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Cervical Lymph-venous Shunt in Experimental Ascites and in Patients with Hepatic Cirrhosis

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Formation of ascites in patients with hepatic cirrhosis develops when the rate of lymph formation exceeds the capacity of lymphatic drainage and excess hepato-splanchnic interstitial fluid "spills" into the peritoneal cavity. The reasons for the limited capacity of lymph drainage are not fully understood, but one postulate is restriction to thoracic duct lymph flow at the lymphatic-venous junction.

Accordingly, the present study examines the dynamics of the thoracic duct-jugular venous junction in dogs with ascites after constriction of the supradiaphragmatic inferior vena cava. Based on the abnormalities uncovered, we revised this junction in fifteen patients with ascites from hepatic cirrhosis intractable to medical treatment.

Methods

I. Experimental Studies

Ten mongrel dogs (weight 15-20 kg) were anesthesized with Nembutal (30 mg/kg) and the thoracic inferior vena cava constricted approximately 60-70% via a right thoracotomy. Two weeks later when ascites developed, the thoracic duct was exposed in the left neck. A side lymphatic channel was cannulated with fine polyethylene tubing, introduced into the thoracic duct and connected to an electro gauge (MP4-Nihon Kodhen-Tokio) for measurement of duct pressure. Venous pressure was similarly determined after introduction of a polyethylene catheter via a collateral vein into the innominate vein. Thoracic duct flow was recorded continuously using a non-cannulating electromagnetic flowmeter (OSMEA Torino-Italy). The pressure gradient between the cervical lymphatic-venous system was ascertained by subtracting the venous pressure from the thoracic duct pressure.

Vascular resistance to thoracic duct lymph flow was determined by the quotient of the thoracic duct minus innominate venous pressure (TDP – IVP).

In ten dogs, the thoracic duct was reanastomosed to the external jugular vein and these measurements repeated.

II. Clinical Studies

In fifteen patients (36-79 years) with hepatic cirrhosis (fourteen with nutritional and one posthepatitic cirrhosis) with massive ascites intractable to dietary salt restriction, diuretic drugs and repeated paracentesis, revision of the thoracic duct-jugular venous junction was performed under local anesthesia (five patients) or general anesthesia (ten patients) (Fig. 1). No fluids were infused during operation.

In five patients, splenic pulp pressure was determined by water manometry before and at six months after operation.

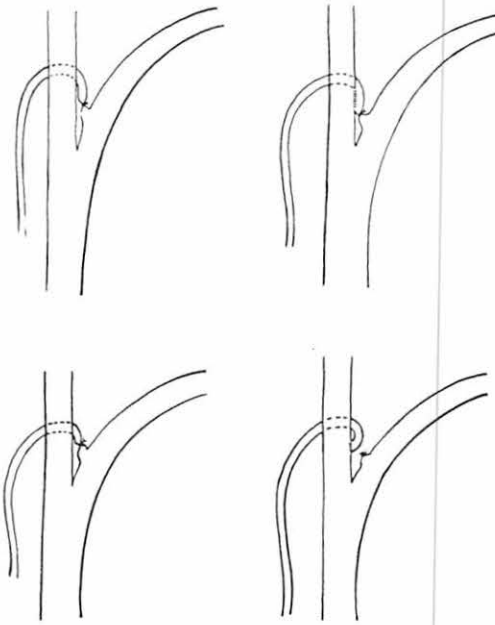

Fig. 1 Technique of thoracic duct-jugular venous anastomosis. An incision is made parallel to the left clavicle and the sternal and clavicular heads of the sternomastoid muscle are separated exposing the cervical portion of the thoracic duct. After isolating the outlet of the thoracic duct and the internal jugular vein, the duct is severed obliquely 0.5 to 1 cm from its venous junction or alternatively a double ligature is placed about the termination of the thoracic duct. An end-to-side or side-to-side anasthomosis is then fashioned between the resected or ligated duct and jugular vein using continuous atraumatic 5 - 0 dacron.

Results

I. Experimental Studies

Table I illustrates the thoracic duct lymph dynamics in normal and ascitic dogs before and after revision of the thoracic duct. Whereas dogs with ascites demonstrated increased thoracic duct lymph flow, there was an increased pressure gradient across the thoracic duct-jugular vein junction and an increased resistance to thoracic duct lymph flow. After transposition of the thoracic duct, however, vascular resistance fell markedly and was accompanied by a marked increase in thoracic duct lymph flow and a fall in the lymph-venous pressure gradient. Ascites uniformly disappeared.

II. Clinical Studies

The technique of thoracic duct-jugular venous anastomosis is shown in Fig. 1. After this operation in thirteen patients, ascites improved in three (23%) and completely disappeared in the other ten (77%) over the ensuing 3-21 months. One patient died of pneumonia and the other of gastrointestinal hemorrhage thirty and seven days after operation respectively. During the first 24 hours after operation, urine output increased on the average from 1050 to 1600 ml/24 hours and this increase was maintained in the surviving patients. The increase in urine output was accompanied by a decline in body weight. During the first 24 hours, there was no alteration in central venous pressure, blood pressure, or heart rate. All patients were continued on dietary salt restriction and varying dosages of spironolactone and furosemide.

