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TRAUMATIC INJURY OF THE THORACIC DUCT

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ABSTRACT

Injuries to the thoracic duct are infrequent but may become life-threatening when chylous leakage persists. This report describes 6 patients with such injuries in whom the leakage resolved spontaneously in one, was corrected using microsurgical lymphatic repair or lymphatic-venous anastomosis in two, successfully treated either by ligation of the thoracic duct or insertion of a peritoneo-venous shunt in two, and was eventually controlled after bilateral pleurodesis and thoracic duct ligation by insertion of a peritoneo-venous shunt in one. Conventional lymphography is superior to lymphoscintigraphy and is usually required to document disruption of the thoracic duct.

The thoracic duct is the primary central lymphatic collector for most visceral and peripheral lymph. Because it is anatomically protected by the ribs, sternum and clavicle and lies deep in the chest cage, it is seldom damaged by blunt or penetrating trauma. Thoracic duct disruption, however, is usually characterized by chylous effusion which when it persists may become life-threatening. Non-operative treatment has included insertion of drainage tubes, diuretic drugs, elimination of long-chain triglycerides in the diet and sodium restricted intake. If chylous leakage becomes intractable, however, several operative approaches have been advocated (1-3).

This report describes 6 patients with unusual thoracic duct injuries including two who were successfully managed using microsurgical repair of the disrupted duct in one and a thoracic duct-azygos venous shunt in the other.

CASE HISTORIES

Case 1 (Argentina)

A 24 year-old man sustained a stab wound to the neck, 2cm above the right sterno-clavicular joint. Except for a small neck hematoma neighboring structures were intact. Twenty-four hours later a second chest x-ray showed a left pleural effusion. A minimal thoracotomy in the seventh intercostal space yielded 1400cc of milky lymph.

Because the injury was in the right neck, direct lymphography (ultra-lipiodol) of the right arm was initially done, which was unremarkable. Conventional lymphography via the right foot demonstrated leakage of contrast media from the thoracic duct into the left thoracostomy tube *(Fig. 1)*.

On the 5th hospital day with persistently high lymph drainage (~1200cc/24 hours), operation was undertaken. Via a 10cm transverse incision above the left clavicle, the thoracic duct injury was located in the upper mediastinum and was repaired using 9-0 suture under a dissecting microscope *(Fig. 1 inset)*. Thereafter lymph drainage rapidly

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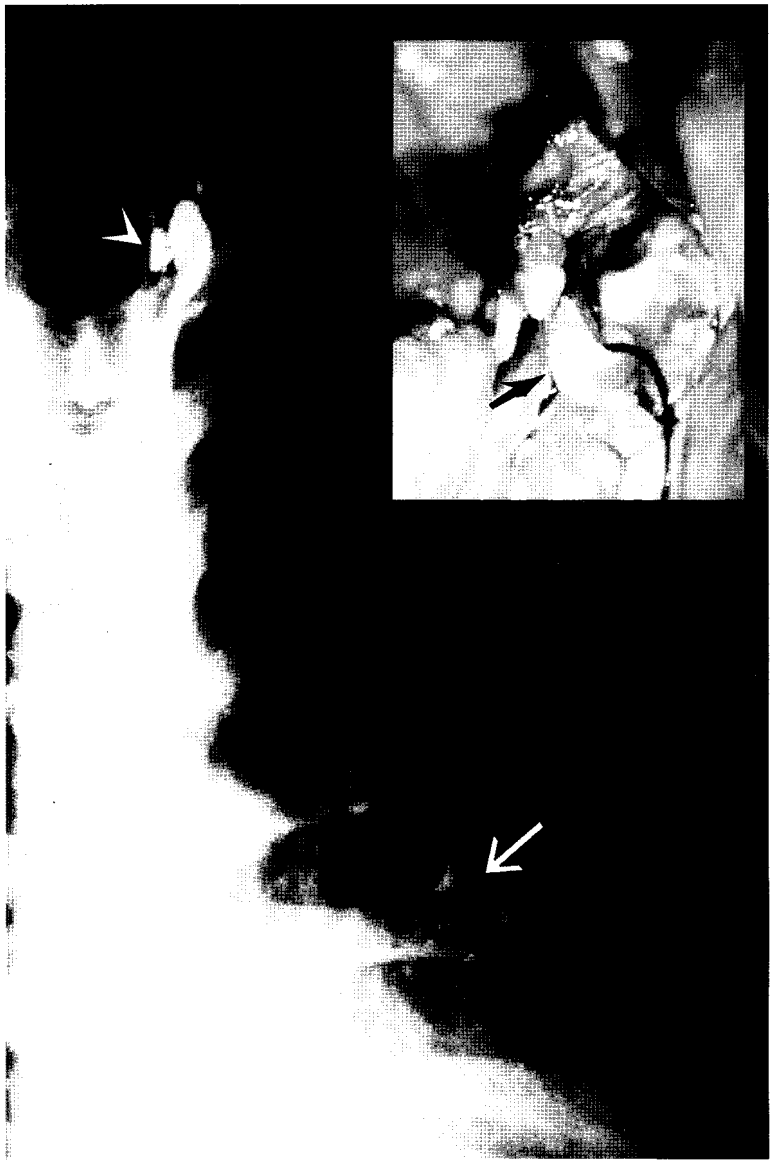

