

ADVANCED MUSCULOSKELETAL PRACTISE

CASE STUDY:

ABDOMINAL AORTIC ANEURYSM

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The purpose of this study is to highlight the case of a 71 year old female patient, who presented in an outpatient physiotherapy department with low back pain and bilateral lower limb symptoms. After initially responding to treatment the physiotherapist referred her back to her G.P., for further investigation. A subsequent lumbar spine x-ray revealed that she had a >5cm Abdominal Aortic Aneurysm (AAA). It is proposed to discuss how this patient may have been managed, had she been assessed by an advanced musculoskeletal therapist (AMP) working in an extended scope role.

Case Report:

The patient presented with an 8 month history of low back pain that was moderate to severe, and intermittent. The pain increased with prolonged standing and radiated into both her anterior thighs, greater on the left than the right. She experienced pain in her left shin and an intermittent loss of sensation in the left foot. The pain was relieved by rest and could be relieved for days at a time with minimal use of paracetamol. The most significant finding was that she was having difficulty climbing stairs, due to a general weakness in both legs. She also felt that she was not walking in a straight line and was pulling to the left on walking. Her bladder and bowel were unaffected. She had no saddle anaesthesia and there was no foot drop.

The patient's past medical history revealed that she had hypercholesterolemia for which she was taking Simvastatin. There was no other significant medical history. She was not unwell. She had no history of weight loss, fever, night sweats or night pain. Her husband had noted that she had lost some height over the last few years. She was able to walk without a stick but found it difficult to rise from a chair due to weakness. She was also a 20 a day smoker.

On examination, she was of slim build and had a marked left scoliosis of the lumbar spine. Active range of movement was reduced in all directions. Lateral flexion bilaterally increased her back pain. Her myotomes were unremarkable when tested in lying. In standing however, she was unable to rise onto her toes on her left foot. Her sensation was normal at the time of testing. Her L3 and L5 reflexes were difficult to elicit. Clonus was absent, but Babinsky appeared more brisk on the left. The slump test brought on the pain in her left thigh and prone knee bend and was restricted and painful bilaterally. Palpation of the lumbar spine was not provocative.

An AAA is caused by the ballooning of the aorta, as a result of the destruction of the elastin in the artery wall, described by Ballard, Fowkes and Powell (1999) and Macsweeney, Powell and Greenhalgh (1994). The U.S. Preventative Task Force (USPTF) in their recommendations on screening for AAA (2005), describe the risk factors as being over 65, with a history of smoking and a family history of AAA. Ruptured AAA occurs six times more in the male population than in women. However Scott, Bridgewater and Ashton in their randomised clinical trial of screening for abdominal aortic aneurysm in women (2002) found that the incidence of ruptured AAA in women is rising.

Ballard et al (1999) state that aneurysms of 6cms and over are treated by surgery to reduce the risk of rupture. Stowell, Cioffredi, Greiner and Cleland (2005), describe symptoms of an AAA rupturing as being acute pain in the lumbar sacral region, a

palpable abdominal mass and a pattern of severe progressive bilateral weakness of the lower extremities. Baird, Gurry, Kellam and Wilson, in their study of 2001 patients with AAA (1968) however described how more than 50% of patients subsequently discovered to have an AAA were 'asymptomatic'. It is important to note that in their study, Chapman, Shaw and Kubik (1964) found that aneurysms were known to rupture within 6 months of the onset of symptoms in approximately 60% of cases. At the time of assessment this patient had had her symptoms for approximately 8 months. She could therefore have been at severe risk.

There are more subtle indications of AAA and physiotherapists working in an extended role should be aware of these. A number of authors Sizer, Brismee and Cook (2007) Stowell et al (2005); Sherelud, Kirsten and Paynter (2002), all describe how AAA should be considered as one of the rarer causes of radiculopathy. Grieve (1994) lists AAA in his chapter on masqueraders in low back pain. Crawford et al in their literary review of AAA (2003) state that AAA should be considered in the differential diagnosis of all older patients with back pain and other risk factors for AAA. As an aneurysm enlarges it can put pressure on the surrounding structures. It is likely that as they enlarge the symptoms increase, depending on the structures that they impose upon. Crawford, Hurtgen-Grace, Talarico and Marley (2003) found that 75% of AAAs are located below the renal arteries, at L2 to L4 level. Therefore the descending nerves at this level are likely to be affected. This patient had pain in the L2 and L4 dermatomes and the reflexes at L3 and L5 were reduced. Sherelud and Paynter (2002), Laslett (2000) and Servant (1998), Dudeney, O'Farrell, Bouchier-Hayes and Byrne (1998), all describe how aneurysms in the abdominal cavity can cause patients to have back pain and lower limb symptoms. These symptoms can include pain, restriction of SLR, altered reflexes, myotomes and dermatomes. They can also include lower limb weakness and gait disturbance, which were the main concerning features in this patient's presentation.

