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ORIGINAL ARTICLE

PORTAL HYPERFLOW IN PATIENTS WITH HEPATOSPLENIC MANSONIC SCHISTOSOMIASIS

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PURPOSE: The purpose of this study was to assess portal hemodynamics in patients with portal hypertension due to hepatosplenic schistosomiasis as well as to assess the contribution of splanchnic hyperflow to the pathophysiology of the portal hypertension.

METHODS: Sixteen patients with schistosomal portal hypertension and previous history of upper digestive bleeding due to esophageal varices rupture underwent elective esophagogastric devascularization and splenectomy and were prospectively studied. All patients underwent intraoperative invasive hemodynamic portal monitoring with a 4F-thermodilution catheter. The intraoperative portal hemodynamic assessment was conducted after laparotomy (initial) and after esophagogastric devascularization (final).

RESULTS: The initial portal pressure was elevated (mean 28.5 ± 4.5 mm Hg), and a significant drop of 25% was observed at the end of the surgery (21.9 ± 4.9 mm Hg). The initial portal flow was elevated (mean 1766.9 ± 686.6 mL/min). A significant fall (42%) occurred at the end of the surgical procedure (1025.62 ± 338.7 mL/min). Fourteen patients (87.5%) presented a portal flow of more than 1200 mL/min, and in 5 cases, values greater than 2000 mL/min were observed.

CONCLUSIONS: Esophagogastric devascularization and splenectomy promote a significant reduction of the elevated portal pressure and flow in schistosomal portal hypertension. These data favor the hypothesis of portal hyperflow in the physiopathology of portal hypertension of schistosomiasis.

DESCRIPTORS: **Hemodynamics. Portal hypertension. Portal system. Mansonic schistosomiasis.**

Mansonic schistosomiasis is a parasitic disease that affects more than 15 million individuals in Brazil. Hepatosplenic schistosomiasis (HS) is the most severe presentation of the disease, which is characterized by extensive splenomegaly, periportal fibrosis, portal hypertension, and upper digestive bleeding due to rupture of esophageal varices[1].

Development of portal hypertension (PH), regardless of its etiology, is due to increased vascular resistance or increased portal venous flow or both[2-5]. In cirrhosis and schistosomiasis, although increased vascular resistance appears to be the initial event, mesenteric hyperflow is important in maintaining portal hypertension[1,3-6]. The pathophysiology of PH also frequently displays a systemic hyperdynamic state[7-10] due to portal shunting of splanchnic vasoactive mediators[11,13-16].

Nevertheless, findings regarding portal hemodynamics in hepatosplenic mansonic schistosomiasis remain controversial. Total hepatic blood flow was reportedly preserved in previous studies[17,18,19,20]. It should be emphasized that none of those studies employed direct measurements[18,21-24]. Another important characteristic of schistosomal portal hypertension is extensive splenomegaly with dilated spleen vessels, suggesting that the splanchnic hyperflow is largely due to splenic hyperflux[22,23,25].

The purpose of this study was to assess portal hemodynamics with the thermodilution technique in patients with PH due to HS as well as to assess the contribution of splenic flow to the pathophysiology of the portal hypertension.

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METHODS AND PATIENTS

From June 1992 to July 1996, 16 patients with schistosomal portal hypertension (PH) and a previous history of upper digestive bleeding due to rupture of esophageal varices underwent elective esophagogastric devascularization and splenectomy and were prospectively studied.

The patients were hospitalized with the Liver and Portal Hypertension Group of the Division of the Digestive Tract Surgery of our Hospital. The Ethics Committee of the Hospital approved the study protocol, and all patients signed an informed consent.

The study included only patients with PH resulting from Manson's HS with esophageal varices and a history of upper digestive bleeding (hematemesis or melena) for more than 30 days. Patients included 8 males and 8 females, their ages ranging from 15 to 70 years (mean 39 ± 13). The diagnosis was based on epidemiological data (living in endemic regions of schistosomiasis in Brazil), history (previous contact with water contaminated by *S. mansoni*), physical examination (hepatomegaly with prominence of the left lobe, extensive splenomegaly, and collateral abdominal circulation), and laboratory evaluation (normal hepatocellular function). Upper digestive endoscopy was performed for detection of esophagogastric varices. The diagnosis was confirmed by identification of *S. mansoni* eggs on parasitological stool examination. Only those patients whose disease was confirmed (Symmer's fibrosis and granulomata with *S. mansoni* eggs) by histological study of the intraoperative hepatic biopsy were included in this series. The exclusion criteria were thrombosis of the portal, splenic, or superior mesenteric veins assessed by ultrasonography or arteriography; chronic viral hepatitis (B and C), with evidence of hepatocellular lesions confirmed by intraoperative biopsy; any type of heart disease; chronic alcoholism; and patients in endoscopic sclerotherapy programs.