Fig. 1. (Case 1): Right lower extremity direct lymphography demonstrating leakage of contrast from the thoracic duct (arrowhead) exiting via left thoracostomy tube (white arrow). Inset shows a disrupted thoracic duct at operation (black arrow). Repair was accomplished using microsurgical tools and magnification and a simple over-and-over 9-0 suture.

decreased and stopped, and after 5 days the thoracostomy tube was removed.

Two weeks later, repeat direct lymphography demonstrated patency of the thoracic duct with a tiny lymphocele adjacent to the repair. One month later, he was well.

Case 2 (Argentina)

A 17 year-old woman was transferred from another hospital 36 hours after a motor vehicle collision. She was quadriplegic, had facial fractures and a traction injury of the

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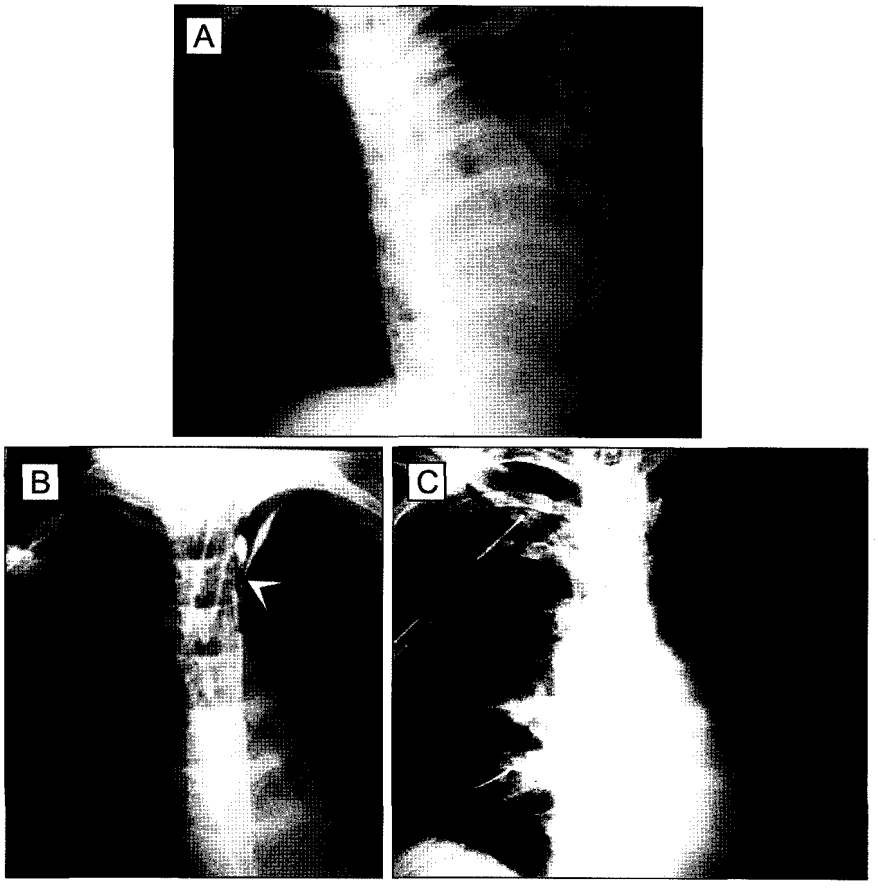

Fig. 2 (Case 2): A–Chest x-ray demonstrating left pleural effusion (chylothorax); B–direct lymphography shows disruption of the thoracic duct near the jugular-subclavian vein juncture (arrowhead); C–chest x-ray 5 days after a thoracic duct azygos vein shunt (see Fig. 3) with resolution of the chylothorax.

right brachial plexus. Plain x-rays and magnetic resonance images of the neck showed a fracture-subluxation of the vertebrae at C_4-C_5 with a central cord lesion. A chest x-ray *(Fig. 2A)* and ultrasound confirmed a left pleural effusion which initially was interpreted as a hemothorax. Because of worsening hydrothorax, a thoracentesis was done, which yielded chyle with a daily output of 2 liters. A pedal conventional lymphogram showed a disrupted thoracic duct at the thoracic inlet on the left side *(Fig. 2B, arrowhead)*. With inability to control lymph losses in this critically injured patient, a right-sided submammary thoracotomy was undertaken. The thoracic duct (~3-4mm in diameter) was isolated adjacent to the aorta *(Fig. 3A)*, and after adequate ductal mobilization, its distal segment was anastomosed end-to-side to the azygos vein using magnifying loops (4x) and 7-0 prolene *(Fig. 3B)*. The postoperative course was uneventful with normalization of the chest x-ray *(Fig. 2C)* and serum protein levels.

