Decompressive shunts and hepatic encephalopathy

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Encephalopathy is a major complication of all decompressive procedures done to reduce portal pressure. There are two major groups of decompressive procedures: surgical portosystemic shunts and transjugular intrahepatic portosystemic shunts (TIPS). Surgical decompressive shunts are of three types: total, partial, and selective, depending on the amount of hepatoportal flow that is maintained in each of them. Encephalopathy with these shunts occurs because of reduction in hepatoportal flow. These shunts have failed to reduce mortality; in fact, some studies have shown an increase in mortality following shunts. TIPS has more or less replaced the need for surgical shunts, but their risk to cause encephalopathy is almost equivalent to that of selective shunts and in some series is even more. Lactulose, antibiotics and protein restriction can easily control severe encephalopathy as a consequence of decompressive shunts. [Indian J Gastroenterol/2003;22(Suppl 2):S21-S24]

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The occurrence of encephalopathy following decompressive shunts in portal hypertension depends on various factors, like the type of underlying disease (cirrhosis or non-cirrhotic liver disease), severity of underlying disease, and type of shunt done (total, partial, or selective). It is however a common problem and severe grades of encephalopathy are seen.

There are two major groups of decompressive procedures that are done for reducing portal pressure: surgical portosystemic shunts and transjugular intrahepatic portosystemic shunts (TIPS).

Surgical portosystemic shunts

Surgical decompressive shunting for portal hypertension was introduced as a definitive solution for reducing portal pressures and the related bleeding.1 The operations were based on the principle of diversion of blood away from the collateral circulation, mainly from esophago-gastric varices, and simultaneous decompression of the portal venous system. These shunts are very effective for control of bleeding and rebleeding and in treatment of ascites.2-7 Unfortunately, there is no increase in survival and there are associated complications, the most distressing being encephalopathy.

Surgical decompressive shunts fall into three categories, viz., total,3-7 partial,8-10 and selective.11,12

Total portosystemic shunts or nonselective shunts

Total portosystemic shunts decompress all parts of the portal system and the cirrhotic liver is deprived of all its portal flow. Total portosystemic shunts include end-to-side portocaval shunt, side-to-side portocaval shunt, mesocaval shunt with or without autologous vein or Gore-Tex interposition, central splenorenal shunts, and percutaneous inferior vena cava-to-portal vein shunts (PIPS).2,11,12 Side-to-side portocaval shunt is different from end-to-side portocaval shunt in that it decompresses both the hepatic and sinusoidal beds and is therefore able to alleviate ascites.8 The mesocaval and central splenorenal shunts physiologically behave as side-to-side portocaval shunts. These shunts are large, remain patent for a long time, reduce the incidence of rebleeding, but reduce hepatoportal flow as well and have a higher rate of encephalopathy.3-5 These are associated with an accelerated rate of progression of the underlying liver disease, presumably because of reduced portal flow. The risk of encephalopathy ranges from 11%-75%, with the incidence of severe encephalopathy being 3%-21% (Table).

Table: Effect of types of shunts on various parameters

<table>
<thead>
<tr>
<th>Type of shunts</th>
<th>Sinusoidal pressure</th>
<th>Portal pressure</th>
<th>Hepatic flow via portal vein</th>
<th>HE</th>
<th>Severe HE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total shunts (&gt;10 mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>End-to-side</td>
<td>No effect</td>
<td>Decreased</td>
<td>Decreased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Side-to-side</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meso-caval</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central splenorenal</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
<td></td>
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<tr>
<td>PIPS</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partial shunts (&lt;8 mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Selective shunts</td>
<td>No effect</td>
<td>Decreased*</td>
<td>Maintained</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disto splenorenal shunt</td>
<td>No effect</td>
<td>Maintained</td>
<td>Maintained</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary caval shunt</td>
<td>No effect</td>
<td>Maintained</td>
<td>Maintained</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIPS</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Maintained</td>
<td></td>
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</tr>
</tbody>
</table>

*Amount of decrease in portal pressure is dependent on size of shunt. **Incidence of encephalopathy is dependent on size of shunt and may reach 50% as shunt diameter increases. ***Exact incidence not reported. HE, hepatic encephalopathy; PIPS, percutaneous inferior vena cava-to-portal vein shunt

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In three randomized trials in which portocaval shunts were compared with medical management, the shunts did not reduce mortality. In fact, an increase in mortality was noted. It is because of these reasons that these shunts are limited to only life-threatening situations and have been largely replaced by TIPS. These shunts make later liver transplantation difficult.

Partial portosystemic shunts

Partial portosystemic shunts are based on the concept of limiting the size of portocaval anastomosis to allow sufficient decompression of the portal system to control variceal bleeding yet maintain enough pressure to keep portal blood flow to the liver. The problem with these shunts is that they either thrombose or enlarge to become total shunts and are then associated with a higher rate of rebleeding or a reduced portal flow to the liver, respectively. It has been shown that if these shunts are more than 10 mm in diameter, they behave like total shunts and have similar rates of encephalopathy and other complications (Table). However, data from uncontrolled trials and a prospective controlled trial have shown good bleeding control and lower rates of encephalopathy in comparison to total shunts.

Selective shunts

Selective shunts have been designed to reduce the pressures in gastroesophageal varices alone and preserve the hepatic portal blood flow in order to reduce the incidence of hepatic encephalopathy. The two shunts that are used often are the distal splenorenal shunt and the coronary-caval shunt. These shunts are as effective as total shunts in decompressing gastroesophageal varices; they maintain hepatopetal flow in most cases and have an incidence of encephalopathy of 4%-9% (Table). Whether selective shunts have a lower incidence of encephalopathy as compared to nonselective shunts is however controversial. Most data have shown mixed results, with only half of the studies showing a lower risk of encephalopathy after selective shunts.

