

PARAPARESIS DUE TO DILATED SPINAL COLLATERALS

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INTRODUCTION

Enlarged arteries in the chest wall serving as collateral blood supply in cases of coarctation of the aorta are well known. This condition may also be complicated by left ventricular failure, subacute bacterial endocarditis, rupture of the aorta and rupture of cerebral aneurysm leading to subarachnoid haemorrhage. However, spinal cord neurological complications are rare and little written on in the literature.

We wish to report a case of coarctation of the aorta who developed gradually progressive paraparesis. This was proven to be caused by pressure on the spinal cord from enlarged spinal collaterals.

CASE REPORT

The patient was a 55 year old man who gave a history of suffering from pain in the scapular regions which radiated down both arms for the past one year. He also noted progressive weakness of both legs. He experienced suprapubic pain and noted distension of the lower abdomen and dribbling of urine.

On examination, he was found to have a blood pressure of 180/100 in his arms. The carotid and upper limb pulses were very strong but the femoral pulses were weak and delayed compared with the radial pulses. The heart was enlarged and a systolic murmur was heard in the aortic area with radiation up the neck. The bladder was distended.

He was weak in both legs with absent tendon reflexes while the plantar reflexes were upgoing. He had loss of pain sensation up to the first lumbar dermatome. Vibration and position sensations were normal.

While in the ward, it was noted that the sensory loss rose rapidly to T3 level. Posterior column sensation remained intact.

A chest radiograph (Fig. 1) showed a moderately enlarged heart with left ventricular hypertrophy. A large rounded opacity filled the medial half of the right upper zone. Rib notching was obvious from the 4th to 9th ribs bilaterally.

Tomograms (Fig. 2) of the upper zone of the right lung showed the rounded opacity to have a smooth, well-defined lateral outline. Medially, it displaced the trachea to the left and there was a curvilinear calcification at the edge. The appearance was consistent with an aneurysm of the innominate artery.

No obstruction or intraspinal mass was detected at myelography. However, from C7 to T3 an enlarged and tortuous anterior spinal artery was outlined (Fig. 3).

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A thoracic aortogram was done via the left axillary artery. Coarctation of the aorta after the origin of the left subclavian artery and a large aneurysm of the innominate artery were confirmed. Besides the hypertrophied intercostal arteries, tortuous spinal collaterals were also demonstrated at the level of T2 and T3. Filling of the anterior spinal artery was not achieved. (Fig. 4-6).

The patient was considered to be too old for repair of the aortic coarctation. The spine was explored via laminectomy of T1 to T3. Dilated epidural vessels were seen. No further surgical procedure was considered possible. Post-operatively, he improved significantly over a period of several months.

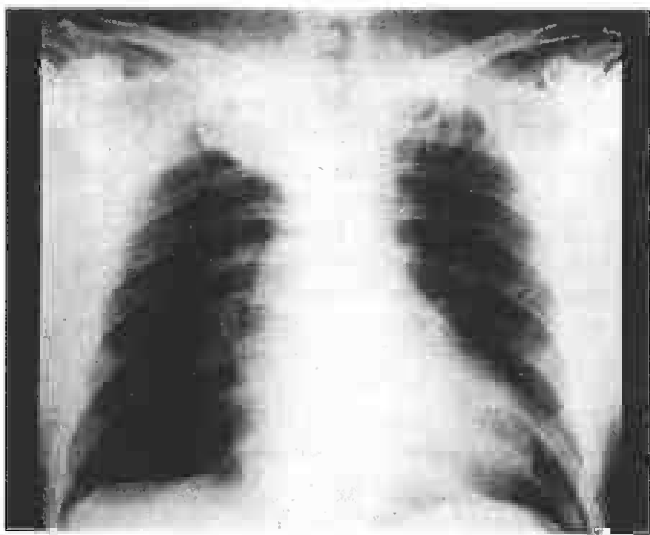


Fig. 1 Chest x ray shows moderate enlargement of the heart and bilateral rib notching. Note the rounded opacity in the medial half of the right upper zone.

