SHORT REPORTS

Ascites and Renal Failure in Primary Liver Cell Carcinoma

Ascites appears in 50-75% of patients with primary liver cell carcinoma (P.L.C.C.)¹ but there have been few investigations of its cause. We studied 25 patients with P.L.C.C. and ascites to assess any renal disturbances present.

Patients, Methods, and Results

Diagnosis was based on histological data, and all except one patient had P.L.C.C. associated with cirrhosis. None received specific treatment for the P.L.C.C. and all died. Liver function was evaluated and renal function studied by determining the creatinine clearance as an index of the glomerular filtration rate (G.F.R.), the sodium, potassium, and urea concentrations in plasma and urine, and plasma and urine osmolality. Ascites was treated by bed rest and a low-salt diet for seven days after admission. Patients who did not lose ascites and body weight were started on distal diuretics (triamterene 300 mg/day or spironolactone 150 mg/day), and if the response was unsatisfactory frusemide (80 mg/day) was added.

Renal failure was defined as a G.F.R. of below 50 ml/min, and we divided the patients into three groups: (1) 14 patients with progressive renal failure; (2) four patients admitted with good renal function but who developed renal failure while in hospital (their renal failure was identical with that in group 1, and in none could a cause be found); and (3) seven patients whose good renal function was maintained.

Necropsy of the 13 patients—10 with renal failure—for whom it was permitted showed normal kidney histology. Liver function tests showed no appreciable differences between patients with renal failure and those without (table). In group 2 the development of renal failure was not accompanied by a worsening liver function.

Only one patient in group 1 had a positive response to diuretic treatment, whereas all of group 2 had a good response initially, but the development of renal failure was followed by a fall of diuresis and natriuresis. Response was excellent in group 3, ascites being lost in all cases. All the patients with renal failure died in hospital (mean survival time 14.5 days), whereas those without renal failure were discharged without ascites and survived for a mean of 173 days ($P < 0.001$).

Discussion

Renal failure is a recognized complication of liver disease.² In 1965, Vesin *et al.*³ described two patients with cirrhosis of the liver, ascites, and P.L.C.C. with functional renal failure. In our series the incidence of renal failure in P.L.C.C. was 72%, and like cirrhosis and fulminant hepatic failure it carried a poor prognosis. All our patients with renal failure died within 45 days of admission, whereas those without renal failure had a mean survival of six months.

Histologically the kidneys of 10 patients who died with renal failure showed no abnormalities. In all patients with renal failure the urinary sodium concentration was low and the urinary urea concentration and urine:plasma osmolality ratio were relatively high. This suggests that renal tubular function was preserved and that renal failure was functional.⁴ Twenty-four of the patients had cirrhosis, so it could be argued that renal failure was related exclusively to cirrhosis. But the incidence of renal failure was much higher than that reported by Bosch (17.7%),⁵ suggesting that P.L.C.C. was an important factor.

Ascites in patients with cirrhosis and P.L.C.C. is classically considered to be refractory to diuretics. Nearly a third of our patients had a diuretic response, apparently related exclusively to the degree of

renal impairment. The seven patients with good renal function lost ascites, two of them with such simple treatment as bed rest and a low-salt diet.

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¹ MacDonald, R. A., *Archives of Internal Medicine*, 1957, **99**, 266.

² Summerskill, W. H. J., *Gastroenterology*, 1966, **51**, 94.

³ Vesin, P., Roberti, A., and Viguié, R., *Semaine des Hôpitaux de Paris*, 1965, **41**, 1216.

⁴ Vesin, P., in *Aktuelle Probleme der Hepatologia*, ed. G. A. Martini, p. 98. Stuttgart, Thieme, 1962.

⁵ Bosch, J., "Tipos de insuficiencia renal asociados a la cirrosis hepática," M. D. thesis, University of Barcelona, 1973.

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Does the Jet of Acid Emerging Through the Pylorus Determine the Site of Duodenal Bulbar Ulcers?

Peptic ulcers are usually attributed to either an increase in acid attack or a decrease in mucosal defence.¹ Duodenal ulcers are characteristically single, discrete, and situated in the middle of the anterior or posterior bulbar wall close to the pylorus.² No constant anatomical features have been identified that would determine the usual sites of ulcer. Animal experiments have indicated that the location of postpyloric peptic ulcers is determined by the jet of acid and gastric contents emerging through the pylorus.³ If this is so for humans the usual site of ulceration should lie in the path of the jet emerging through the pylorus. It should also be possible to show that duodenal ulcers or scars lie directly in the path of such a jet.

Methods and Results

The duodenal bulbar wall was inspected in 200 patients through the pylorus at routine upper gastrointestinal endoscopy. The tip of an end-viewing fiberoptic gastroduodenoscope was manoeuvred close to, and squarely facing, the pylorus. The duodenal wall could be seen through the pylorus. The instrument was then carefully withdrawn, avoiding rotation, while keeping the pylorus centred in the field-of-view to maintain the tip of the instrument as near as possible in the axis of the gastric lumen. Withdrawal was continued until the gastric angulus could be identified providing a point of reference.

Successful views of the duodenal bulb were obtained through the intact pylorus in 162 of the 200 examinations. A straight tunnel was seen in 16

Renal and Hepatic Function in the Three Groups of Patients. Figures are Means $\pm$ S.D.

Group	G.F.R. (ml/min)	Plasma		Urine			Urine: Plasma Osmolality Ratio	Serum Albumin (g/l)	Serum Bilirubin (mg/l)	Prothrombin Time (%)
		Sodium (mmol/l)	Urea (mmol/l)	Sodium (mmol/l)	Urea (mmol/l)	Volume, 24 h (ml)				
1	22.1 $\pm$ 13.4	133.8 $\pm$ 6.0	18.7 $\pm$ 10.5	6.6 $\pm$ 6.5	231 $\pm$ 116	476 $\pm$ 319	1.64 $\pm$ 0.46	30 $\pm$ 6	120 $\pm$ 12.1	65 $\pm$ 15
2 { On admission Before death	70.0 $\pm$ 6.2	133.7 $\pm$ 1.8	5.0 $\pm$ 1.5	38.3 $\pm$ 56.6	329 $\pm$ 227	941 $\pm$ 220	1.91 $\pm$ 0.67	32 $\pm$ 3	54 $\pm$ 57	90 $\pm$ 14
	15.0 $\pm$ 6.3	127.5 $\pm$ 8.1	24.9 $\pm$ 12.8	12.0 $\pm$ 2.7	254 $\pm$ 118	182 $\pm$ 42	1.79 $\pm$ 0.50	33 $\pm$ 4	65 $\pm$ 54	91 $\pm$ 17
3	85.6 $\pm$ 19.6	135.3 $\pm$ 5.5	4.3 $\pm$ 1.0	36.9 $\pm$ 34.8	338 $\pm$ 112	1320 $\pm$ 228	1.75 $\pm$ 0.43	30 $\pm$ 3	50 $\pm$ 47	84 $\pm$ 15

Conversion: SI to Traditional Units—Plasma sodium: 1 mmol/l $\approx$ 1 mEq/l. Plasma urea: 1 mmol/l $\approx$ 6 mg/100 ml. Urinary sodium: 1 mmol/l $\approx$ mEq/l. Urinary urea: 1 mmol/l $\approx$ 0.006 g/100 ml.