Post-Bypass Extensive Ascites due to Splanchnic Bypass and the Effectiveness of Hyperalimentation Treatment

Veysel Temizkan, Murat Ugur, Alper Ucak, Gokhan Arslan, Ahmet Turan Yilmaz

Abstract
Reperfusion edema may develop in the early periods of chronic ischemic tissue reperfusion. Reperfusion edema may be represented after the splanchnic bypass with ascites, abdominal distension, and liver and kidney function impairment. In this article, we are reporting the hyperalimentation treatment and its results for the common ascites and hepatorenal syndrome, after a coeliac and superior mesenteric artery bypass.

Key words: Splanchnic bypass, edema, ascites, hyperalimentation

Introduction
The aim of the treatment of chronic mesenteric ischemia is to provide decreased mesenteric blood flow and protect patients from postprandial pain, weight loss and malnutrition. It is also to prevent acute life-threatening complications, like ileus. As conservative treatment is possible for the treatment of atherosclerotic diseases in the visceral organs, endovascular or surgical interventions can be applied to the diseased arteries [1,2].

In tissue that has been ischemic for a long time, venous and lymphatic drainage regress due to ischemia. After restoration of the flow, the reperfusion could cause post-bypass edema in the adaptation period. Post-bypass edema, which develops after bypass of the splanchnic vessels, may cause ascites and abdominal distension [1,3]. We are reporting our treatment strategy for common ascites developed in the patients who were operated on for coeliacus truncus (TC) and superior mesenteric artery (SMA) bypass.

Case Report
A 74-year-old female patient was admitted to our clinic with the complaints of postprandial pain, cytophobia, weight loss, nausea and vomiting. Her abdomen was
tender and intestinal sounds of the patient were hypoactive. Preoperative biochemical parameters were normal (Table 1).

The operation was planned according to the current findings of the patient who had a history of failed endovascular intervention toward TC and SMA previously (Figure 1). Following a standard upper and lower abdominal median incision, the aorta was explored. A TC and SMA bypass operation was then performed with a 16/8 mm bifurcated Dacron graft. The Dacron graft’s proximal end was anastomosed to the aorta. While one leg was anastomosed to TC, the second leg of the bifurcated graft was passed behind the pancreas, and then was anastomosed to the SMA.

The patient was re-operated on the postoperative 2nd day due to abdominal distention and higher liver functional tests. In the examination, TC bypass was occluded; the leg of the bifurcated graft, anastomosed to TC, was then transected and endarterectomy was applied to TC. Embolectomy was performed on the TC with a Fogarty catheter but significant backflow could not be obtained and the arteriotomy of TC was primarily closed. An 8 mm PTFE graft was then prepared and the proximal end of this graft was anastomosed to the splenic artery. The distal end of this PTFE graft was anastomosed to the hepatica propria artery. One arm of the bifurcated graft, previously anastomosed to TC, was anastomosed to the middle part of the 8 mm PTFE graft. The operation was finished after a drain was placed. The patient was extubated on the 14th postoperative hour.

Compared to the first and second days, drainage followed an increasing trend that became 1000 ml on the 3rd postoperative day. After the liver enzymes peaked on the 2nd postoperative day, they started to decline on the 3rd postoperative day. In abdominal ultrasonography, portal vein flow and vascular structure were normal, although their pressures were high on the 3rd postoperative day. The patient’s status was evaluated as a hepatorenal syndrome (HRS) developed as a result of the post-bypass edema. Hepatamine and diuretic treatment was initiated with amino acid-rich fluid for the patient who had low total protein and albumin values (Table 1). Due to intestinal sounds being hypoactive, hyperalimentation and albumin treatment was started with total parenteral nutrition (TPN) (of 2000 kcal/day). Urine output of the patient that was oliguric on the 3rd postoperative day increased gradually to 1200 ml/day and 2500 ml/day on the following days, respectively. Drainage, on the other hand, increased even more to 2200 ml and 5400 ml; it then started to decrease and fell to 550 ml on the 6th postoperative day and finally 200 ml on the 7th postoperative day. In examinations on the 7th postoperative day, the total protein increased to 5.9 g/dl and albumin increased to 3.5 g/dl; transaminase and creatinine levels fell to normal.

Table 1. Biochemical parameters of the patients.

