Subacute steroid-induced paraparesis: surgical treatment of a devastating “invisible” side effect

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Abstract

Purpose To emphasize an underestimated side effect following long-term use of steroids.

Methods We report on surgical treatment of two patients with serious neurologic deficits caused by epidural spinal lipoma following long-term intake of cortisone.

Results Early decompression of the spinal cord by removal of epidural lipoma was the most effective treatment in these patients with progressive symptoms.

Conclusion Diagnostic work-up of such patients should include early spinal MRI resulting in surgical intervention, if indicated. Decompression of the spinal cord eventually combined with fusion is necessary.

Keywords Lipoma · Paraplegia · Spinal instrumentation · Spinal tumor · Steroids

Introduction

Cortisone may be one of the most important miracle drugs of the last century. Its impressive effect in many diseases without curative treatment options has made cortisone almost indispensable in certain conditions [2, 5, 6, 13, 15, 16, 19].

However, cortisone may have serious side effects, especially following long-term use. One of the side effects following its chronic use is fat deposition, predominantly

centripetal in the subcutaneous area of the face, neck, and trunk [12]. Marginal attention so far has been paid to epidural spinal fat accumulation following long-term use of steroids, since it is clinically “invisible”. Its clinical presentation with pain, hypesthesia, ataxia, incontinence, and paresis does not differ from other epidural spinal mass lesions. Conservative treatment with reduction or withdrawal of cortisone has been advocated as a gentle and less invasive therapy in asymptomatic and even in some symptomatic patients [7, 8]. However, this is not an effective option in severely affected patients for whom surgical approaches may be required [7].

Although about 50 patients with steroid-induced myelopathy have been reported earlier, this entity remains largely unknown [7, 8, 11].

Here, we report our experience with surgical decompression in two patients.

Case 1

This 65-year-old man was admitted to the emergency room suffering from a progressive paraparesis for several weeks. Neurological examination disclosed proximal paresis at the lower limbs, urinary dysfunction, and hypesthesia below T4. Magnetic resonance imaging (MRI) of the spine showed a dorsal epidural mass extending from T2 to T9 with compressive displacement of the spinal cord (Fig. 1a, b). He had medical history which was remarkable for following renal transplantation, because of diabetic nephropathy. The patient underwent dorsal decompression of the spinal cord via interlaminar fenestration at six levels, three on each-side alternating. The epidural fat deposition was removed until unhindered expansion of the dura mater was achieved. The postoperative MRI confirmed appropriate

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Fig. 1 Sagittal and axial MRI scans of the thoracic spine of patient 1 show a dorsal epidural lipoma with compressive displacement of the spinal cord

decompression of the spinal cord with signs of myelopathy. Three months postsurgery, the patient was able to walk despite residual ataxia and the urinary dysfunction was resolved. This remained unchanged at the last follow-up 2 years later.

Case 2

This 22-year-old man developed a progressive subacute paraparesis which finally resulted in an inability to walk. On admission, spastic paraplegia and incontinence were present. Sensory loss was localized below the level of T4. His medical history revealed that he took high doses of prednisolone for 8 years for treatment of rheumatoid

polyarthritis. A centripetal fat deposition on the face, neck, and trunk as well as dermatologic side-effects of cortisone was evident.

MRI and computed tomography (CT) scans of the thoracic spine showed a dorsal epidural mass compressing the spinal cord. In addition, there was a reduction in the height of several vertebral bodies due to osteoporosis.

Decompression of the spinal cord was performed on the same day by interlaminar fenestration at four levels to remove the hypertrophic epidural fat. Few hours after surgery he was able to move the lower limbs. MRI showed decompression of the spinal cord after removal of the epidural fat. One week later, transpedicular instrumentation was performed to secure bony stability.

During a prolonged course there was further recovery of the paraplegia. He died 6 months later from heart failure.

Discussion

Epidural spinal fat accumulation following long-term use of steroids appears to be both under-recognized and underdiagnosed. This clinically “invisible” side-effect seems to be wide, and might be neglected in particular in case of gradual development of symptoms as it can occur in the absence of the more common steroid side-effects.

The clinical presentation with muscle weakness caused by the spinal cord compression can be misdiagnosed as steroid-induced myopathy [9]. Other myelopathy symptoms like sensory disturbance, ataxia, and bladder dysfunction, however, should raise the suspicion of a spinal lesion. In such cases or in case of doubt, an MRI of the spine including T1-weighted images would disclose the amount of the space occupying epidural fat accumulation. Thickness >6 mm is recognized as suggestive and >7 mm as a definitive diagnostic criterion for spinal epidural lipoma [10, 17, 18]. Spinal epidural fat accumulation in our patients was much more prominent.

We should emphasize that the presence of spinal lipomatosis, although “invisible” is also not clinically relevant, unless clinical symptoms arise and might be ignored unless it is presented clinically. Treatment depends on the severity and acuteness of the onset of symptoms [14]. To avoid prolonged or even irreversible neurological deficits, urgent decompression of the spinal cord is indicated [1, 4, 20]. The importance of surgical decompression is underlined by the fact that several chronic conditions necessitate steroid intake and make withdrawal or reduction of the steroids impossible. A cumulative effect of steroids and insulin as reason for the development of an epidural spinal lipoma like in the first case also has to be considered. Less invasive approaches such as interlaminar fenestration may be an

alternative to extensive laminectomy which is the standard approach for early surgical decompression.

In case of moderate to severe osteoporosis as described in case 2 more extensive surgery may be required [4]. Emergency decompression of the spine by laminectomy for removal of the compressive fat tissue may be followed by secondary fusion.

The development of steroid-induced spinal lipoma is independent of steroid dosage, and it may occur even in patients receiving low dosage of prednisolone [3]. The pathogenesis of this possible harmful condition is not fully understood. Growth of the epidural fat can possibly be explained because of direct stimulation by cortisol or indirect stimulation by the autonomic nervous system leading to progressive hypertrophy in a similar manner like the centripetal fat [3].

Time to onset of symptoms of SEL varies between 2 months and 30 years after initiation of glucocorticoid treatment. BMI and tissue sensitivity to glucocorticoids are probably important factors. There seems to be a direct correlation between the concentration of the glucocorticoid receptor in a cell and the sensitivity of the cell to glucocorticoids [3].

The widespread use of steroids emphasizes the importance that not only medical specialists but also general practitioners, who prescribe steroids, should be aware of the possibility of such catastrophic consequences besides the more common and well known side effects of long-term use of steroids even in the absence of the latter.

Finally, it should not remain unmentioned that you may also see spinal lipomatosis in patients who have never taken any steroid at all.

Conclusion

Diagnostic evaluation of patients with signs and symptoms of myelopathy under long-term use of steroids should result in early imaging studies. Surgical decompression of the spinal cord eventually combined with fusion is indicated in patients with rapid progression of symptoms.

Conflict of interest None of the authors has any potential conflict of interest.

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