

Chylothorax after high translumbar aortography

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Dupont, P. A. (1975). *Thorax*, 30, 110–112. **Chylothorax after high translumbar aortography.** Two patients who had high translumbar aortograms for suspected arterial disease developed shortness of breath 48 hours after aortography and were found to have chylothoraces. Conservative management was initially adopted for both, but surgery had to be resorted to, although the first patient died before arriving in the operating theatre. The literature and the management of this condition are reviewed.

Chylothorax is now a recognized, though rare, thoracic complication of translumbar aortography. First described in 1955 by Maluf and McCoy, a further seven cases have been recorded in the literature (Gaspar and Secrest, 1957; Cook *et al.*, 1960; Schwarz, 1960; Wellmer and Schmitz-Dräger, 1963; Reinhardt, 1965; Biasi and Tomassini, 1968; Fagan and Robinson, 1971). Accidental puncture and injection of contrast into the thoracic duct or its tributaries without complication is of more common occurrence in most radiological departments specializing in translumbar aortography (Fischer, 1965).

Two cases are reported from a thoracic unit working in conjunction with a busy radiological department specializing in arterial investigations carrying out an average of 330 translumbar aortographies per year over the past ten years.

Conventional lumbar aortography is carried out by inserting the aortographic needle approximately 3 inches (76 mm) to the left of the midline posteriorly, aiming initially towards the L3 vertebra. In high lumbar aortography, however, the needle is introduced 1 inch (25 mm) nearer the midline, passed directly under the 12th rib and angled upwards and medially at an angle of 45 degrees until pulsation is felt.

CASE REPORTS

CASE 1 (1963) A 63-year-old emphysematous man was admitted for the investigation of lower abdominal pain thought to be due to a small aortic aneurysm. He underwent easy and uneventful high translumbar aortography by an experienced radiologist and the diagnosis of aortic aneurysm was not confirmed

radiologically. The patient was discharged home the following day fit and well but readmitted 48 hours later to another hospital with progressive shortness of breath and right-sided chest pain. There were signs of a large right pleural effusion with respiratory embarrassment. Chyle, 1.1 litres, was aspirated and daily thoracenteses were then performed, gradually increasing amounts of chyle being removed (Table I). During this time the patient received a normal diet with an unlimited fluid intake but by the eighth post-aortographic day this was supplemented by intravenous plasma and saline in view of falling levels of total plasma proteins and plasma sodium. The patient's condition gradually deteriorated, and immediately after transfer to the thoracic unit for surgery he went into acute congestive cardiac failure and died despite active medical treatment including repeat thoracentesis. Necropsy revealed gross mediastinal displacement by a very large chylous effusion (volume unrecorded) with kinked and obstructed venous return to the right heart and a collapsed right lung. Despite a careful search no laceration or perforation was found of an apparently intact thoracic duct system.

TABLE I
PATIENT 1: VOLUME OF CHYLE ASPIRATED AND
BIOCHEMICAL CHANGES

Days after Aortography	Vol. of Chyle Aspirated (ml)	Total Serum Proteins (g/100 ml)	Plasma Sodium (mEq/l)
		6.3 (pre-aortography)	
4	1100		138
5	850		139
6	1500		138
7	1500		132
8	2000	5.3	125
9	2400		123
10	3000		120
11	400+		

TABLE II
PATIENT 2: VOLUME OF CHYLE ASPIRATED, URINARY OUTPUT, AND BIOCHEMICAL CHANGES

Days after Aortography	Vol. of Chyle Aspirated (ml)	Urinary Output (ml)	Plasma Sodium (mEq/l)	Plasma Potassium (mEq/l)	Plasma Urea (mg/100ml)
			140 (pre-aortography)	3.4	42
4	850				
5	1500	630			
6					
7		490	127	5.1	190
8		220	124	4.5	205
		(IV therapy started)			
9		1980	121	4.9	180
10		1530	129	4.5	84
11	2300	350	134	4.3	49
12	1750+		132	4.2	54

