The clinical symptoms were SAH in 15 patients, ischemia in 15, and aneurysmal mass effect in six. Treatment included surgical or endovascular intervention, antiocoagulation or antiplatelet agent administration, and conservative management. Surgery should be considered if the dissecting aneurysm continues to cause intramural hemorrhage or increases in size. However, such interventions often cause neurological deficits, whereas dissecting aneurysm of the PCA is relatively benign compared with other aneurysms in the posterior fossa. The result of conservative treatment was not different from that of intervention among the 40 cases, so conservative treatment is recommended if neither rebleeding nor enlargement of the aneurysm is observed. Spontaneous resolution is sometimes observed, so annual follow-up is recommended.

Dissecting aneurysm of the PCA is rare, but should be included in the differential diagnosis of young patients presenting with SAH.

References

Abstract

We illustrate a patient with a migrating lumbar disc fragment that caused a change in radicular symptoms from the L3 nerve root on one side to the L5 nerve root on the contralateral side, documented by magnetic resonance imaging (MRI). Our patient presented with 3 months of L3 pain on the right side with sensory and motor changes. Over a 24-hour time period, the right leg pain disappeared and he developed left leg pain attributable to left L5 nerve root compression. Investigation with MRI revealed an epidural mass, which was hypointense on T1-weighted and T2-weighted images that had migrated, initially compressing the right L3 nerve root, to now compress the left L5 nerve root. The patient did not wish to pursue surgery. Disc fragment migration patterns are discussed. We conclude that extruded disc fragments may migrate distant from their initial origin.

Keywords: Migration; Lumbar disc; Herniation

1. Introduction

Lumbar disc disease is common, and symptomatic lumbar disc herniation has been recognised for over 70 years. Dandy (1929) was the first to recognise the clinical syndrome of radicular pain due to an extradural mass, and Mixter and Barr were the first to document that extruded disc material caused sciatic pain and detailed a surgical approach to the problem (1934). The most commonly recognised clinical syndrome is that of radiculopathy resulting from posterior or posterolateral disc rupture. Sequestered disc fragments account for 28.6% of all symptomatic disc herniations. Herniated disc fragments are known to migrate within the spinal canal in many directions, including rostral, caudal and lateral, and posterior epidural disc fragment migration has been reported rarely. We report an unusual case of separate clinically symptomatic nerve root compression syndromes from the one disc fragment.

2. Case report

A 32-year-old man initially presented with three months of severe right-sided buttock and lateral thigh pain. He sought medical advice when he noticed that when walking down stairs, he lost power in the thigh and would nearly fall. Clinical examination revealed decreased sensation in the lateral aspect of the thigh, a reduced knee reflex on the right and weakness of hip flexion. He was unable to perform his usual activities as a restaurateur and was motivated to seek a rapid solution. An MRI scan was organised which revealed a significant disc herniation that caused impingement of the right L3 nerve root (Fig. 1A,B). After appropriate discussion the patient was booked for microdiscectomy following initial consultation. Two days prior to the date of surgery he was reviewed at the preadmission clinic. The patient reported an unusual history of events that had occurred over the previous few days. He stated that one evening during a ‘muscle spasm’ of his low back, he rotated and flexed his spine in sev-
eral directions in an attempt to relieve his pain. The follow-
ing morning, much to his surprise, his right leg pain had dis-
appeared. However, during the course of the day, he now
noticed that he had left leg pain radiating into his knee and
lateral aspect of his leg. On examination his right thigh sen-
sory loss had recovered and knee reflexes were equal. There
were no sensory or motor deficits in his left leg. He no longer
stated that he had trouble walking down stairs and his leg
would not ‘give way’. The lumbar spine MRI was repeated.
The disc fragment on the right side was no longer in position
on the right at the L2/3 level. However, there was a new frag-
ment on the left side at L4/5 (Fig. 2A,B). Comparison of the
L2/3, 3/4 and 4/5 discs showed there was no new disc protrus-
sion, rather the disc appeared to be the same disc fragment
that had migrated to the contralateral side and migrated
inferiorly to compress the left L5 nerve root. The initial
MRI was re-examined by an independent neurosurgeon
and radiologist to verify that there was no disc extrusion at
the L4/5 level on initial presentation. The patient was no
longer keen to proceed with microdiscectomy, as his new
problem was not as disabling as his previous one.