All patients underwent elective esophagogastric devascularization and splenectomy. The procedure consisted of ligature of the splenic artery close to the body of the pancreas followed by splenectomy and devascularization of the distal esophagus (5 to 7 cm) and of the upper two-thirds of the stomach (proximal to the incisura angularis). During surgery, portal pressure (PP) and flow (PF) were measured using a 4F thermodilution catheter (94-010H-4F, Baxter Corporation, EUA) inserted from a jejunal vein through the inferior mesenteric vein, locating the thermistor close to the tip of the catheter beyond the portal bifurcation and the injection hole in the middle of the portal vein (Fig.1), similar to the technique employed for measurement of cardiac output with the pulmonary artery catheter. When the pulmonary artery catheter is properly placed, the injection hole is located at the right atrium, and the thermistor is located at the right pulmonary artery.

The PP was electronically measured using a Hewlett Packard model 1290 C pressure transducer, while PF was measured in triplicate using the thermodilution technique via the Hewlett Packard (model 783390A) Cardiac Output (CO) computer after the injection of 5 mL of 5% glucose at 0 to 4 °C. Portal pressure and PF were assessed after laparotomy (initial) as well as after esophagogastric devascularization and splenectomy (final).

In this study, a PP between 5 and 10 mm Hg and a PF between 800 and 1200 mL/min were considered normal[26]. Statistical analysis was accomplished using a paired *t* test, and *p* values <0.01 were considered statistically significant.

RESULTS

The individual results are reported on table 1.

The initial PP was elevated (mean 28.5 ± 4.5 mm Hg), and a significant drop of 25% was observed at the end of the surgery (21.9 ± 49 mm Hg). The initial PF was elevated (mean 1766.9 ± 686.6 mL/min). A significant fall

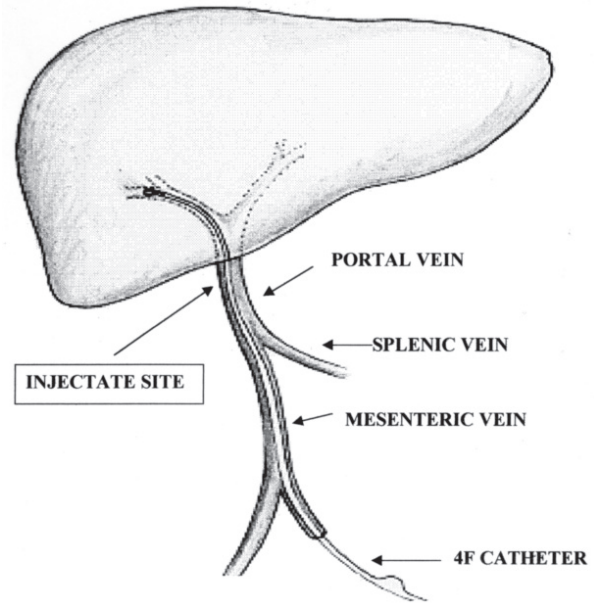

Figure 1 - Illustration showing the correct positioning of the 4F thermodilution catheter inside the portal vein.

Table 1 - Individual and mean results of portal pressure and portal flow before and after esophagogastric devascularization and splenectomy.

Case	SEX	PPi	PPf	FPi	FPf
01	F	29	17	2640	810
02	F	24	12	1500	980
03	F	26	23	1310	1000
04	M	30	20	3210	920
05	M	26	20	2336	1050
06	F	29	17	2590	1250
07	F	28	23	1930	1610
08	M	29	25	1270	1500
09	M	28	22	690	580
10	F	30	19	2240	1310
11	M	30	22	1090	1130
12	M	43	38	1850	500
13	F	30	23	1140	720
14	M	22	17	1380	540
15	F	27	25	1860	1400
16	M	26	26	1240	1110
Mean ± SD		28.6 ± 4.50	21.8 ± 5.7*	1767 + 686*	1026 + 339*

$p < 0.0001$
Ppi: Initial portal pressure; PPf: Final portal pressure; Fpi: Initial portal flow; FPf: Final portal flow

(42%) occurred at the end of the surgical procedure (1025.62 ± 338.7mL/min).). Fourteen patients (87.5%) presented a PF greater than 1200 mL/min, and in 5 cases, values greater than 2000 mL/min were observed.