Case 3 (USA)

A 46 year-old man sustained acute pain in the left neck after an automobile "jack" slipped and the car partially compressed his upper chest and neck. Initially a fluid

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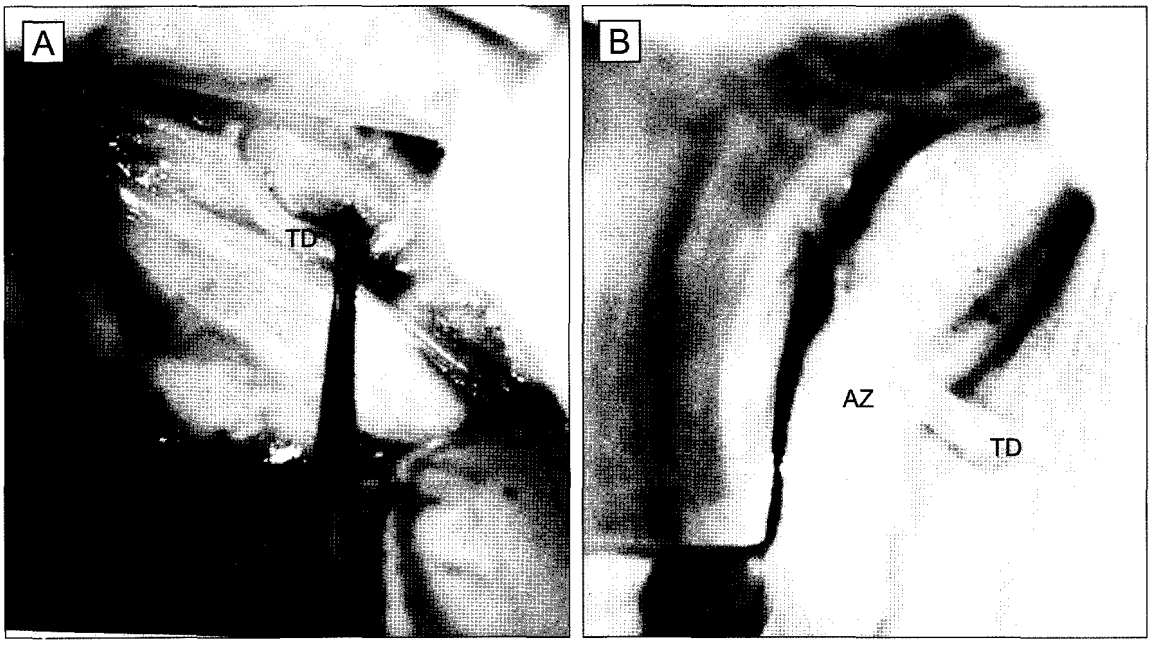

Fig. 3 (Case 2): A–Exposure of the intact thoracic duct (TD) in the right chest; B–end-to-side anastomosis of the TD to azygos vein (AZ).

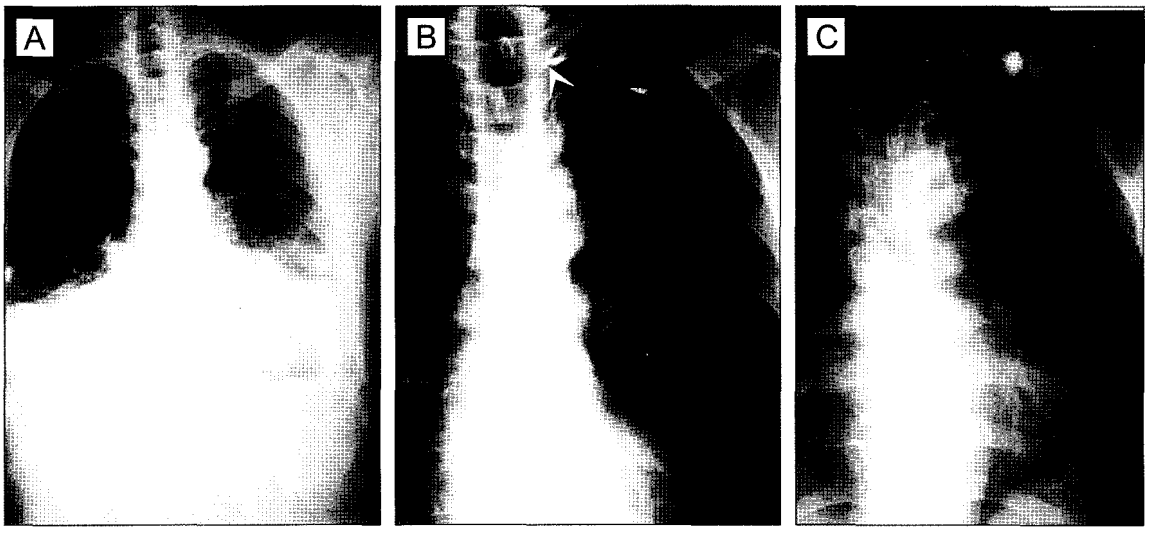

Fig. 4 (Case 3): A–Chest x-ray demonstrating bilateral chylous effusions after chest wall trauma. B&C–direct lymphography showing disruption of the thoracic duct (arrowhead) with spillage of contrast into the left axilla (C).