<table>
<thead>
<tr>
<th></th>
<th>Preop</th>
<th>PO 1</th>
<th>PO 2</th>
<th>PO 3</th>
<th>PO 4</th>
<th>PO 5</th>
<th>PO 6</th>
<th>PO 7</th>
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<th>PO 9</th>
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<th>PO 11</th>
<th>PO 12</th>
<th>PO 13</th>
<th>PO 14</th>
<th>PO 15</th>
<th>PO 16</th>
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<tr>
<td>Platelet (10^9 /ul)</td>
<td>265</td>
<td>216</td>
<td>118</td>
<td>82</td>
<td>61</td>
<td>29</td>
<td>12</td>
<td>60</td>
<td>31</td>
<td>106</td>
<td>150</td>
<td>210</td>
<td>370</td>
<td></td>
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<tr>
<td>Urea (mg/dl)</td>
<td>34</td>
<td>140</td>
<td>90</td>
<td>154</td>
<td>174</td>
<td>123</td>
<td>93</td>
<td>79</td>
<td>68</td>
<td>70</td>
<td>65</td>
<td>57</td>
<td>27</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Creatinine (mg/dl)</td>
<td>0,8</td>
<td>1,1</td>
<td>1,4</td>
<td>1,91</td>
<td>0,7</td>
<td>0,6</td>
<td>0,6</td>
<td>0,09</td>
<td>0,4</td>
<td>0,4</td>
<td>0,5</td>
<td>0,6</td>
<td>0,7</td>
<td></td>
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<tr>
<td>SGOT (u/l)</td>
<td>26</td>
<td>289</td>
<td>2241</td>
<td>691</td>
<td>203</td>
<td>98</td>
<td>34</td>
<td>29</td>
<td>32</td>
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<tr>
<td>SGPT (u/l)</td>
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<td>233</td>
<td>958</td>
<td>358</td>
<td>129</td>
<td>85</td>
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<td>25</td>
<td>20</td>
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<td>24</td>
<td>26</td>
<td>37</td>
<td></td>
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<tr>
<td>Total Protein (g/dl)</td>
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<td>-</td>
<td>4,1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>5,9</td>
<td>-</td>
<td>-</td>
<td>6,1</td>
<td>7,8</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Albumin (g/dl)</td>
<td>4</td>
<td>-</td>
<td>-</td>
<td>2,7</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>3,5</td>
<td>-</td>
<td>-</td>
<td>3,7</td>
<td>4,1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Urine (cc)</td>
<td></td>
<td>2200</td>
<td>1700</td>
<td>850</td>
<td>1200</td>
<td>2500</td>
<td>2200</td>
<td>2800</td>
<td>-</td>
<td>-</td>
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<td>-</td>
<td></td>
<td></td>
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<tr>
<td>Drainage (cc)</td>
<td></td>
<td>-</td>
<td>300</td>
<td>200</td>
<td>1000</td>
<td>2200</td>
<td>5400</td>
<td>550</td>
<td>200</td>
<td>-</td>
<td>1500*</td>
<td>500*</td>
<td>-</td>
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* fluid drained by paracentesis. (Preop: Preoperative, PO: Postoperative).
limits and only the value of urea (urea: 79 mg/dl) was evaluated as being slightly over normal. In the ultrasoundography control of the abdomen, portal pressures were higher but they were found to be lower than before. After the patient’s oral feeding was organized with a protein-rich diet, TPN was discontinued.

During the postoperative follow-up, the platelet count fell to 12000 on the 6th day and to 31000 on the 8th postoperative day; as a result of this, randomized thrombocyte replacement was made consisting of 6 units twice daily in this period. On the 7th postoperative day when the drainage fell to 200 ml, drains were taken to prevent liquid and protein loss from the peritoneum by maintaining the peritoneal hydrostatic pressure. On the following days, acid follow-up was made by the measurement of the abdominal circumference from the level of umbilicus and no increase was seen in the abdominal circumference of the patient in these follow-ups. A serious leak that continued as an ongoing littering for the first days of 35-40 pads was cut off gradually in a week. In the paracentesis made on the 9th and 11th postoperative days due to abdominal distention, 1500 ml and 500 ml of fluid was drained, respectively. The total drainage was 13500 ml for 14 postoperative days. On the 18th postoperative day, the patient was discharged with normal kidney and liver function tests, a platelet count of 370000, a total protein value of 7.8 g/dl and an albumin value of 4.1 g/dl. In the control examinations made on the patient during the 6th postoperative month and the first postoperative year, renal and liver functions were normal and grafts were patent in the abdominal tomographic angiography (Figure 2).

**Discussion**

Stenosis of visceral organs is a rarely diagnosed pathology and it is generally asymptomatic due to the extensive network of collateral arteries. If one of TC or SMA occlusion or stenosis occurs, pancreaticoduodenal arteries are the most important origin for collateral arteries. In SMA occlusion, blood flow goes to the gastroduodenal artery through the hepatic artery and to SMA from there through the superior and inferior pancreaticoduodenal artery. In case of a TC occlusion, TC flow may be provided the same way. In case of an SMA and TC occlusion, flow can be directed to these arteries through IMA. Flow, in this situation, can be redirected to the marginal arteries of Drummond through the Riolan arch (the ascending branch of the left colic artery), reaching the middle colic and pancreaticoduodenal arteries [1,2]. Symptoms generally occur when there are lesions in at least two of these three arteries [2,3]. Intestinal angina, weight loss and cytophobia are common clinical features. Abdominal pain typically occurs after 15-60 minutes from a meal and it can last 1-4 hours. Weight loss occurs to decrease angina as a result of the decrease in the food intake. Despite the rare occurrence of the symptomatic intestinal ischemia, atherosclerotic stenosis or occlusion of visceral arteries are not very uncommon. It was reported that 6-10% of the patients had at least 50% and over stenosis in at least one of their three arteries according to their autopsy results [3].