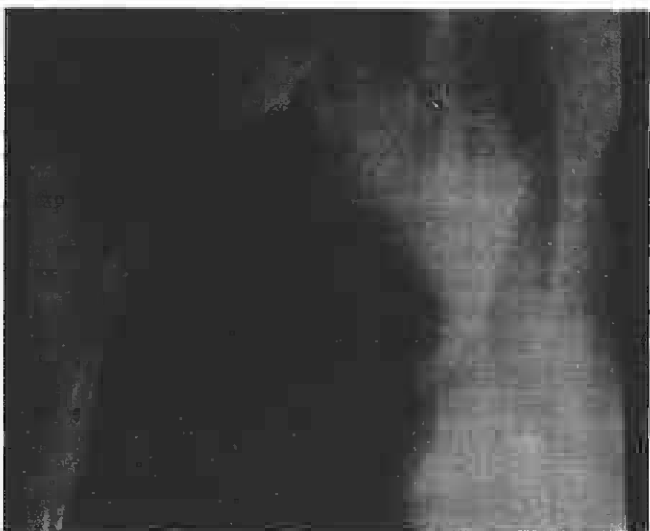


Fig. 2. Tomogram of the right upper zone shows the innominate aneurysm displacing the trachea to the left. A curvilinear calcification is present on its medial wall.

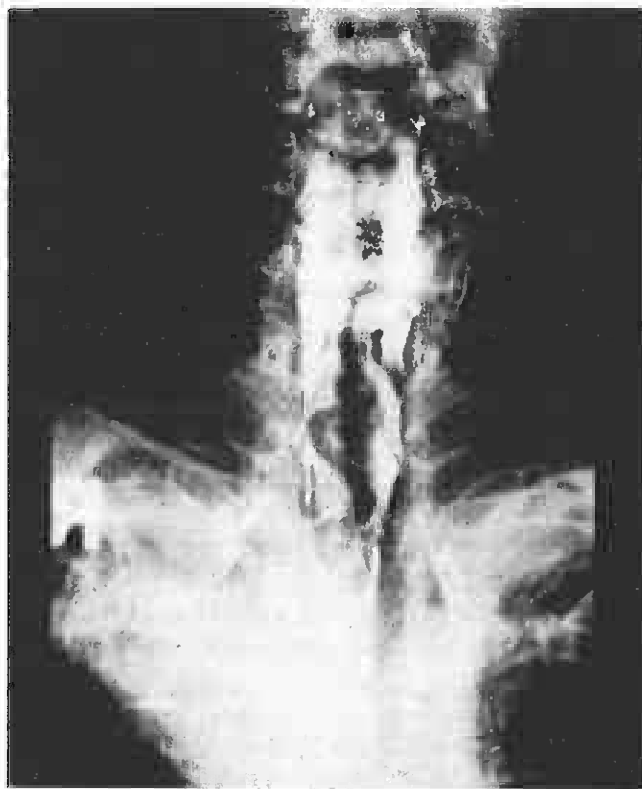


Fig. 3 Prone myelogram shows the enlarged and tortuous anterior spinal artery at the cervico-thoracic junction.



Fig. 4. Aortogram. Frontal view shows opacification of the innominate aneurysm. Arrow points to spinal collateral vessels.

DISCUSSION

According to Doppman et al (1969) only 11 cases of spinal complication in coarctation of the aorta had been reported since 1903. 7 patients had progressive paraplegia, of which 5 demonstrated sensory levels between T1 and T12. One patient was quadriplegic and had a sensory level at T4. Another patient had upper limb weakness with a sensory level at C7-T1. The tenth patient was reported by Herron et al (1958) and presented with a partial Brown-Sequard syndrome and a C7 sensory level. The last patient was simply described as a cervical cord compression syndrome.

Autopsy proof of pressure on the spinal cord by an enlarged anterior spinal artery was available in 4 patients. In two patients the neurological signs improved over a period of time after surgical correction of the coarctation. One patient showed spontaneous improvement (Bramwell, 1947) making the relationship with coarctation uncertain.

In 1973, Banna et al reported a 40 year old man with coarctation of the aorta who suffered three episodes of subarachnoid haemorrhage. Cerebral angiography and ventriculography were negative. He succumbed to the third episode of haemorrhage. At autopsy an enlarged anterior spinal artery was seen from C5 to T5. At the level of C6-7 a pseudo-aneurysm with a clot was seen involving the artery. The patient did not have focal neurological signs.

