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Myofascial and nerve compression pain simulating bone involvement in a patient with prostate cancer

Abstract

Many patients with advanced disease suffer of pain. Not all this pain is induced by the disease itself like tumour growth or infiltration of the nerves. Some of the pains are due to debilitation and degeneration due to immobilisation, atrophy and poor nutrition and are only indirectly related to the progression of the disease. Atrophic changes in the subcutaneous tissue, muscles and tendons can be accompanied by the compression of the nerves against protruding bony edges. This type of pain, not specific to any advanced disease in particular, can be extremely difficult to be treated. We present here a case of patient with a metastasised prostate cancer who suffered of this type of pain and did not respond to standard treatment targeting tumour related pains. Compression of the small cutaneous nerves was successfully treated with injections of depo steroids and local anaesthetics. Not every pain in disseminated prostate cancer is a bone pain. It is our impression that this type of pain is much more common among the patients in palliative care.

Key words: myofascial pain, nerve compression pain, opioid resistant pain, steroid injections

Introduction

Pain in patients with disseminated prostate cancer is usually associated with bone secondaries [1]. This pain is treatable with palliative radiotherapy and when appropriately diagnosed it is unusual when the pain does not respond to the treatment. Each pain, however, including pain in prostate cancer, needs a thorough workup and diagnosis and not all pain in these patients is due to bone secondaries. One patient can suffer from more than one type of pain [2]. All of the pains need to be recognised and treated specifically. This case illustrates how important it is to think analytically and to be aware of other types of pain and their treatment. Clinicians should not be "blinded" by the paradigm "prostate cancer — always bone pain".

Case

Peter was a gentleman of 76 years of age. He was a pensioned unskilled dockworker. He was married to Jane and they had two adult sons, one of whom was a military officer who was killed in Iraq a year ago. According to his father, his son died because of a mistake by a colleague. Peter and Jane are still mourning their son. Every day.

Four years ago, Peter was diagnosed with prostate cancer (T2aNoMo). Initially there were no secondaries and Peter was treated with a prostatectomy and radiotherapy. The Prostatic Specific Antigen (PSA) was initially 35 ng/L but decreased to nearly normal values, 8 ng/L, after the operation. Peter was confident that he was cured and he was enjoying a "second life", as he said. However, when Peter and

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Jane received the message about their son's death, nothing was the same any more. Peter did not sleep at night. He started to feel pain in many locations, in both his shoulders, in the left groin and in the lower back. He was devastated when his surgeon noticed that the PSA was increasing. The bone scan carried out twice, 19 and 8 months before admission to the hospice, showed progression of multiple bone metastases. Plasma calcium was determined several times but remained normal. When the PSA values began to rise, Peter started on anti-androgen treatment. The urologist also prescribed 2 mg of dexamethasone daily for improvement of the antitumour therapy. Unfortunately, the PSA value did not fall and after 3 months Peter was changed to other hormone treatment.

The pain increased slowly. It was also more diffuse and ill defined. He was treated with ibuprofen 200 mg tds and paracetamol for the pain. After 6 weeks his GP decided to add tramadol 50 mg tds. This treatment was also ineffective and, according to the analgesic ladder, he was changed to controlled release oral morphine 20 mg bd. This dose was gradually increased to 80 mg bd. At about this time, Peter was referred to the pain clinic at our hospice with a diagnosis of intractable pain.

Despite the dexamethasone and a reasonable appetite, Peter lost 10 kg of his body weight. He became weak and fatigued. The pain history revealed moderate, sometimes severe pain which was exacerbated on movement. Peter could precisely show, using his fingers and hands, where the painful spots were. Analysis of these spots revealed pain in the right shoulder, just above the scapular ridge in the supraspinatus muscle. Peter could not move his right shoulder properly, raising his arm laterally above the level of the shoulder was impossible and especially painful. There was a typical "twitching" reaction when the spot was pressed with a finger. The other location was in his lower back, on both sides, laterally from spinous processes at the level of L5/S1. The muscles were very tender and tense during palpation. There was no pain on tapping the spinous processes of all vertebrae. There was also a painful spot around the left superior anterior iliac spine. When this location was pressed the pain radiated to the front of the thigh. On physical examination there was hyperalgesia discovered on the skin of the left thigh. Finally, there was a painful spot on the iliac crest on the left side, some 14 cm behind the superior anterior iliac spine. There was no pain on palpation in the left distal femur, the place indicated by Peter as painful.

The drugs Peter was using are listed below:

— morphine sulphate controlled release 80 mg bd;
— oral morphine 15 mg per dose (usually 4–6 doses per day);
— ibuprofen 400 mg tds;
— paracetamol 1 g qds;
— lansoprazole 30 mg od;
— diethylstilbestrol 5 mg od;
— zolendronate 4 mg iv 4 weekly for the past 3 months;
— dexamethasone 2 mg od, for the past 5 months;
— movicolon 13.6 g, 1 sachet per day.