CASE 2 (1972) A 56-year-old housewife was admitted for investigation of hypertension, repeated urinary infections, and intermittent claudication, mainly of the left leg. On examination an aortic bruit and absent leg pulses were noted. A high translumbar aortogram was performed with some difficulty. The following day she was discharged but later the same day she experienced right chest pain and the subsequent day became extremely dyspnoeic at rest with severe right chest pain. She was readmitted to hospital and a large effusion was found clinically, confirmed radiologically and by aspiration (volume unrecorded) shown to be chylous. A small effusion was also noted at the left base. Repeated thoracenteses were carried out while reducing the patient's oral fluid intake to cover her urinary and insensible fluid losses only and allowing her a high-protein but low-fat diet in an attempt to reduce her chyle production to a minimum. This regimen was successful in that re-accumulation was halted but owing to deterioration in the patient's condition, as reflected biochemically (Table II) by rising plasma urea and potassium levels, intravenous fluid replacement therapy was instituted with rapid improvement. On the eleventh post-aortographic day there was a sudden re-accumulation of chyle which was aspirated and the following day, as the effusion again rapidly re-accumulated, thoracotomy was considered necessary. At operation a small puncture in the pleura was found inferiorly in the right thoracic cavity, posterolateral to the descending aorta, from which chyle was draining. The puncture was oversewn but continued to ooze. The pleura was then widely opened and a lymph sac found, connected to the thoracic duct system by four trunks, one of which was perforated immediately adjacent to the sac. All four trunks were ligated, resulting in immediate stoppage of the chyle leak. The patient made an uneventful recovery.

DISCUSSION

During high translumbar aortography the most important aetiological factor predisposing to chylothorax is the insertion of the aortographic

needle too laterally into the back, enabling the needle to pass between the aorta and the vertebrae, endangering structures to the right of the sagittal plane (Schwartz, 1960). These structures include the right pleural cavity, the azygos vein, and the thoracic duct which at T 11-12 vertebral level lies to the right of this plane. However careful the insertion, an anomalous course of the thoracic duct or tributaries or an aberrant position of the cisterna chyli will predispose to damage and probably accounts for the left-sided chylothorax reported by Fagan and Robinson (1971).

Clinical suspicion should be aroused by the onset of shortness of breath and pain in the post-aortographic period. Clinical examination and radiology of the chest will confirm the presence of a pleural effusion but only chest aspiration will distinguish between a chylothorax and the more common haemothorax.

An aggressive policy should be adopted in the management of traumatic chylothoraces. Initially the condition can be managed by aspiration or preferably by continuous tube drainage. This can be supplemented for a trial period by oral starvation and intravenous alimentation which will result in a reduction of chyle production, hopefully permitting the fistula to close. Should these measures fail, or should the chylothorax show evidence of loculation, thoracotomy should be resorted to, preferably without delay (Ross, 1961). The first patient reflects the need for a logical policy, as no attempt was made to reduce chyle production despite increasing volumes of aspirate, and surgical aid was not requested until the patient's condition had deteriorated.

The second case shows that chyle production can be reduced and recurrence of a chylothorax can be prevented for a period of time. In this patient the fistula did not close spontaneously and surgery had to be resorted to.

TABLE III

SUMMARY OF REPORTED CASES: ONSET OF MAIN SYMPTOM, SITE OF EFFUSION, MANAGEMENT, AND PATHOLOGY

Author	Onset of Shortness of Breath after Aortography (h)	Site of Chylothorax	Management	Pathology
Maluf and McCoy (1955)	24	Right	Surgery	Duct divided
Gaspar and Secrest (1957)	96	Right-chylothorax Left-small, not investigated	Surgery	Duct partially severed
Cook <i>et al.</i> (1960)	96	Right	Drainage	—
Schwarz (1960)	48	Right	Surgery	Duct perforated
Wellmer and Schmitz-Dräger (1963)	96	Right	Aspiration	—
Reinhardt (1965)	48	Right	Aspiration	—
Biasi and Tomassini (1968)	24	Right	Drainage and aspiration	—
Fagan and Robinson (1971)	96	Left	Drainage and aspiration	—
Present paper (1963)	48	Right	Surgery proposed	PM—no abnormality found
(1972)	48	Right-chylothorax Left-small, not investigated	Aspiration and surgery	'Tributary' perforated

Of the eight cases recorded in the literature, five were managed successfully by conservative means consisting of aspiration and/or drainage (Table III). When surgery was undertaken, including the second case reported here, oversewing the pleura alone was not sufficient to stop leakage of chyle. In each case the pleura had to be opened and the duct and/or tributary isolated and ligated proximal and distal to the site of damage.

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