RECOVERY FROM HEPATORENAL SYNDROME AFTER SUCCESSFUL ORTHOTOPIC LIVER TRANSPLANTATION

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INASMUCH AS THE KIDNEY failure of the hepatorenal syndrome (1) is believed to be secondary to hepatic dysfunction, replacement of the diseased liver should improve renal function. This objective was realized in three patients with the hepatorenal syndrome treated by orthotopic liver transplantation.

CASE MATERIAL

The patients, who were 34, 42, and 44 yr old, suffered from cirrhosis. They had massive ascites and edema and two of them were in stage III or IV coma. All had had normal renal function documented within a few weeks of transplantation, but progressive renal failure had then supervened with azotemia and oliguria. Two patients had a preoperative urine sodium concentration of less than 1 mEq/liter, while in case 3 it was 40 mEq/liter. The degree of combined renal and hepatic failure can be seen in Table 1.

RESULTS

Hepatic function in all three patients steadily improved after liver replacement (Table 1), but the course of recovery of kidney function varied. In cases 1 and 3 the characteristic urine findings, including

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<table>
<thead>
<tr>
<th>Case number</th>
<th>URINE VOLUME (ml/day)</th>
<th>URINE Na (mEq/L)</th>
<th>CR (ml/min)</th>
<th>TOTAL BILIRUBIN (mg/100 ml)</th>
<th>PROTHROMBIN TIME (%)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
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Liver transplantation.

Table 1—Renal and Hepatic Function in Three Patients With Hepatorenal Syndrome and Orthotopic Liver Transplantation

<table>
<thead>
<tr>
<th>Case number</th>
<th>Preoperative</th>
<th>1 day postoperative</th>
<th>4 days postoperative</th>
<th>10-14 days postoperative</th>
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</thead>
<tbody>
<tr>
<td>Cr (mg/dL)</td>
<td>2.345</td>
<td>8.16</td>
<td>3.32</td>
<td>5.8</td>
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<tr>
<td>BUN (mg/dL)</td>
<td>2.390</td>
<td>3.24</td>
<td>2.37</td>
<td>2.47</td>
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<tr>
<td>Creatinine</td>
<td>1.690</td>
<td>1.960</td>
<td>2.271</td>
<td>3.6</td>
</tr>
</tbody>
</table>

* Cr, creatinine clearance.
oliguria, high specific gravity, and low sodium content persisted for several days. However, in case 2 a massive diuresis and natriuresis were immediately established. Within ten days all three patients had regained adequate although subnormal renal function (Table 1).

Subsequently, one patient (case 2), who died of extensive bronchopneumonia on the 42nd day, had mild terminal deterioration of hepatic and renal function. Another (case 3) died on the 124th day from severe hepatic failure, caused by rejection, ten days after a second liver transplantation; mild renal dysfunction developed terminally. At autopsy, these two patients had essentially normal kidneys. The third patient (case 1) is alive after nine months but with abnormal liver function, probably due to chronic rejection; renal function remains normal.

Berkowitz et al (2) have suggested that in hepatic failure, renin substrate, synthesized by the liver, is deficient, thereby causing renal blood flow aberrations with secondary kidney failure. This hypothesis was tested in case 3. Before transplantation the plasma renin activity was high, 11.9 ng angiotensin I/ml/hr (normal 0.2-3.6), and renin substrate was low, 110.7 ng angiotensin I/ml (normal > 800). Both became normal (renin 1.5 and substrate 808.2) soon after operation. However, renal improvement was delayed for several more days after these corrections.

CONCLUSION

The hepatorenal syndrome is completely reversible by liver transplantation, but the recovery of renal function may be slow. Improvement of liver function probably corrects an abnormality of renal blood flow, but the precise mechanism remains unknown.

REFERENCES


CHAPTER X—Gastric

ELEVATED SERUM GASTRIC CASUALTIES WITH CNS IN

JOHN C. BOWEN, MD, W AND JAMES C. THOMPSON

GASTRIC HYPERACIDITY after CNS injury (1,2) and the release of gastrin may be released after CNS pathogenesis of CNS-related stress ulcer. Serum gastrin levels determine serum gastrin levels and, in addition, to examine the stress ulcer.

SG was determined in 39 severe 30 yr, who were injured in Vietnam had non-CNS injuries. Patients with those not followed up until their return to the study.

Fasting serum samples were drawn, and later transported to be measured simultaneously for line SG values were obtained in 2 and determinations were repeated if bleeding occurred. Six patients (three with CNS injuries) had only a single St u: bleeding. The diagnosis of stress u: intestinal bleeding occurred requ

RES1

The results are summarized in Table 1 after CNS injury than after non-C group, SG values were consistently between SG and stress ulcer. Altho be a difference between those with CNS injury group, the findings w

From the Division of Surgery, Walter Army Institute of Pathology, Galveston. Assistance of Col. Robert T