The remaining physical examination, including a neurological examination, did not show any abnormalities. The Thompson and Kopell test [3, 4] for both shoulders was negative. There were no signs suggesting involvement of the skeletal structures. Pressure from front-to-rear and from the sides of the chest did not induce any significant pain. A similar pressure on the pelvis did not result in pain either. Percussion of the spine was not painful. Blood analysis revealed that the liver and renal functions were close to normal. Corrected plasma calcium and plasma magnesium were also normal. A full blood count revealed normal haemoglobin and platelets. The PSA was 41 ng/L.

A new bone scan was requested and the appointment for this procedure was made for 2 weeks in the future. In the meantime Peter was admitted to the ward to optimize his pain control medication.

The above mentioned pain points (to be called trigger points here) were infiltrated with 15 mg of bupivacaine and 40 mg methylprednisolone per point. This was carried out in two different sessions. In total there were five points infiltrated. Two days later Peter reported that the experienced pain intensity was clearly less than before. He had not even used one dose of breakthrough oral morphine. The controlled release doses of morphine sulphate could be decreased to 40 mg bd. When even lower doses of this drug were tried the patient started to complain of moderate pain in his left leg. With morphine sulphate 40 mg bd his condition was again better and his mobility was markedly improved. His bowels also worked much better.

The bone scan disclosed a number of hot spots suspected of being bony secondaries. The hot spots were localized in the skull, right chest in at least two ribs and in the distal part of the left femur. Except for the left femur, the other localizations were clinically "inactive" or were causing no pain at all . The X-rays of these localizations revealed significant bone

defects in the skull and left femur but not in the right chest. The two positively matched localizations were treated with a single dose of radiotherapy (8 Gy).

The pain intensity decreased almost to zero. The patient was discharged home and was seen monthly for an infusion of zolendronic acid 4 mg. Three months later he is still free of pain.

Discussion

The pain seen in Peter's case can best be defined as myofascial and nerve compression pain [5]. Finger pressure at certain points induces pain. This kind of pain is frequently observed in terminally ill and cachectic patients, those on steroids being especially prone to developing muscle atrophy. The precise epidemiology of this kind of pain is unknown. In one study 12% of the pains observed in cancer patients were classified as myofascial and nerve compression pain [6]. The localizations of pain suggest that not only muscles and their insertions were causing pain; in some places compression of the small cutaneous nerves may be expected to be the reason for this pain [7].

Our patient complained of lower back pain and had two trigger points parasacrally at the level of L5/S1. These two trigger points are commonly found in low back pain [8]. The pain is most probably caused by compression of the superior cluneal nerve against the posterior iliac crest [9]. There are some data from at least two controlled trials that infiltration of these points with slow release steroids may be effective in controlling pain [8].

Another pain syndrome found in this patient is meralgia paraesthetica [10]. This syndrome is well known in the literature. The painful spot is localized just under the anterior spina where the lateral cutaneous inguinal nerve emerges under the inginal ligament. It is believed that this nerve may be impinged under this ligament (peripheral form) or that the pain in the thigh results from impingement of the nerve root in the pathological changes of the spine [11]. This pain can be accompanied by abnormal sensitivity in the front part of the thigh and can be treated with local steroid injections.

Another pain syndrome can originate by compression of the small cutaneous nerves piercing the internal oblique muscles inserting to the iliac crest [12]. The two nerves which can be damaged are the cutaneous branch of the last subcostal T12 nerve and 5–7 cm behind it, the cutaneous branch of the iliohypogastric nerve. In many cases one can find two characteristic points on the iliac crest sensitive to pressure. In many patients either one or both points is active. The reason for this compression is probably loss of supporting underlying tissues (muscle, fat) and compression of the nerve against the iliac crest bone. This type of pain is well described in patients after bone graft harvesting from the ilium bone [13]. In the literature, a similar syndrome of painful iliac crest points is described but its origin is attributed not to irritation of the nerve against the iliac crest bone but to intervertebral dysfunction at the thoracolumbar junction [14]. We did not carry out any investigation of the spinal cord to prove or disprove this. However, if the steroid injection to the painful point of the iliac crest is succesful and the effect is maintained for weeks or even months, for us it is proof of the peripheral origin of this pain. We would request an MRI of the spine if the treatment as described above were unsuccessful.

