

PARAPLEGIA FOLLOWING COELIAC PLEXUS BLOCK WITH PHENOL

Case Report

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SUMMARY

Paraplegia complicating a block of the coeliac plexus with 6% aqueous phenol for carcinoma of the pancreas is described. The patient had no previous neurological abnormality and it is postulated that vascular ischaemia of the spinal cord was responsible for the sequence of neurological events.

The occurrence of paraplegia following a coeliac plexus block would seem to be an obvious possible complication in view of the anatomical proximity of the coeliac ganglion to the vertebral column. This danger has not been stressed sufficiently and we feel that the following case should be recorded in order to focus attention on this potential problem.

CASE HISTORY

A 69-year-old female patient had a carcinoma of the pancreas diagnosed at laparotomy, at which time a cholecystojejunostomy was performed to relieve her jaundice. She was readmitted 14 months later in good general health because of increasing upper abdominal pain radiating to her back. Systemic analgesics gave no relief, and it was decided to perform a coeliac plexus block in an attempt to eliminate her pain.

The procedure was undertaken by one of us (S.K.L.), with an experience of nine successful similar blocks. With the patient in the prone position, the needle was inserted 7 cm from the midline at the level of the spine of L1, at an angle of 45° to the skin. After locating the body of L1, this angle was increased gradually until the bevel of the needle no longer made contact with bone. The needle was then advanced a further 1 cm, and the procedure repeated on the opposite side. Following careful aspiration, 5 ml of 6% aqueous phenol was injected very slowly through the needle on the right side. A similar injection was then commenced through the needle on the left side, but when only 1 ml of the solution had been injected, the patient complained of paralysis of both her legs and the procedure was abandoned. After she had been turned on to her back she was found to have total sensory and motor loss in both legs, but 2 hours later sensation and power had returned to her right leg. On the following day, however, her right leg was found to have deteriorated, although her abdominal pain had been relieved. Sensory loss and motor weakness in the L1-5 distribution were present, more marked on the left side. All leg reflexes were absent, as was vibration sense to the level of D12. Bladder control was lost also. X-rays of the lumbar and dorsal spine were normal, and it was decided that a myelogram was not indicated. During the following month she remained free from pain, and was treated with physiotherapy. However, there was no improvement in her neurological state, and she then developed intestinal obstruction and died following a haematemesis. An autopsy was not performed.

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DISCUSSION

It is difficult to explain the sequence of events in this case; a direct intrathecal injection seems unlikely in view of the negative aspiration, although it is conceivable that one could fail to aspirate c.s.f. from a small projecting dural sleeve in a paravertebral space. Injection in the vicinity of a lumbar artery at L1 seems the most likely explanation, transient vascular ischaemia occurring either as the result of vascular spasm, intramural injection, or because of an immediate increase in pressure in a confined space around the artery. Subsequent partial recovery followed by further deterioration might occur as the result of the sclerosing effect of the phenol.

The anatomy of the blood supply of the spinal cord is variable, and the major anterior radicular artery of Adamkewicz may be found anywhere between the levels of T8 and L4. This artery may supply the lower two-thirds of the spinal cord (Gray, 1973), and its occlusion may result in ischaemic necrosis of that portion of the cord. Restricted infarction of the cord may cause asymmetrical weakness and sensory impairment (Brain and Walton, 1969). Our knowledge of this subject has been enhanced recently by selective arteriography of the spinal cord. This technique has been used to evaluate obstructive vascular disease of the cord (Di Chiro, 1971), and there is a possibility that it may be used in confirming the diagnosis in cases where vascular occlusion is the suspected cause of the neural disturbance. However, this procedure might itself carry a small risk, as the blood supply to the cord in these patients is already in jeopardy, and the contrast medium may exert its own neurotoxic effect (Killen and Foster, 1966).

Bridenbaugh, Moore and Campbell (1964), had no serious complications in their series of 41 cases of coeliac plexus block employing alcohol. A local

anaesthetic agent was used initially to evaluate pain relief, and if this was successful, the block was repeated on the following day with alcohol. This seems a logical course of action to take, as the occasional unnecessary permanent block with its possible complications would be avoided. Unfortunately, this routine would not have prevented the catastrophe we report above, if vascular ischaemia of the spinal cord was indeed responsible for the clinical picture. Similarly, insertion of the needle under X-ray control would probably not have been of any benefit to this patient. This additional precaution, however, certainly carries no risk to the patient, and in view of our experience we intend to follow this practice in the future.

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