The most persuasive evidence for a lower risk of encephalopathy comes from a meta-analysis of four prospective randomized trials involving selective shunts and endoscopic sclerotherapy. Selective shunts are more effective in patients with nonalcoholic liver disease as compared to patients with alcoholic liver disease. In addition, patients with normal hepatocellular function, such as those with schistosomiasis or extrahepatic portal vein thrombosis, are excellent candidates for selective shunts. Despite the lower incidence of encephalopathy with selective shunts, many surgeons feel that this is outweighed by the technical difficulty of the operation. Also, variceal rebleeding rate is more common with selective shunts. It is also not suitable in emergency situations and in decompensated cirrhosis.

Still, it is the preferred surgery in patients who are potential candidates for liver transplantation.

Decompressive portocaval shunts have been used in patients with presinusoidal portal hypertension, like noncirrhotic portal hypertension (NCPH) and schistosomiasis and extrahepatic portal venous obstruction (EHPVO). These achieve good control of bleeding along with a significant reduction of rebleeding rate. The incidence of encephalopathy does not show any increase in patients with schistosomiasis after shunt surgery. However, in patients with NCPH who had been operated on with a proximal lienorenal shunt, tests revealed an abnormal EEG in 38% and abnormal psychometric profile in 30%. The incidence of clinical hepatic encephalopathy (HE) is however not clear. Patients with both presinusoidal hypertension and EHPVO are excellent candidates for surgical shunts and have a lower incidence of significant clinical encephalopathy.

Transjugular intrahepatic portosystemic shunt

TIPS was introduced in the 1980s for the control of refractory variceal bleeding and has been the mainstay for this indication where other methods have failed. It has also been shown to give good results in the control of acute and chronic portal hypertension, refractory ascites, in decompression of the portal system before liver transplantation, and in the management of hepatorenal syndrome. Promising indications for TIPS are the Budd-Chiari syndrome uncontrolled by medical therapy, severe portal hypertensive gastropathy, and refractory hepatic hydrothorax. In most centers where facilities are available, TIPS has replaced shunt surgery and also acts as a bridge in patients awaiting liver transplantation.

However, TIPS is also associated with HE, which can vary from subclinical encephalopathy causing only sleep disturbances to frank loss of consciousness. The incidence of encephalopathy varies from 3%-9% (Table) but most often is equal to that of patients with selective shunts or having smaller diameter H-graft portocaval shunt, i.e., 10%-25%. Encephalopathy is most often seen after the first month.

An increased risk of encephalopathy has been seen in elderly patients, in females, in patients having nonalcoholic liver disease, patients with hypoalbuminemia or renal dysfunction, patients with wider shunts, history of encephalopathy before TIPS, and in patients in whom portal pressures are reduced by more than 9 mmHg during TIPS. Encephalopathy is more often seen in patients with Child C cirrhosis but other classes are also affected.

Pathogenesis

As in all other situations of HE, the pathogenesis of encephalopathy in patients with decompression is pos-
sibly multifactorial and has been discussed elsewhere in this issue. In patients with chronic liver disease there is alteration of the liver structure with fibrosis and increased resistance to portal venous influx. These changes, coupled with a decompressive shunt / TIPS and collaterals, further reduce hepatic blood flow and hepatocyte mass and/or function. Hyperammonemia, which is the most important factor for causation of encephalopathy, has been documented in patients.

**Treatment**

Most patients with cirrhosis with HE following decompressive surgery respond to drugs and dietary restrictions. A combination of antibiotics, lactulose and protein restriction is able to treat most cases of encephalopathy following decompressive surgery. These treatment modalities, however, have to be continued on a long-term basis.

Correction of factors that precipitate an episode of encephalopathy should be done. The severity of underlying liver disease is an important determinant of response to therapy.

There is a role for preventive therapy with lactulose in patients undergoing TIPS, but data about its role in prophylaxis in patients undergoing surgical decompressive shunts are not available. Only 10% of patients with chronic liver disease have history of encephalopathy on lactulose undergoing TIPS had worsening of encephalopathy. In patients who do not respond to the above treatment modalities, other methods like shunt closure or reduction in the lumen of the shunt, loop ileostomy, and colonic exclusion have been tried. These methods cannot be routinely recommended. Recently, L-ornithine L-asparatate has been shown to give encouraging results but more studies are needed. L-carnitine has been shown to be effective in rats but human studies are not available. Hepatocyte transplantation has been shown to be helpful in some cases but further studies are needed.

In post TIPS encephalopathy, in addition to lactulose, antibiotics and protein restriction, a reduction in the size of the TIPS stent size with the help of latex balloons or placement of constrained/modified Wallgraft endoprosthesis has been shown to treat encephalopathy. There is little information about the treatment of patients with NCPF or EHPVO who develop encephalopathy following a shunt or spontaneously. Lactulose, antibiotics and protein restriction have been shown to be effective in certain cases. There are isolated reports of patients with EHPVO with spontaneous shunts responding to closure of these shunts.

**Conclusion**

Encephalopathy is a common complication of portal decompressive procedures but severe encephalopathy is a relatively rare occurrence. Selective shunts are associated with a lower risk of encephalopathy that is equal to that of TIPS. Patients with good liver function (preprocidinal and extrahepatic portal venous obstruction) have a good outcome with decompressive shunts, with a relatively lower risk of encephalopathy.

**References**