collection was noted along the left outer chest wall and axilla. Two days later, he complained of shortness of breath, and a chest x-ray revealed bilateral pleural effusions *(Fig. 4A)*, which on aspiration yielded chyle. Mild ascites was also detected. Lower extremity lymphangioscintigraphy showed prompt radiotracer transport but on delayed images

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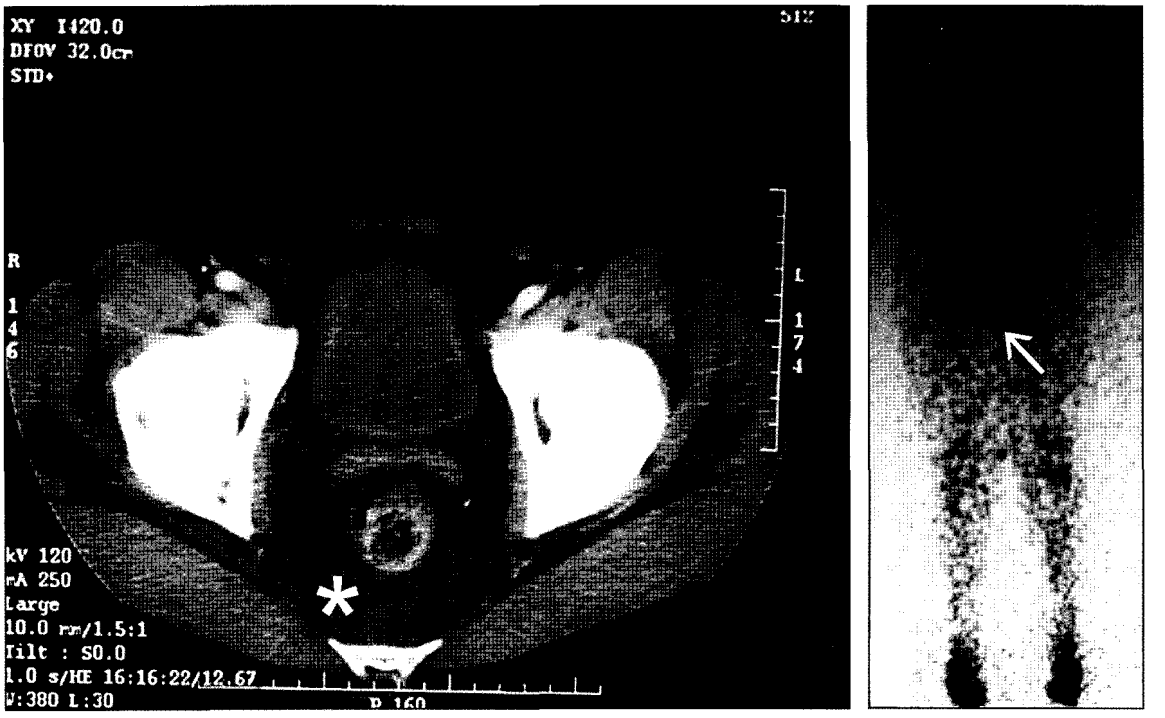

Fig. 5 (Case 3): A–Computer tomography of the pelvis showing retroperitoneal (perirectal) edema (asterisks); B–lymphangioscintigram confirming dermal backflow into the perineum (arrow).

tracer accumulated in the perineum *(Fig. 5, right)* consistent with retroperitoneal and perirectal edema as seen on computer tomography of the abdomen *(Fig. 5, left)*. Conventional lymphography confirmed disruption of the thoracic duct at the inlet with contrast extravasation into the axilla *(Fig. 4B,4C)*. After several days of non-operative management his signs and symptoms spontaneously regressed and he was discharged. He did not return for follow-up.

Case 4 (USA) [Reported previously in Lymphology 25 (1992), 62-68]

A 14 year-old boy sustained a fracture-dislocation of the spine (T_8-T_{10}) with paraplegia after a fall from a horse. After the vertebral column was stabilized via internal fixating rods, he developed a right chylothorax. Lymphangioscintigraphy and conventional lymphography both confirmed leakage of lymph from the lower thoracic duct exiting via a right thoracostomy tube *(Fig. 6)*. Because of a persistent high output fistula, the thoracic duct was ligated in the right chest just proximal to the site of disruption. Thereafter chyle leakage ceased.