DISCUSSION

Portal hypertension is a syndrome characterized by increased vascular resistance and/or increased portal venous flow[5,26]. Characteristically, portosystemic collateral circulation and digestive hemorrhage through esophageal varices occur independently from hepatocellular function.

The study of portal hemodynamics is very important for the understanding of the pathophysiology of portal hypertension in hepatosplenic mansonic schistosomiasis and for the determination of the physiological effects following surgical treatment. Since there is no consensus about the ideal surgical procedure for treatment of schistosomal portal hypertension, portal and systemic hemodynamic profiles could help to determine the best surgical technique for these patients—either esophagogastric devascularization and splenectomy or splenorenal distal shunt[27-31].

There are no other studies that have assessed intraoperative PF by thermodilution in schistosomiasis. In our series, both the initial PF (1767 ± 692 mL/min) and PP (28.5 ± 4.5 mm Hg) were elevated. These findings are in agreement with the theory that portal hyperflow plays a major role in the origin and maintenance of schistosomal portal hypertension[32-33].

Nine patients (56.25%) presented portal flow values greater than what is considered normal for total hepatic flow (1500 mL/min). These results are in disagreement with previous findings of other authors who report portal hyperflow with preserved total hepatic blood flow[22-25].

The 40% portal flow decrease at the end of the surgery (from 1767 to 1025 mL/min) suggests a greatly increased contribution of splenic flow to the total portal flow. Interestingly, our group had previously demonstrated that mansonic schistosomiasis presents a hyperdynamic systemic circulatory state that is also corrected by esophagogastric devascularization and splenectomy, demonstrating the importance of the spleen and collateral circulation in these circulatory states[34].

In conclusion, these data favor the hypothesis of portal hyperflow in the physiopathology of portal hypertension of schistosomiasis.

RESUMO

CLEVA R de e col. - Hiperfluxo portal na forma hepatosplênica da esquistossomose mansônica. **Rev. Hosp. Clín. Fac. Med. S. Paulo 59**(1):10-14, 2004.

OBJETIVOS: o objetivo do presente estudo é estudar a hemodinâmica portal em pacientes com hipertensão portal secundária a forma hepatoesplênica da esquistossomose e avaliar a contribuição do hiperfluxo esplênico na sua fisiopatologia

CASUÍSTICA E MÉTODOS: Foram estudados prospectivamente 16 pacientes portadores de hipertensão portal secundária à forma hepatoesplênica da esquistossomose mansônica com indicação de tratamento cirúrgico. Todos foram submetidos a avaliação hemodinâmica portal com cateter de termodiluição 4F antes e após a realização de desvascularização esofago-gástrica com esplenectomia.

RESULTADOS: Na avaliação intra-operatória inicial observou-se pressão (28,5 + 4,5 mmHg_) e fluxo (1750,59 ± 668,14 ml/min) portais iniciais bem acima dos valores considerados normais. Houve queda signi-

ficante de 25% na pressão (21,65 ± 5,55 mmHg) e de 42% no fluxo (1011,18 ± 332,73 ml/min) ao término da cirurgia. Quatorze pacientes (87.5%) apresentavam fluxo portal superior a 1200 ml/min e, em 5 casos, valores superiores a 2000 ml/min foram observados.

CONCLUSÕES: A pressão e o fluxo portais estão aumentados na hipertensão portal esquistossomótica. A desvascularização esofago-gástrica com esplenectomia reduz significativamente tanto a pressão quanto o fluxo portais. Estes dados favorecem a hipótese do hiperfluxo esplâncnico (esplênico e mesentérico) na fisiopatologia da hipertensão portal na esquistossomose forma hepatoesplênica.

DESCRITORES: **Hipertensão portal. Hemodinâmica. Sistema porta. Esquistossomose mansônica.**

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