The ability to recognise the presence of serious pathology relies heavily on the assessor's ability to recognise red flags. Sizer et al (2007), state that red flags are signs and symptoms that may tie a disorder to a serious pathology. Greenhalgh and Selfe (2006) discuss the importance of physiotherapists recognising red flags and determining how they interact with each other to indicate certain conditional probabilities. This patient was displaying a number of red flags and risk factors for serious pathology. These included age, the fact that she was a smoker and had an abnormal neurological presentation. Lyschneya and Henderson (2004), Quebec Task Force (1996), CSAG (1994) Bigos (1994) all indicate age >50-55 as a potential red flag. This is because the risk of presenting with serious pathologies increase with age.

The most significant red flag was gait disturbance and lower limb weakness. Sizer et al (2007), Deyo, Ranville, David and Kent (1992) all classify a neurological deficit not explained by a monoradiculopathy as being a category 1 red flag. Gifford (1991) describes this high rating as being due to the association with myelopathy. Myelopathy can create motor neurone lesions resulting from sagittal narrowing of the spinal canal (Cook et al 2007). This causes abnormalities within the spinal cord, producing gait disturbance and weakness. In his case study of metastatic disease mimicking lumbar disc herniation, Malone (2003) described how his patient reported a slight feeling of weakness bilaterally. Sherelud et al (2002) also found that radiculopathy, a lower motor neurone lesion in which chemical or nerve root

compression causes nerve root pain (Cook et al 2007) had been reported in up to 90% of cases of spinal tumour. Therefore gait disturbance, lower limb weakness and radiculopathy, all described by this patient, and could at this stage have indicated a number of pathologies.

The fact that this patient was a smoker was highly significant. Smoking is linked with a number of pathologies including osteoporosis (National Osteoporosis Foundation 2007). Osteoporotic fracture and nerve root compression could have been the cause of her symptoms, particularly as she had a slim build and had lost some height. Leyshon, Kirwan and Wynn-Parry (1981) state that nerve root compression as a result of osteoporotic fracture can cause pain, loss of reflexes, restriction of SLR and tender motor points. Smoking is also linked to a number of malignancies (ASH 2002) which could also cause the presenting symptoms. Smoking also greatly increased her risk of developing arteriosclerosis and AAA because, as MacSweeney et.al. (1994) suggest the toxic components of tobacco enhance the risk of atherosclerosis in the aortic wall. Cates in his study of 3 patients presenting with sub acute AAA (1997) states that cigarette smoking increases the risk of developing AAA in a ratio of 8:1.

Having established a number of red flags, further questioning of the patient could have been used to narrow down the possible diagnoses. It could have revealed a family history of AAA, Marphans or Ehlers-Danlos syndrome, which Crawford et al (2003) describe as having an association with AAA. The patient should be questioned as to the presence of abdominal symptoms, such as pain radiating into the groin or pulsation as described by Cates (1997). This would have been highly suggestive of AAA. In their article on the diagnosis and management of 528 Abdominal Aortic Aneurysms, Fielding, Black, Ashton, Slaney and Campbell (1981) state that in 91% of acute cases, AAA can be diagnosed by palpating a pulsatile abdominal mass. Unless the AMP had training in that particular technique, or the mass were immediately obvious, as described by Cates (1997) this should be left to the specialist. This concurs with CSP paper on the scope of practise (2004) that the physiotherapist should “demonstrate an awareness of other professionals’ expertise, which may be of more benefit to the patient”. It also highlights that carrying out a technique for which competency has not proven would not be covered by the appropriate professional liability indemnity.

