CONSULTANT: Dr Lee
ADMISSION DATE: 2.2.94
DISCHARGE DATE: 4.3.94

The Royal Free Hospital
Pond Street
London NW3 2QG
Telephone: 01-794 0500

G.P.: Dr.
ADDRESS:
Dr G P Tannen
25, Fairfax Road
LONDON NW6

UNIT NO: 115001
NAME: Michael MORROW
DATE OF BIRTH: GRO-A 29
ADDRESS: GRO-C

SUMMARY OF CASE

Principal diagnosis:

PRINCIPAL DIAGNOSIS/PROBLEMS — OPERATION(S) AND DATE(S)

Severe Factor IX deficiency

Secondary diagnoses:

Chronic liver failure
Hepatitis C
Hepatitis A

This 65 year old man was admitted from home with an exacerbation of his chronic liver disease. He has had a short In-patient stay from the 1st January until the 14th January 1994, complaining of indigestion. This had responded to prescription of Ranitidine. However, on the 28th January he started to notice increased swelling of the ankles and was complaining of lower abdominal pain. He was again reviewed on the 2nd February, but he had increased bilateral leg oedema, sacral oedema and ascites. He was, therefore, admitted for investigation and management of his ascites.

Examination:

On examination he had no evidence of encephalopathy, but did have gross ascites, sacral oedema and leg oedema. His pulse was 80 regular, blood pressure 130/70, JVP was not raised and heart sounds were normal. His respiratory system was normal.

On admission his sodium was 125, potassium 4.3, creatinine 108 and urea 8.1. Alkaline phosphatase was 99, ALT 42, AST 17 and bilirubin 27. His albumin was 30. Gamma GT was 65. A CT scan of the liver revelled no evidence of hepatocellular carcinoma and an alpha fetoprotein liver was normal.

Management:

He was put onto a fluid restriction of 1.5 litres daily and was treated with Frusemide and increasing doses of Spironolactone. He was reviewed by the hepatology team, who suggested a salt-free diet and to continue with diuresis. On admission his weight was 90 kg and on this regime his weight gradually improved over the next three weeks. With this his symptoms of abdominal pain improved and his oedema improved. His weight stabilised at 80 kg and he was discharged on the 4th March 1994. On the 1st March 1994 he was seen in the Oral Surgery Out-patients and had five teeth removed under factor IX cover. He was, therefore, put onto Tranexamic acid mouthwash following this procedure. Although the worst of the decayed teeth were removed, he still has severe dental problems and will require further review in this clinic.

DISCHARGE SUMMARY

PC/LRB/115001

8 March 1994

Michael MORROW - GRO-A 29

Drugs on discharge:

On discharge he was taking:

Frusemide	80 mg in the morning and 40 mg mid-day.
Spironolactone	500 mg daily
Amphotericin mouthwash	4 times a day
Mucogel	4 times a day
Lactulose	20 ml twice daily
Ranitidine	150 mg twice daily
Dexamphetamine	15 mg in the morning

He will be reviewed twice weekly by the Community Haemophilia Nurse and we are arranging for home help and meals on wheels.

On discharge his haemoglobin was 11.9 gm/dl, white count 11.1 x 10^9/l and platelets 66 x 10^9/l. His platelet count will have to be kept under close review, but it is probably secondary to the chronic liver disease. His urea was 9.3, potassium 4.8, sodium 125 and creatinine 145. Alkaline phosphatase 91, ALT 36, AST 57 and total bilirubin 37 and albumin 30. We will continue to keep him under review.

Yours sincerely,

GRO-C

Dr Peter Collins
Senior Registrar/Lecturer

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CONSULTANT: Dr Lee
ADMISSION DATE: 14.7.93
DISCHARGE DATE: 23.7.93

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UNIT NO: 115001
NAME: Michael MORROW
DATE OF BIRTH:
ADDRESS: GRO-C

SUMMARY OF CASE

PRINCIPAL DIAGNOSIS/PROBLEMS

OPERATION(S) AND DATE(S)

1. Haemophilia B
2. Hepatitis C infection
3. Chronic liver disease with ascites
4. Possible urinary tract infection

History

This patient with severe Haemophilia B was re-admitted because of a recurrence of abdominal distention and discomfort. He had been discharged from hospital on the 2nd July, and his symptoms had recurred a few days following discharge. He had been on a low salt diet and fluid restriction regime, to which he had been sticking, as well as he could. He had had some alcohol on one occasion, but had found this quite unpalatable and had subsequently abstained from alcohol.

He also gave a history of hesitancy of micturition, and urinary urge incontinence over the previous few days; these symptoms made it very difficult for him and his wife to cope at home.

Medications on admission

He was taking:

Dexamphetamine	15 mg mané
Spironolactone	200 mg mané
Mucogel	10 mls PRN
Thiamine	T per day
Zantac	300 mg nocte

He had also been taking Co-dydramol - 4 to 5 tablets daily and DF118 - 2 tablets daily for the abdominal discomfort.

DISCHARGE SUMMARY

GA/LRB/115001
27 July 1993

GRO-A GRO-A 29

Examination

On examination he was alert. He was characteristically slow of speech, but there were no signs of encephalopathy. Gynaecomastia and palmar erythema were noted. Cardiovascular system - prolonged, pulse 100 per minute and regular, blood pressure 150/96. Apex beat - fifth intercostal space mid clavicular. Heart sounds normal. No murmurs. JVP not elevated. No peripheral or sacral oedema. Examination of the chest was normal. Abdominal examination - the spleen tip was palpable. The abdomen was distended - girth 45 inches - with shifting dullness, indicating the presence of ascites. There was generalised moderate tenderness. There was no scrotal oedema. Bowel sounds were normal or slightly increased. Rectal examination - the prostate was not obviously enlarged, but it was difficult to examine. There were no faeces in the rectum.

Investigations

Haemoglobin 11 gm/dl, MCV 74 fl. White cell count 4.8 x 10^9/l, platelets 130 x 10^9/l.
Urea and electrolytes: urea 4.6 mmol/l, sodium 132 mmol/l, potassium 4 mmol/l.
Liver function tests - total bilirubin 22 umol/l, albumin 31 gm/l, alkaline phosphatase 135 u/l, AST 44 u/l, ALT 16 u/l.
Blood sugar 5.3 mmol/l.
An EEG was performed which showed some slight changes suggestive of mild encephalopathy.

Management

He was reviewed by the Registrar from the liver team, who had seen him during his previous admission, and felt that there had been a mild degree of decompensation with the re-accumulation of a moderate amount of ascites, perhaps due to less than strict adherence to the food and dietary recommendations. We therefore continued the low salt diet, fluid restriction and diuretics and in addition started treatment with oral Trimethoprim for a possible urinary tract infection. The MSU showed greater than 200 cells/cm^3, with a mixed growth; the sample may have been taken after the first dose of Trimethoprim. He was given Temgesic sublingually, intermittent for the abdominal discomfort. The lactulose was increased in an attempt to increase the frequency of bowel motions. His symptoms settled very quickly and there was some weight reduction, indicative of a diuresis, although the abdominal girth remained unchanged. The Spironolactone was increased to 300 mg mané on the basis of urinary electrolyte measurements, which showed a potassium greater than sodium (69 and 67 respectively). There was some improvement in the liver function tests.