Case 5 (USA)

A 75 year-old man underwent an uneventful replacement of an aortic valve for bicuspid stenosis but 3 weeks later he developed bilateral leg edema. He had no other previous medical ailment. Two weeks thereafter, ascites occurred, and when heart failure was eliminated as a causative factor, an abdominal paracentesis was done, which yielded chyle. Despite non-operative management, peripheral edema and ascites worsened. Conventional lymphography demonstrated huge intraabdominal lymphangiectatic "lakes" consistent with

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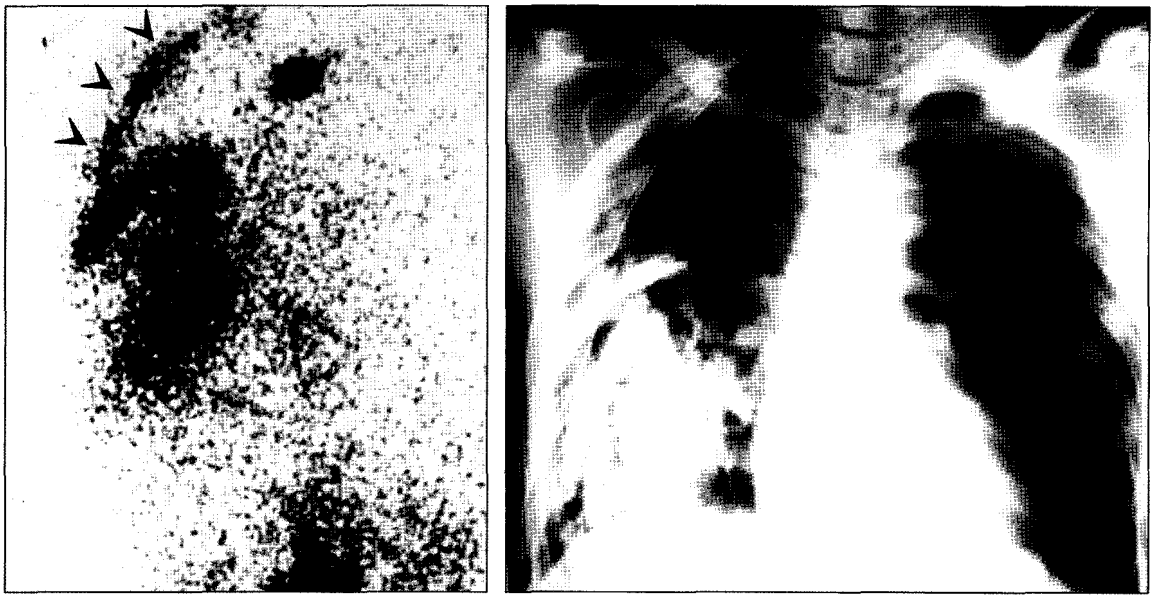

Fig. 6 (Case 4): Lymphangioscintigraphy (left) and conventional lymphography (right), both showing extravasation of marker into the right chest and exiting via a right thoracostomy tube from a disrupted thoracic duct.

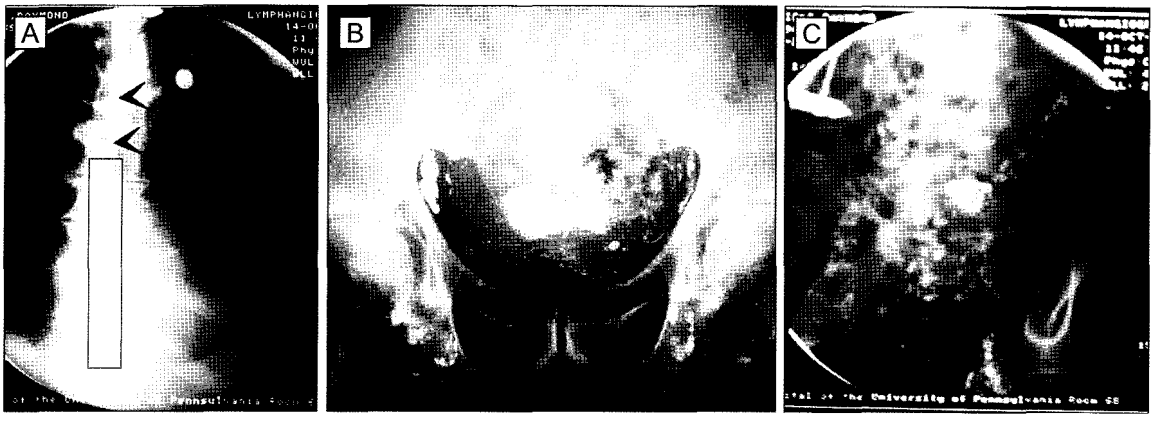

Fig. 7 (Case 5): A–Direct lymphography showing a segment of "missing" distal thoracic duct (bracket) with reconstitution of a thoracic duct in the upper mediastinum (arrowheads); B&C–massive lymphangiectatic "lakes" and free-floating contrast in the peritoneal cavity (ascites).

lymph stasis *(Fig. 7B,7C)*. Although the thoracic duct was reconstituted in the upper chest and left neck, it was consistently absent in its ductal portion above the aortic hiatus *(Fig. 7A)*. After seven months of non-operative management (dietary substitution of medium and short chain triglycerides for long-chain triglycerides, and sodium restriction, diuretic drugs and wearing of a compression garment) but with worsening malnutrition and weakness, a peritoneo-jugular venous shunt (LeVeen) was inserted. Thereafter, clinical improvement was dramatic and he gradually was weaned from dietary restriction and diuretics. A lymphangioscintigram 6 months later showed minimal radiotracer accumulation in the abdomen.