Splenic pulp pressure fell in four of these patients from 28.3 ± 3.6 to 19.8 ± 2.2 cm water (Fig. 2). In each patient, lymphangiography demonstrated reduction in size and tortuosity of the thoracic duct (Fig. 3).

Table I Thoracic duct lymph flow (TDF), thoracic duct pressure (TDP), innominate venous pressure (IVP), thoracic duct-innominate venous pressure gradient (PG), and thoracic duct vascular resistance (R) (mean ± S.E.) in dogs with ascites from contriction of the thoracic inferior vena cava. Each group includes ten dogs.

Dogs	TDF (ml/min)	TDP (cmH_2O)	IVP (cmH_2O)	PG (cmH_2O)	R (units)
Normal	0.66 ± 0.2	2.5 ± 0.7	1.6 ± 0.4	1.0 ± 0.2	4.0 ± 1.5
Ascitic	1.9 ± 0.8	9.1 ± 0.8	1.8 ± 0.4	7.3 ± 0.3	4.0 ± 3.8
Ascitic with transposed thoracic duct	6.2 ± 1.0	6.0 ± 1.0	1.8 ± 0.2	4.2 ± 0.4	1.0 ± 0.6

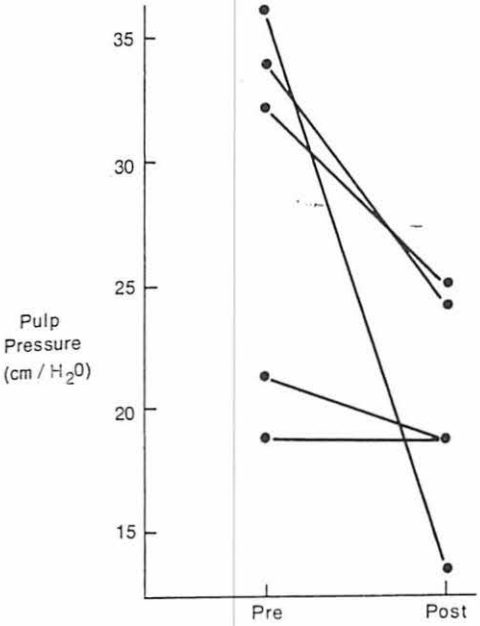

Fig. 2 Splenic pulp pressure before and six months after thoracic duct-jugular venous shunt for treatment of ascites in five patients with hepatic cirrhosis. In four of five patients, a marked reduction was observed.

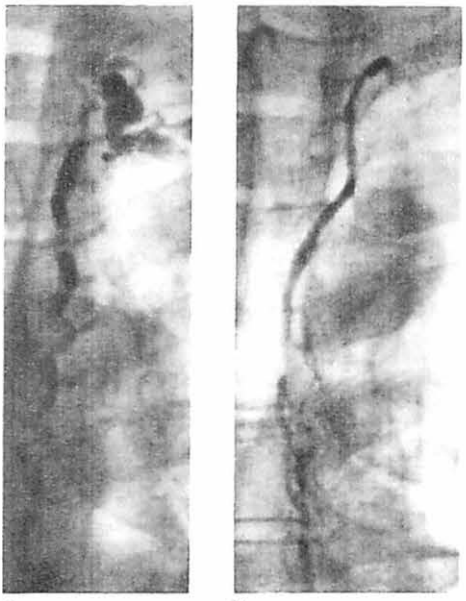

Fig. 3 Lymphangiogram before (A) and after (B) thoracic duct-jugular venous shunt in patient with hepatic cirrhosis. Note the diminution in size and tortuosity of the thoracic duct consistent with potentiation in lymph flow and decrease in lymphatic pressure. Patient's ascites disappeared.

Discussion

These experimental and clinical findings provide further support for the notion that narrowing at the thoracic duct-jugular venous junction represents a restriction to maximal lymph flow and thereby an important factor in the pathogenesis of ascites in hepatic cirrhosis. Whereas the thoracic duct dilates and becomes more tortuous, the opening valve-junction of the thoracic duct with the jugular-subclavian vein is probably restricted, remains only half open and later becomes "stenotic".

By revising the junction through reanastomosis of the thoracic duct to the jugular vein, the lymph-venous pressure gradient is actively enhanced. Vascular resistance declines and lymph flow rises. As the lymphatic-venous anastomosis is technically feasible only when the thoracic duct is dilated and constitutes a single channel in the neck, lymphangiography is recommended preoperatively to assess the anatomy of the cervical thoracic duct.

The relation of the thoracic duct-venous shunt to remission of ascites in these patients is very suggestive. Although each patient required salt restriction and diuretic drugs postoperatively, the same measures had been ineffective prior to operation. Moreover, in four of five patients, splenic pulp pressure decreased following successful lymph-venous shunt.

We conclude that in patients with cirrhosis a significant factor contributing to ascites formation is anatomic restriction to the maximal flow of splanchnic lymph at the thoracic duct-venous junction in the neck. Transposition of the thoracic duct into the jugular vein