affected nerve root over the protrusion occurring with each step will cause friction of the nerve within its sheath. This could result in congestion of the vessels, oedema, or interference with the normal blood circulation within the nerve root. Angulation of the root over the summit of the protrusion would result in impaired conduction through the nerve with a transient change in the neurological signs such as hypotonia, weakness, sensory, or reflex disturbance. However, the most important factor in the production of the symptoms and signs is probably mechanical. When the critical size of the protrusion is reached, the nerve root will become tense and subject to repeated friction; pain in the leg will be experienced, and there will be a reduction of straight leg raising, and transient slight neurological signs will be found, such as hypotonia of the glutei or the loss of the Achilles reflex.

If the patient stands still, then the nerve will no longer be subject to friction, and there may be some reduction in the size of the protrusion. The pain will then fade and the neurological signs regress.

SUMMARY

Three cases have been described of intermittent claudication due to pain in one leg, when the history was strongly suggestive of vascular insufficiency to the affected lower limb. In each case the cause of the symptoms was found to be due to a laterally placed lumbar disc protrusion. The underlying pathology remained unsuspected in two of these until the patient had been made to walk sufficiently far to feel pain, when the physical signs of a lumbar disc protrusion appeared. In the third patient the existing signs were increased. Myelography revealed the diagnosis in each case, and there was symptomatic relief following excision of the diseased disc. The disc protrusions were all of a most unusual consistency, in that they were soft and reducible by pressure over the summit. The disc material itself was granular, degenerate, and soft. The cause of the

syndrome is thought to be largely mechanical, in so far as there appears to be a gradual increase in the size of an otherwise symptomless lumbar disc protrusion with each weight-bearing step until a critical point is reached and the patient experiences pain and begins to limp.

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