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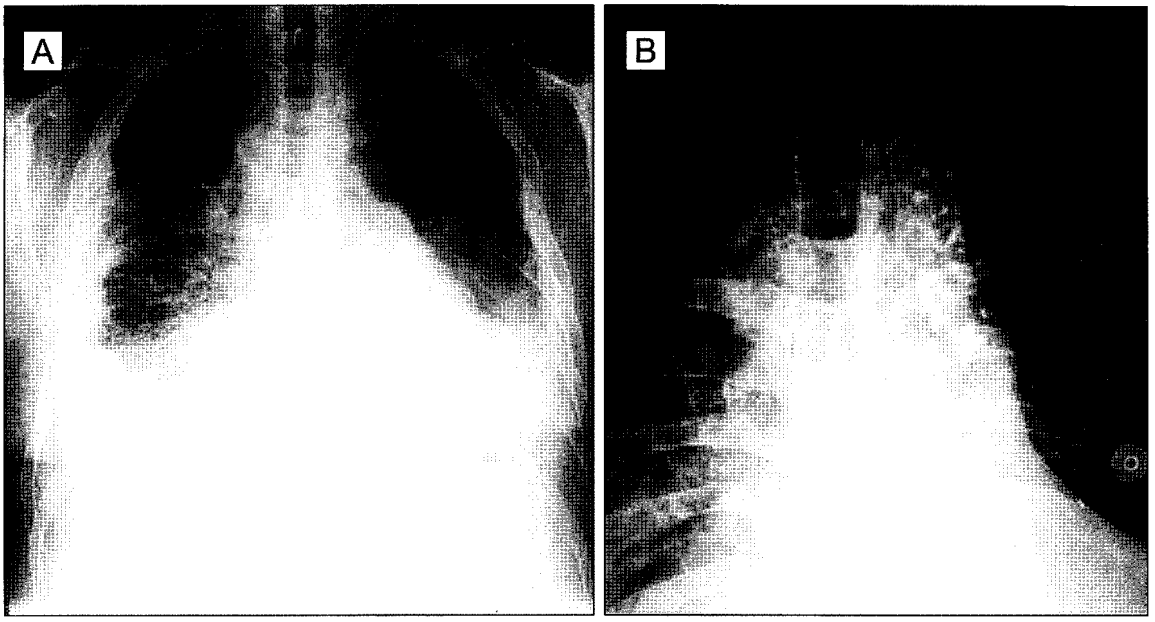

Fig. 8 (Case 6): A–Bilateral chylothoraces and chylopericardium 3 weeks after a motor vehicle collision; B–leakage of contrast from a disrupted thoracic duct near the aortic knob as shown by conventional lymphography.

Case 6 (USA)

A 34 year-old man was involved in a rollover motor vehicle collision and was restrained by a 3-point seat-belt. Initial evaluation was negative for a serious injury but 3 weeks later he complained of shortness of breath and swelling of both arms and the head and neck. A chest X-ray, echocardiogram, and insertion of thoracostomy tubes demonstrated bilateral chylothorax and chylous pericardium *(Fig. 8A)*.

Venography confirmed superior vena cava (SVC) thrombosis *(Fig. 9A)* with high grade stenosis of both innominate and jugular veins *(Fig. 9A and 9B)*. Tissue plasminogen activator was administered intravenously in both arms for 24 hours after which residual blood clot was extracted from the SVC using balloon angioplasty under fluoroscopy. Although patent, the innominate veins remained extremely narrowed. Because of persistent chylous effusions, a thoracotomy was done with drainage of the chylopericardium and insertion of talcum powder into both chest cavities for pleurodesis. These maneuvers were only partially successful. Conventional lymphography demonstrated disruption of the thoracic duct at the aortic knob with diffuse leakage of contrast into the mediastinum *(Fig. 8B)*. Six weeks later when thoracostomy output remained greater than 500cc for 24 hours despite dietary restriction (fat and salt) and diuretic drugs and with ongoing nutritional depletion, the thoracic duct was ligated in the right chest. Initially he did well but returned 6 weeks later with respiratory distress and a left chylothorax, ascites and bilateral leg edema. P^{32} was inserted into the left chest for effective pleurodesis but he remained with severely restricted lung disease, massive ascites and peripheral edema. A peritoneo-inferior vena caval (IVC) shunt was not possible because of a congenitally hypoplastic IVC. With great difficulty, the partially occluded right internal jugular vein was accessed percutaneously, and over a wire inserted into the SVC, a balloon angioplasty and insertion of a Wallstein stent effectively dilated and stented