Doppman et al (1969) reviewed 40 aortograms in coarctation of the aorta. 7 patients had an enlarged and tortuous anterior spinal artery at the cervico-thoracic junction. The involved segment was relatively short. The artery emptied into enlarged radicular arteries. None of these patients had any neurological problems.

Our patient with coarctation showed the typical enlarged intercostal collateral arteries. Enlarged spinal collaterals around T1 and T2 were filled at aortography but not the dilated anterior spinal artery which was seen in the myelogram. Though his primary condition could not be remedied surgically, decompression of the spinal cord led to slow and incomplete but significant improvement of his neurological deficits.

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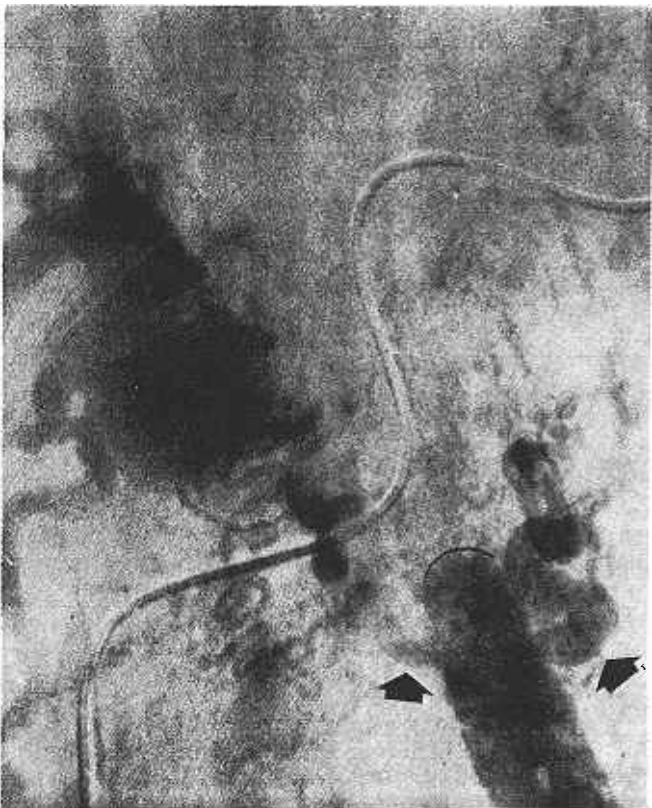
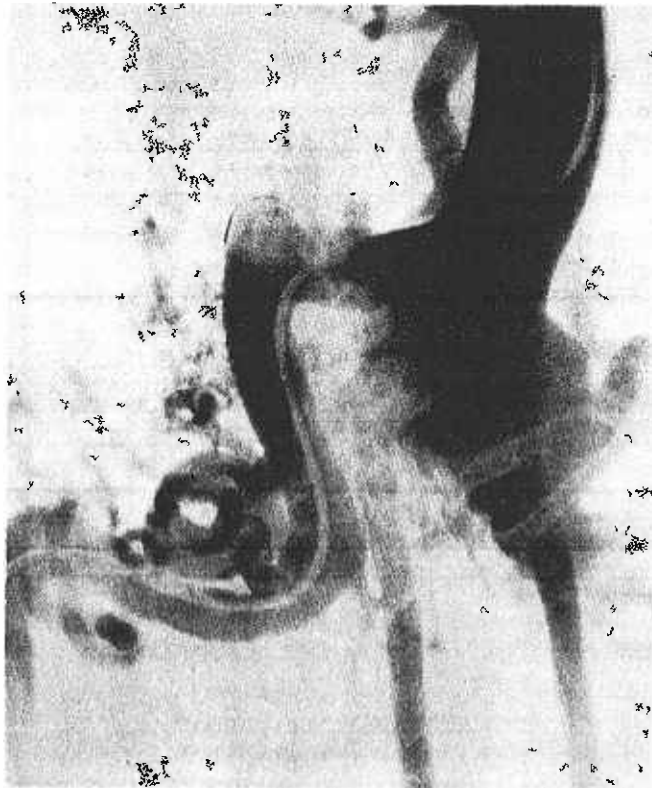


Fig. 5. Aortogram, oblique views. a) early phase shows the proximal aorta completely occluded after the origin of the left subclavian artery. b) later phase shows filling of the distal segment of aorta from huge intercostal arteries (arrows).