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Horner's Syndrome Resulting from a Lumbar Sympathetic Block

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Lumbar sympathetic blockade provides sympathectomy of the lower extremity. Numerous complications have been reported with this procedure, including neuralgia, subarachnoid block, bleeding,[1] and kidney damage.[2] We report a case of Horner's syndrome following this procedure.

REPORT OF A CASE

A 32-yr-old man was referred for evaluation of chronic left knee pain. The patient sustained a knee injury in August of 1985. Following three arthroscopies and a repair of the medial meniscus and anterior cruciate ligament, he continued to have pain. Repeat arthrogram was normal. The persistent pain was described as a continuous aching sensation that occasionally radiated down the leg to the foot. He also complained of coolness in the left lower extremity, but denied any alteration in skin color, texture, or nail or hair growth.

Physical examination revealed a well-developed, healthy man with equal skin temperatures in the lower extremities (as measured by cutaneous thermistors taped to corresponding areas of the lower extremities and recorded with a Yellow-Springs Telethermometer), and no alterations in skin texture or hair or nail growth. The left leg showed no edema or vasomotor or sudomotor changes, and neurologic examination was normal.

Roentgenographic studies showed a small amount of juxta-articular osteoporosis of the left knee joint. An area of homogeneous calcification with an irregular outline was also noted lying behind the extreme lower end of the femoral shaft.

The etiology of the patient's pain was unclear to the orthopedist, and this was the basis for the referral. Based on the pain's persistent nature and the complaint that the left leg felt cooler than the right and had intermittent edema by history, a reflex sympathetic dystrophy was felt to be a possible etiology.

A left lumbar sympathetic block was discussed with the patient and performed in the manner of Carron *et al.*[3] A 20-gauge, 15-cm needle was walked off the vertebral body of L2 into the left anterolateral paravertebral area. No paresthesias were obtained, and, after aspiration of the needle was negative for CSF or blood, 8 ml of 1% lidocaine were slowly and easily injected. Skin temperature of the left foot was noted to increase from 26 to 32.5° C over a period of 5 min. Right foot temperature remained 26° C. No sensory deficit in the lumbar dermatomes was present. After a repeat negative aspiration for CSF or blood, 6 ml of 0.25% bupivacaine were slowly injected, again without paresthesias, and the needle was removed.

Approximately 15 min following this second injection, the patient began to complain of mild numbness of the entire left side of his body. On repetitive examinations, the patient was noted to have slightly decreased sensation to pinprick in this same distribution. The sensory deficit extended to the midline. Reflexes were symmetrical bilaterally, and there was no motor weakness. No cardiovascular changes were noted, and vital signs remained stable throughout. A left-sided Horner's syndrome with obvious miosis, ptosis, and marked conjunctival injection, which was not present prior to the block, was noted to be present by multiple observers.

Despite the clearly apparent sympathetic block, there was no improvement in the patient's knee pain. The Horner's syndrome and sensory deficit resolved in 40 min, and the patient was sent home. Subsequent follow-up was remarkable for the complaint of blurred vision until late in the evening following the block and a gradual clearing of all symptoms by the next day. His knee pain remained unchanged.

DISCUSSION

Sympathetic innervation to the head arises from preganglionic sympathetic fibers that exit the spinal cord in the anterior roots of C_8 and T_1, but may come off as low as T_4.[4] These neural fibers travel through the stellate ganglion; then, through the middle and superior ganglia. Horner's syndrome and related features of cervical sympathetic block (characterized by ipsilateral miosis, ptosis, anhidrosis, enophthalmos, and facial blushing) results after interruption of these sympathetic fibers, as occurs with stellate ganglion block. We report the occurrence of a *lumbar* sympathetic block associated with

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a Horner's syndrome, which has not been previously described.

Many of this patient's clinical findings are difficult to explain. The onset of the Horner's syndrome was definite, and was observed by several experienced pain specialists. This suggests that, in addition to a lumbar sympathetic block, the entire upper left sympathetic chain became anesthetized, implying that the local anesthetic injected at L_2 extended cephalad to at least a T_3 level. Cousins and Bridenbaugh have demonstrated that 0.1 ml of contrast material spreads one vertebral segment.[5] Our 14-ml total dose, therefore, may achieve higher levels than we would normally expect. Variations in the dimensions of the paravertebral space could vary the amount of cephalad spread. Our patient may have had an unusually restricted space in cross-sectional area, resulting in increased cephalad spread with subsequent total sympathetic block on the left side.

Alternative explanations for this clinical observation include abnormal sympathetic chain anatomy or, possibly, a weak, one-sided, high epidural block due to a misdirected needle entering the epidural space. Numerous reports of Horner's syndrome following lumbar epidural and caudal block have been noted,[6–8] with an incidence of 1.33% following labor epidurals and a 4% incidence following lumbar epidurals for Caesarean sections.[6] Despite sensory anesthesia to only a T_7 dermatomal level, Horner's syndrome has occurred as a complication of lumbar epidural anesthesia.[7] Uneven epidural blocks have been reported, and may be due to a dorsomedian connective tissue band, as described by Blomberg.[9] Although this could possibly explain the complaint of unilateral abnormal sensory perception in this patient, no reflex or motor changes were noted. Also, the patient's sensory deficit included the cranial nerve distributions, which would not be explained by epidural anesthesia. A unilateral epidural block, therefore, was unlikely to have occurred. A psychosomatic etiology would be another explanation for the subjective complaint of abnormal sensation, but not for the occurrence of the Horner's syndrome.

Sympathetic blockade appears to cause changes in pain threshold in humans.[10] A unilateral, total sympathetic block, as achieved in our patient, may have affected somatic afferent nerve transmission in the ipsilateral side. This might explain the analgesia to pinprick, as reported by this patient.

In summary, we report the occurrence of a Horner's syndrome following a lumbar sympathetic block associated with a mild sensory deficit of the entire left side of the body. The sympathetic nervous system may play a role in modulating somatic pain threshold in humans, thus explaining the mild sensory deficit.

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