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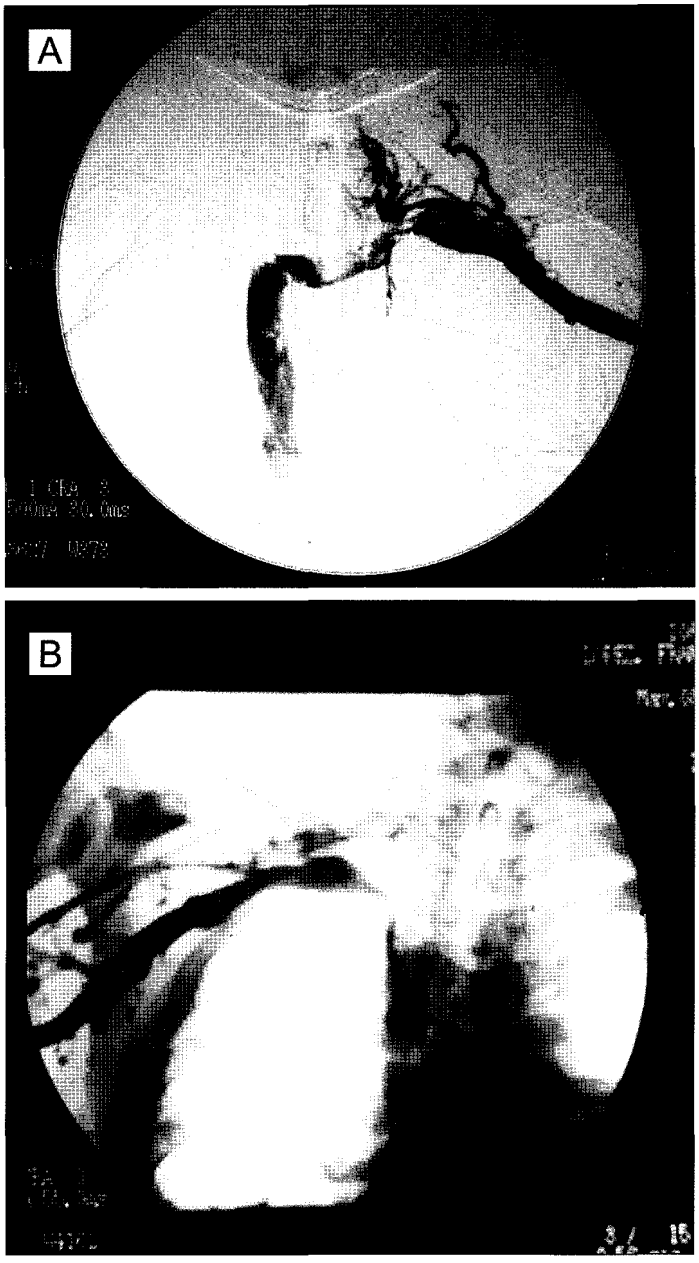

Fig. 9 (Case 6): A–Left arm venography shows occlusion of the left innominate/jugular/subclavian veins with thrombi in the superior vena cava; B–right arm venography shows stricture/stenosis of the right innominate vein.

the strictured right innominate vein. Thereafter, a peritoneal-SVC shunt (Denver) was able to be constructed through the nearly stented innominate vein. Five days later, a massive diuresis ensued and generalized anasarca remitted. Six weeks later, however, he was readmitted for chylous cardiac tamponade necessitating a subxiphoid pericardial window and tube mediastinostomy. A soft coagulum on the peritoneal-SVC shunt

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tip was disimpacted, the patient was anticoagulated (INR 2.71-3.5), and maintained on a fat restricted diet of short and medium chain triglycerides and aqueous vitamins A, D, and E. Pericardial drainage thereafter gradually ceased and the tube mediastinostomy was removed. Two months later, or almost 14 months from the time of initial injury, he remains asymptomatic except for mild peripheral edema.

DISCUSSION

From a clinical standpoint, the thoracic duct may be divided into an upper mediastinal — left-sided thoracic inlet segment and a right-sided portion lying within the complex of the aorta, esophagus and azygos vein adjacent to the vertebral column (4). A chylous effusion signifies a transport disorder of mesenteric (lacteal) lymph, which may have its origin in congenital lymphvascular anomalies (e.g., intestinal lymphangiectasia), obstructed lymphatics (e.g., lymphoma or melanoma), or injuries to lymphatic collectors (e.g., iatrogenic or surgical; blunt or penetrating trauma) (5).