Once a number of differential diagnoses have been established, it is the role of the AMP to use the diagnostic tools at their disposal, to narrow down the diagnosis further with a view to referring the patient to the most appropriate specialty Greenhalgh et al (2006). X-ray is often the initial imaging option in patients presenting with spinal pathologies, because images can be available immediately. The British Journal of Haematology guidelines on the management of multiple myeloma by Smith, Wisloff and Samson (2005) recommend that initial screening should include x-ray of the symptomatic areas because, x-rays may show weakened areas of the bones caused by the cancerous plasma cells or areas of fracture Rogers (2007). Various studies including those of Malone (2004); Wurtz, Peabody and Simon (1999) and Lischyna and Henderson (2004), demonstrate the value of using radiographs in the detection of other malignancies in the spine. As Sherelud et al (2002) state however, MRI or C.T. provide the best contrast of tumour, bone marrow and soft tissue. They also point out that radiography lags behind bone scan, in detecting metastatic lesions by 4-18 months, because of the percentage bone loss required,

before being identifiable on x-ray. X-rays can also indicate osteoporotic fracture. The limitations of X-rays however, need to be considered when the AMP refers a patient for imaging.

In the study of AAA carried out by Kauppila, MacAlindon, Evans, Wilson, Douglas and Felton (1997), lumbar spine x-ray was found to be sufficient in detecting an AAA because of calcification of the aorta. This was the case with this patient. If this patient had been assessed by an AMP, the presence of an AAA on x-ray would have resulted in the patient being immediately referred to a vascular consultant for further management. Ultrasound would then have been the imaging method of choice to provide detailed imaging of the AAA (UPSTF 2005). It is not known how this patient's management progressed as she did not re-attend the physiotherapy department, once she had been referred back to her G.P.

Blood tests would have been requested after the initial assessment to determine specific markers of pathology and assess the patient's general health. Sheralud et al (2002) state that a typical laboratory evaluation in suspected spinal pathology, would include assessing the general markers of inflammation; ESR, CRP and alkaline phosphatase levels. If Myeloma were suspected, the primary blood tests should include FBC, ESR, electrolytes, urea, creatinine, calcium, albumin and uric acid, (as described by Smith et al 2005). Electrophoresis of the urine also is carried out to identify the possible presence of the Bence-Jones protein. In AAA, ESR would be elevated because of the inflammatory mediators in the aortic wall (Crawford, Hurtgen-Grace, Talarico and Marley 2003; Cates 1997; MacSweeney et al 1994). Cates describes how patients with AAA may have impaired cardiac and renal function. They may also display anorexia and weight loss (Crawford et al 2003). Therefore a full blood count, ESR, CRP, chemistry profile, liver and thyroid function tests would be indicated in this patient's initial screening.

The benefit of having specialist physiotherapists assessing orthopaedic patients in an extended scope role is well documented in terms of their ability to screen and manage patients in primary care. This is described in the articles of Milligan (2003), Daker-White, Carr, Woolhead, Bannister and Kammerling (1999); Durrell (1996); Hourigan and Weatherley (1995 &1998) and Hockin and Bannister (1995); Byles and Ling (1989). As such, physiotherapists are now often required to assess patients, presenting with musculoskeletal problems, who may have serious underlying pathologies. This situation is likely to increase as we move towards self referral, as described by Littlejohn et al (2006). Abdominal aortic aneurysm is a potentially life threatening condition. The size of this patient's AAA, could have indicated that she required immediate surgery. She had also been symptomatic for a considerable length of time and as such she was at risk of rupture. Had she been assessed by an AMP appropriate investigations could have been requested immediately for this patient to refine the possible diagnosis and the patient directed to the correct speciality for treatment. Prodigy (2005) asks that referral to a specialist within 4 weeks be considered for anyone presenting with a possible serious spinal pathology. This patient was not referred for imaging until 8 weeks post initial assessment and an x-ray had to be requested through her G.P. thus delaying her diagnosis further.

As advanced practitioners we must be aware of the possible signs and symptoms of AAA as they are not rare. Scott, Bridgewater and Ashton (2002), state that there was

a 14% rise in the mortality of women from ruptured AAA in the period between 1989 and 1998. They now cause 0.8% of all female deaths in those aged 65 or over. By recognising the signs, symptoms and risk factors for AAA the AMP is contributing to 'the health status of society and the individuals within that society.' van der Meene (1998).

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