Many terminally ill patients present with shoulder pain. The pain is maximal in the case of the lateral abduction of the arm. The examining physician should ask patients to reach with their hand to the contralateral ear, keeping the elbow at eye level. This is called the Thomson and Kopell test [3, 4]. Sharp pain during this manoeuvre suggests the stretching and irritation of the suprascapular nerve that enters the shoulder blade through a very narrow suprascapular canal. When the muscles fixing the shoulder blade to the chest are atrophic, shoulder blade excurions will increase and the tension and irritation of the suprascapular nerve will emerge [15]. When the Thompson and Kopell test is negative, one can expect the trigger point to be rather localized in the suprascapular muscle, usually in the distal 1/3. Pressure with the finger against the bone of the scapula crest is usually enough to see this. Many patients will show a characteristic twitching response, simply to avoid more pain. Palpation of the muscle may disclose a taut band or even a painful induration in the muscle. Usually, no abnormal sensitivity is seen as the suprascapular nerve normally consists only of motor neurones. The pain mechanism, therefore, is the irritation and inflammation of the perineural sheath. Chronic compression or irritation of the nerve can result in atrophy of the scapular muscles.

We do not know exactly if terminally ill and frail people are more likely to develop this kind of pain. This problem has not yet been studied in detail. One may speculate that chronically ill and cachectic patients may have atrophic muscles and this may in

turn make them vulnerable when the muscles are overloaded. Apart from the direct effect of the tumour on the muscles, leading to nutritional deficiencies, cachexia and muscle dystrophy, Peter had also used dexamethasone for a long time. Glucocorticoids are responsible for excessive muscle wasting. In Peter's case dexamethasone was prescribed to enhance the response of prostate cancer to hormone treatment. As Peter very much wanted to live normally and remain ambulant, he overloaded his own muscles. In this way, he had considerable opportunity to do this. As many nerves pierce the muscles, when the muscles are atrophic the nerves lose their natural support and may become vulnerable. This happens especially in the places close to bony edges and to tendons.

The response of the myofascial and nerve compression pain to analgesics is variable [16]. In general the drugs are effective against pain at rest but much less succesful with pain on movement. Many patients report that the pain responds only briefly to opioids, suggesting that the sensitivity threshold to opioids has increased. This is also the reason why the dose of opioids is frequently and rapidly increased, leading to opioid toxicity. When the pain is rapidly diminished, either by radiotherapy or steroid soft tissue injection, it may be that the opioid toxicity is exacerbated.

There are several methods for treating myofascial and nerve compression pain. However, none of the methods has been adequately tested. In two placebo-controlled studies, injections of slow release steroids in the prasacral points helped more against low back pain than injection of an inert placebo [8]. Apart from steroids, short-acting local anaesthetics are frequently used in so-called "injection therapy". Lidocaine and procaine are most often used in this context. After injection of the drug the muscles are stretched to relieve the cramped areas. Dry needling, or piercing the muscle with a needle without injecting anything, may also sometimes be helpful [17]. It is unclear, however, how long this effect will last. Moreover, the systemic analysis of all available data neither confirmed nor denied this effect. Some non-pharmacological methods such as massage, warmth and ice cubes may also be effective.

Neuropathic pain frequently responds to drugs such as tricyclic antidepressants or anti-convulsive drugs. It is not known if nerve compression pain also responds to these drugs [16]. The treatments can at least be tried and should not be refuted without trial. High doses of dexamethasone may theoretically be helpful if there is inflammation and oedema around the nerve but injection of slow release steroids is certainly less toxic than systemic ones.

Thanks to the steroid injections, some of the pain in Peter's case could be adequately controlled and the dose of morphine decreased. However, when the dose of morphine sulphate was decreased to the level of 40 mg a day, Peter started to complain about the pain in his left leg, close to the left knee. It is probable that this pain was under adequate control with higher doses of morphine and "reemerged" only when the morphine dose was decreased. There are no known myofascial trigger points or cutaneous nerves above the knee and, above all else, the bone scan and bone X-ray suggested a "hot spot" at this localization. Radiotherapy, as expected, was effective in controlling this pain within several days. This is a good reason why pain localizations with a negative match on the bone scan and X-rays should not be treated "blindly" with radiotherapy. Theoretically, radiation could increase the irritation of the small cutaneous nerves and even increase the pain.

Without doubt, psychological factors are able to increase pain sensations. The chronic grief after the death of his son could be the reason for many complaints with an unclear pattern. Above all, the time when these complaints started coincided with the loss of his son. Once the pattern has been discovered, nobody can say that the pain is imagined or psychological. Many patients are started on antidepressants, although there is little evidence that this helps. Although the psychological factors need to be recognised in a case like Peter's, this should not deter doctors from looking for the physical cause of pain.

Conclusions

1. The automatic perception of disseminated prostate cancer — increased levels of PSA — bone metastases thus bone pain is probably wrong.
2. Patients with actively growing tumours may experience pain due to non-malignant causes, as well as cancer pain.
3. Myofascial and nerve compression pain still need to be recognised in oncology
4. A positive response to the injection of local anaesthetic can confirm or refute a diagnosis of myofascial or peripheral nerve compression pain.
5. Radiotherapy to the sites with a negative match on a bone scan should be avoided.

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