Spinal cord compression by extradural fat after prolonged corticosteroid therapy

Case report

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This young man was operated on twice for thoracic spinal cord compression. He had been on corticosteroid therapy for the last 2 years subsequent to a renal transplant. The only anomaly discovered during the operation was a large quantity of extradural fat that did not present the characteristics of lipoma. The remarkable postoperative clinical improvement suggested that the fat deposit was responsible for the spinal cord compression.

Key words: spinal cord compression • extradural fat • lipomatosis • steroid therapy

Renal transplants have become very common, and patients often require follow-up corticosteroid therapy for a long period of time. Corticosteroid therapy is well known to have some undesirable effects, including Cushing's syndrome and cutaneous or mediastinal lipomatosis. We are reporting the case of a patient presenting with spinal cord compression. At surgery an unusual amount of fat was found deposited in the extradural space. Such a localization suggests that there is a cause and effect relationship between the fat accumulation and the spinal cord compression.

Case Report

This 30-year-old man was hospitalized at the beginning of 1978 for diffuse proliferative glomerulonephritis secondary to purpura rheumatica (Henoch-Schonlein purpura). Renal function worsened rapidly, causing severe arterial hypertension, and the patient underwent a renal transplant on June 2, 1978. His postoperative course was satisfactory. His drug regimen was azathioprine (Imuran), antihypertensive drugs, and prednisone at an average daily dose of 60 mg (the upper and lower limits were 120 and 40 mg). However, in 1979, he developed poorly controlled arterial hypertension, obesity (his weight increased 16 kg, from 66 to 82 kg), and neurological signs of painless intermittent spinal claudication. He had difficulty going up and down stairs and could not walk long distances. These problems slowly worsened after September, 1979, and the patient was hospitalized in December, 1979.

First Admission. Clinical examination revealed almost total spastic paraplegia. Only a few movements were possible in the proximal part of the lower limbs. He presented well defined hypesthesia below the T-8 dermatome, along with impaired proprioception. There was a band of painful hyperesthesia at T-6 and T-7. A urinary catheter was inserted because of sphincter difficulties. Emergency myelography showed a total obstruction at T-3, suggesting extradural compression (Fig. 1 left).

First Operation. On December 21, 1979, laminectomy was performed between T-1 and T-5. The extradural space contained extremely abundant fatty tissue about 1 cm in width. This fatty tissue had a normal microscopic pattern, did not resemble a lipoma, and was not obviously compressive. The intradural space and the spinal cord were intact. Postoperative recovery was rapid, and the patient began to move his lower limbs promptly. Proprioceptive and sphincter difficulties disappeared. He was discharged able to walk with two canes. The prednisone was continued at a daily dose of 20 mg. The patient progressed until August, 1980, when the neurological signs reappeared and he could not walk.
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Second Admission. He again presented with spastic paraplegia, and had hypesthesia below T-8. Myelographic films showed an incomplete obstruction from T-4 to T-8 below the level of the previous laminectomy (Fig. 1 right). The paraplegia suddenly became complete after myelography.

Second Operation. On August 20, 1980, a laminectomy was performed from T-4 to T-10. Once again a large amount of fat occupied the entire extradural space. The fat was removed by aspiration. Histological examination of the fat revealed normal adipose tissue. The patient’s motor power rapidly improved the day after surgery, but some degree of spastic paraplegia persisted and walking was difficult.

Discussion

Many publications have described either iatrogenic or spontaneous lipomatous complications in the face, neck, or mediastinum. Vertebral, extradural, or even intradural localizations are less common. The excessive amount of extradural fat over the vertebrae in our patient provoked actual stenosis of the spinal cord, although clinically and anatomically the usual signs of stenosis were not found.

This patient had been taking corticosteroid therapy for 2 years after a renal transplant. Twice he underwent laminectomy to relieve signs of spinal cord compression. At each operation, abundant extradural fatty tissue was discovered, and its removal caused rapid improvement. Consistency of the bone during laminectomy, the ligamentum flavum, and the dura mater appeared normal, whereas the fatty tissue, usually diminished in cases of stenosis, was abundant. Histologically, this fatty tissue had a normal but hypertrophic appearance and was not encapsulated. It was thus different from a lipoma, which is an encapsulated tumor frequently found in the filum terminale.

We found only one other published case similar to ours. It also involved a man taking corticosteroids after a renal transplant. The first signs of spinal cord irritation were sensory and appeared 15 months after transplant surgery. The patient was taking 40 mg of prednisone a day. Myelography revealed an obstruction between T-2 and T-9. The patient was operated on three times in 1 month. Extradural fat of normal morphology was as much as 5 mm in width. That patient recovered rapidly, and his daily dose of prednisone was reduced to 12.5 mg. It has been found that fat deposits disappear or regress in the mediastinum when corticosteroid treatment is discontinued or when Cushing’s disease is cured. Radiographic or computerized tomographic (CT) films that previously showed a mediastinum enlarged by fat deposits, become normal postoperatively.

Price and Rigler noted that such mediastinal complications after kidney transplant occurred after an average daily dose of 44 mg of prednisone. There was no such complication when the patient took less than 30 mg a day. Butcher and Sahn reported on a patient taking 20 mg a day of prednisone who had some signs of spinal cord irritation. Autopsy revealed an extradural space slightly infiltrated by fat. Those authors believed that lipomatosis occurs after the 2nd month of treatment when the daily dose of prednisone is greater than 60 mg and after the 5th year of treatment for low daily doses of 7.5 mg. This phenomenon has been described in patients with kidney transplants, collagenosis, asthma, and severe blood disorders. Thus, corticosteroids appear to have this undesirable effect when taken in low daily doses for a long time or when taken in high daily doses for a short time. In the case presented here, the decrease in the daily dose of prednisone from 60 to 20 mg did not prevent a recurrence of the extradural fat proliferation. This suggests that the duration of the treatment is a more important factor than the amount of the daily dose. Radiological mediastinal signs are easier to detect than vertebral signs, and evolution of mediastinal fat proliferation is easier to follow and thus have established anatomobiological correlations.

The etiology of the change in fat distribution and

FIG. 1. Left: Myelogram before the first operation showing complete obstruction at T-3 (arrow). Right: Myelogram before the second operation. There is partial obstruction of metrizamide from T-4 to T-8 (arrow).
Symptomatic extradural fat with corticosteroids

metabolism is not clear. Our patient illustrates the pathogenic puzzle: the extradural fat was abundant during the first operation but did not appear to exert a compressive effect. The dura mater had to be opened and explored to verify the integrity of the dural space and of the spinal cord. It was only when the patient rapidly recovered after each operation that the extradural fat was thought to be responsible for the spinal cord irritation. Other publications appear to confirm this hypothesis, but have the same difficulty in explaining how the highly abundant extradural fat can irritate the spinal cord without presenting actual signs of compression.

Diagnosis of mediastinal widening due to lipoma has been facilitated by CT scanning, with a tumor density between 40 and 50 EMI units. Perhaps CT scanning can be used in the future to diagnose extradural fat proliferation. It is possible that complete interruption of corticosteroid treatment when the initial neurological signs appear might result in a decrease of extradural fat and thus avoid surgery.

References

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