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

Case number	URINE VOLUME (ml/day)	Ccr* (ml/min)	URINE Na (mEq/L)	TOTAL BILIRUBIN (mg/100 ml)	PROTHROMBIN TIME (%)
1	2	3	1	2	3
2	2	1	2	1	2
3	3	2	3	3	1

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RENAL SYNDROME
 TOPIC LIVER

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MATERIAL

One 44 yr old, suffered from cirrhosis.
 One and two of them were in stage III
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 anemia. Two patients had a preopera-
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RESULTS

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

Case number	URINE VOLUME (ml/day)	Ccr* (ml/min)	URINE Na (mEq/L)	TOTAL BILIRUBIN (mg/100 ml)	PROTHROMBIN TIME (%)
Preoperative	1	3	1	2	3
	2	3	1	2	3
	3	1	2	3	1
1 day postoperative	90	227	6	19.8	7.3
	4,140	781	9	6.1	4.0
	2,990	2,345	32	27	4.9
10-14 days postoperative	675	8	16	3.7	4.9
	1,960	55	77	111	3.0
	1,690	2,395	50	10	27

* Ccr, 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.

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CHAPTER X—Gastric

ELEVATED SERUM GASTRIC ACIDITY IN CASUALTIES WITH CNS INJURY

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GASTRIC HYPERACIDITY after CNS injury (1,2) and the release of gastrin (3) after CNS injury may be released after CNS injury. The pathogenesis of CNS-related stress ulcers is not determined. We determine serum gastrin levels (4) after CNS injury and, in addition, to examine the relationship between stress ulcer and gastrin.

ME

SG was determined in 39 severely injured patients, 30 yr, who were injured in Vietnam. Six patients had non-CNS injuries. Patients with CNS injuries and those not followed up until their death were included in the study.

Fasting serum samples were drawn before surgery, frozen, and later transported to the laboratory. SG values were measured simultaneously for all patients. SG values were obtained in 39 patients and determinations were repeated in 10 patients. Six patients (three with CNS injuries) had only a single SG value. The diagnosis of stress ulcer and gastrointestinal bleeding occurred require

RESI

The results are summarized in Table 1. The SG values after CNS injury were consistently higher than after non-CNS injury. SG values were consistently higher between SG and stress ulcer. Although there is a difference between those with CNS injury group, the findings were

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