3. Discussion

Disc herniation is defined as a localised displacement of
disc material beyond the limits of the intervertebral disc
space. The disc material may be nucleus, cartilage, frag-
mented apophyseal bone, annular tissue, or any combina-
tion thereof. A fragment of disc that has separated from
the disc of origin and has no continuous bridge of disc tissue
with the disc of origin is called a ‘free fragment’ or ‘sequested
fragment’ and the term ‘migration’ is used to signify displace-
ment of disc material away from the site of extrusion.7 There
are several methods for standardising the nomenclature used
in reporting the location of disc lesions in the lumbar spine;
however, the system most in use now is that presented by
Whiltse et al.8 In this system, areas in the axial plane (medial
to lateral), are called ‘zones’, and in the craniocaudal direc-
tion, they are called ‘levels’. The zones are the central canal
zone, the subarticular zone, the foraminal zone, and the
extraforaminal zone. In the caudocranial direction, the levels
from above downward are the suprapedicle level, the pedicle
level, the infrapedicle level and the disc level.

Disc fragment migration is frequent. When a fragment
ruptures the posterior longitudinal ligament and enters the
anterior extradural space as a distinct entity, it migrates in
35–72% of cases.9,10 Subligamentous herniations (con-
tained) and noncontained herniations, which rupture the
posterior longitudinal ligament, can migrate away from
the interspace. Lateral migration (for example, from the
subarticular zone to the foraminal zone), at the level of the
interspace is probably the most common pattern.11 Rostral
and caudal migration (for example, from disc level to pedicle
level), together, are the most clinically important modes of
migration, as they may result in retained fragments. Previ-
ous reports have noted a predominance of superior over
inferior migration, whereas others have noted these motion
patterns to occur with approximately equal frequency.12,13
Ebeling found that the probability of upward migration
was higher with far lateral discs, whereas more medial herni-
ations tended to move caudally. Lombardi first described
posterior extradural migration of a sequestered disc frag-
ment in 1973.14 Migration of disc material posterior to the
dura is a very uncommon condition and has been reported
in only 21 cases. Posterior epidural migration in the cervical spine has also been reported. Various structures are hypothesised to prevent posterior migration of disc fragments, and several authors attribute the rarity of these lesions to the presence of these anatomic barriers. These include the sagittal midline septum, peridural membrane, the nerve root, the dura, epidural vascular structures, and epidural fat. The sagittal midline septum spans the space between the vertebral body and the posterior longitudinal ligament and appears to limit side-to-side migration. An additional more delicate connective tissue membrane limits postero-lateral migration. The peridural or lateral membrane, attaches to the free edge of the posterior longitudinal ligament medially and laterally to the wall of the spinal canal. Once a fragment transgresses the peridural membrane, epidural fat and the epidural venous plexus, the nerve root itself presumably acts as an impediment to further posterior migration. The current case is the first report of migration to the contralateral side associated with an inferior migration over two levels. A defect in any one of these potential barriers may assist a fragment to migrate into the posterior extradural plane. Kuzeyli states there is a connection between posterior migration and the position of the nerve root relative to the intervertebral disc space following the observation of three cases. In addition, they theorise that heavy labour, traction, spinal manipulation and conditions of hypermobility may also predispose to posterior migration of disc fragments. Of note, our patient states that he had an unusual episode of back spasm resulting in a rotatory/flexion movement prior to the change in his pain profile.

Magnetic resonance imaging is recommend in the evaluation of radiculopathy. The differential diagnosis of a posterior extradural lesion includes abscess, metastatic tumour and haematoma; however, a sequestered disc fragment should be considered. The patient has been advised that he can expect to have a good result following surgery as a recent study demonstrates that discectomy for a sequestered fragment appears to correlate with an excellent clinical outcome as compared with a contained, broad-based disc bulge. Our patient is keen to proceed with conservative management. Disc reabsorption can occur and has been reported on multiple occasions. Typically disc reabsorption occurs over months to years. In our patient it is unlikely that the L2/3 disc had reabsorbed due to the time-course of events. The authors consider that the unusual movements the patient underwent resulted in the migration of the disc fragment distant from its original position.

References