Disruption of the thoracic duct with blunt trauma is usually associated with sudden flexion/hyperextension of the thoracic spine and stretching-tearing of the thin-walled central lymphatic tethered to the thoracic vertebral column (6,7). This explanation probably accounts for the pathogenesis in Cases 2 and 4, both of whom sustained serious spinal cord damage although in Case 1 the lower cervical and in Case 4 the lower thoracic vertebral column was fractured/dislocated. On the other hand, even relatively minor injuries to the back or chest (8,9), extreme Valsalva maneuvers (10) or birth trauma (11) may be associated with thoracic duct disruption. Case 3 sustained such an injury after sudden chest compression although the leakage proved self-limited and resolved spontaneously. The circumstances in Case 6, however, are even more complex. Presumably the shearing blunt force to the left neck and upper chest transmitted through the seatbelt not only caused disruption of the thoracic duct at the inlet to the mediastinum but also caused acute thrombosis of the superior vena cava and both innominate veins. Despite partial restoration of central venous flow, it is likely that alternative lymphatic collateral pathways were also damaged or their venous entry sites occluded.

Penetrating trauma from a variety of instruments and tools can damage the thoracic duct directly. Whereas operations on the esophagus, aorta and heart are implicated in less than 0.5% of chylothorax post-operatively (12), the true incidence of thoracic duct ligation after these procedures is probably underestimated (see below). Case 1 represents such direct trauma (stabbing) but the explanation for thoracic duct injury in Case 5 is more perplexing. The aortic valve replacement was uneventful, the operative incision was via a mid-line sternotomy and during placement of the patient on cardio-pulmonary bypass, the cava cannulae were inserted within the pericardium which is considerably anterior to the thoracic duct. Nonetheless, the temporal relationship to the operation is clear in that before the procedure, he was well except for bicuspid aortic stenosis, and after the valve replacement, developed intractable chylous ascites and peripheral edema with findings of an obstructed thoracic duct above the aortic hiatus.

Accumulation of chyle may begin to manifest itself clinically soon after injury (Cases 1-3) or be delayed for several weeks (Cases 4-6). The delay probably relates to build up of chyle within the mediastinum followed later by rupture through the mediastinal pleura (13) (Case 4) or along the aortic hiatus into the abdomen (Case 5). Although isotope lymphography (lymphangioscintigraphy) may document on occasion disruption of the thoracic duct (Case 4), conventional (direct) lymphography is more definitive and is usually required. After

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thoracic duct injury, spontaneous closure of the fistula may occur with parenteral feeding (Case 3) when there is no obstruction to more proximal flow of central lymph. When, however, fistula output remains high, operative correction is mandatory. Traditionally, ligation of the thoracic duct has been advocated with anticipation that collateral lymph-venous shunts will circumvent lower torso lymph stasis (14). Indeed, thoracic duct ligation in otherwise healthy experimental animals is usually well tolerated as demonstrated in Case 4. It is for this reason that the true incidence of thoracic duct injury after operations in the left chest is probably underestimated as occlusion may need not have overt clinical manifestations. On the other hand, interruption of a major vascular conduit is probably never desirable as illustrated by Case 5 where an interrupted distal thoracic duct led to lower torso lymph stagnation and by Case 6 where potential lymphatic-venous collateral pathways were probably blocked by phlebothrombosis of central mediastinal veins. It is also of interest that acute injury to the thoracic duct may be accompanied by delayed lymph return (?lymphangioparalysis) as seen on direct lymphography in Case 2 and in Case 3 by transient retroperitoneal edema. Accordingly, the approach taken in Cases 1 and 2 to restore thoracic duct lymph flow is a superior overall strategy. Lymphatic repair and/or anastomosis is feasible using magnification and microsurgical tools. Whereas long-term patency in patients with chronic lymphostatic disorders remains controversial, prolonged patency of lymphatic-venous shunts in otherwise healthy experimental animals is feasible (15). An alternative recommendation is lymph *nodal*-venous shunt (16). Whereas, an azygos vein shunt was considered in Case 6, particularly in light of compromised lymphatic collateral pathways, the persistence of superior vena cava hypertension (into which the azygos vein empties) and inflammatory changes in the right chest secondary to talc pleurodesis discouraged this option. This patient persisted with near complete blockage of lymph return from his lower torso until a peritoneo-venous shunt was successfully constructed in conjunction with dilation and stenting of a high grade stricture of the right innominate vein.

Other possibilities suggested for treatment of thoracic duct injury include pleuroperitoneal shunt for chylothorax (17) (implausible in Case 6 where mediastinal lymphatics were obliterated by poudrage and inflammation rendering absorption of peritoneal lymph via the diaphragm impossible). More recently, videoscopic thoracic surgery has been recommended (18).

In conclusion, although injury to the thoracic duct is relatively rare, the sequelae if chylous effusion persists can be life-threatening. Whereas thoracic duct ligation has long been advocated as operative treatment, this approach is not always innocuous, and where feasible, thoracic duct repair or anastomosis to the azygos vein using microsurgical tools and magnification is preferable. For accurate diagnosis of the injury, conventional lymphology is preferred over isotope lymphography.

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