Endovascular or surgical procedures may be done for the treatment of atherosclerotic diseases of splanchnic arteries. Bypass surgery may be performed antegrade from the thoracic or supracoeliac aorta or retrogradely from the infrarenal aorta or iliac arteries. In the 3.5 years of follow-up results of a study that evaluated the endovascular angioplasty and open surgery, perioperative complications and hospital stay were found to be lower in the angioplasty group, but no significant difference was seen between two groups during the follow-up period [4].
It should not be forgotten that post-bypass edema, which is expected after lower-extremity revascularization, may develop after revascularization of the splanchnic vessels. In abdominal distension, which may develop after endovascular or surgical revascularization of the visceral organs, medication should be organized by keeping post-bypass acid in mind. When the signs of post-bypass edema develop, medical treatment to increase the plasma oncotic pressure should be started. In a patient with severe ischemia before the revascularization, abdominal pain, tachycardia, leukocytosis and an intestinal edema-characterized “revascularization syndrome” may occur in the postoperative period [3]. This is due to the post-bypass edema. There are two main reasons for post-bypass edema. The first reason is the lymphatic loading due to the increase in the interstitial fluid after the reconstruction; the second reason is the damaging of deep and superficial lymph vessels during the dissections. Distal blood pressure increases due to development of vasodilation after reperfusion. Due to increased capillary permeability, interstitial edema and luminal fluid accumulation occur. The transition of macromolecules to the interstitial area causes lymphatic loading (Table 2). Damaged intestinal microcirculation loses its resistance against bacteria like it loses it against water; bacterial translocation, endotoxemia or bacteremia thus occur. This bacterial translocation may cause the development of a systematic inflammatory response syndrome, an adult respiratory distress syndrome or a cardiac dysfunction.

HRS is the development of renal failure in the acute or chronic severe liver disease despite there being no clinical, laboratory or histological reason for its development. The principal finding in HRS is vasoconstriction in the renal vascular bed without an effect on renal tubular functions and a decrease of renal perfusion as a result of this. The decrease in splanchnic vascular resistance and arterial vasodilation reduces effective arterial blood volume. As a result of this, vasoconstrictor systems, such as the renin-angiotensin aldosterone system and sympathetic nervous system, are triggered and renal vasoconstriction, which is the principal finding of HRS, develops [5,6]. A combination of renal vasoconstriction and systematic vasodilation, and a decrease in systematic vessel resistance and arterial blood pressure despite the increase of activity in vasoconstrictor systems (e.g. renin-angiotensin system and sympathetic nervous system) are characteristics of HRS [7].
Splanchnic arterial vasodilation may be compensated with hyperdynamic circulation. Splanchnic vasodilation increases portal venous system return and portal pressure. Portal hypertension triggers vasodilation and hyperdynamic circulation. As a result of this vicious cycle, the increase of hydrostatic pressure and splanchnic capillary permeability cause ascites formation [8].

HRS is characterized with low artery pressure, increased plasma renin, norepinephrine and antidiuretic hormone (ADH) levels and a low glomerular filtration rate (<40 ml/min) [8]. It has two types: Type 1 HRS is the form that is characterized with a creatinine value higher than 2.5 mg/dl. It can develop spontaneously or it may be related to a bacterial infection, gastrointestinal hemorrhage or may be associated with acute hepatitis, which is induced by cirrhosis. Type 2 HRS is characterized with moderate renal insufficiency (serum creatinine <2.6/dl). Type 2 HRS is related to liver failure and arterial hypotension, but this relationship is relatively weaker compared to type 1.

Medical treatment is preferred in the treatment of post-bypass edema and its complications. Disorder in the microcirculation balance is corrected within the first week by way of medical treatment and follow-up. HRS may be treated with volume expanding agents, vasopressin derivatives (ornipressin or terlipressin) or alpha-adrenergic agonists (noradrenaline, midodrine) [5,8].

Impairment in the effective arterial blood volume and decrease in venous return and cardiac output due to splanchnic vasodilation are responsible for HRS pathogenesis. For this reason, it should not be surprising that it responds to intravenous albumin and vasoconstrictor treatment [8]. The ornipressin and albumin combination increases renal perfusion and GFR by neutralizing increased vasoconstrictor activity [6]. We also increased oncotic pressure with a protein-rich diet and hyperalimentation treatment in our patient with HRS after visceral bypass. Thus, we have contributed to the reduction of ascites amount by shifting extravasated fluid to the intravascular area. Clinical findings of the patient improved dramatically after initiation of the hyperalimentation treatment and the patient was discharged on the 18th postoperative day.

Post-bypass edema, which develops after revascularization of the splanchnic arteries, may cause ascites and hepatorenal syndrome. When HRS develops, hyperalimentation treatment decreases the symptoms by increasing oncotic pressure. We hypothesized that a protein-rich high-calorie diet, which will increase the plasma oncotic pressure, would be beneficial, especially in the treatment of reperfusion syndrome, which develops after the transperitoneal approach.

**Conflict of interest statement**
The authors do not declare any conflict of interest or financial support in this study.

**References**

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