The Lothian University
Hospitals NHS Trust

NHS
Lothian

Royal Infirmary of Edinburgh
Lauriston Place, Edinburgh, EH3 9YW. Telephone: 0131 536 1000

26/9/02

To: DR RR JAMIESON
BRIDGETON HEALTH CENTRE
201 ABERCROMBY STREET
GLASGOW

G40 2DA

Admission date: 08/08/2002
Discharge date: 16/08/2002
Consultant: Dr Alastair MacGilchrist
Ward: SLTU
Our reference: NK/JF

Name: **VICTOR TAMBURRINI**

Number: 2704571796
CHI no:
D.O.B.: 27/04/1957

Address: 284E LONDON ROAD
GLASGOW
G40 1PT

LIVER /
ICD LSA 1200990000

No Complications

26 August 2002

DIAGNOSIS:

1. Hepatitis C and alcohol induced liver cirrhosis - now off alcohol for 6 months
2. Progressively rising AFP - no evidence of tumour on repeared imaging and liver biopsy; now AFP falling

Mr Tamburrini was admitted to the Liver Transplant Unit in view of his progressively rising AFP. As you know he recently had triple face CT scan of his liver on the 21st June which did not reveal any focal abnormality in his liver. Since his AFP had risen we were concerned about this and brought him for liver biopsy. His bloods on admission revealed haemoglobin 138, white cell count 6.4, platelets 84, PT 18, bilirubin 97, ALT 112, ALP 249, GGT 71, albumin 25, sodium 134 and potassium 4.1. An ultrasound scan of the abdomen revealed a generalised increase in echogenicity of the liver, spleen of 10cm, normal flows in portal vein, hepatic artery, hepatic veins and inferior vena cava. He underwent a liver biopsy. The procedure was uncomplicated and he was discharged home the following day.

The liver biopsy revealed cirrhosis with mild ongoing inflammation due to hepatitis C. There was no evidence of dysplasia or tumour around the biopsy specimen. In view of his poor synthetic function of liver with an albumin of 25, PT 13 and a bilirubin of 97, it was decided that Mr Tamburrini should be brought in again to do a formal assessment regarding liver transplantation and discuss the results of the biopsy.

Mr Tamburrini was re-admitted to the Liver Transplant Unit on the 13th August 2002. He underwent the formal assessment process when he was here. I will briefly go through his past medical history.

Mr Tamburrini was well until about 1998 when he was found to have gynaecomastia and a history of alcohol excess was noted in 1999. He underwent bilateral mastectomies for gynaecomastia. In 2000 he was admitted with abdominal pain and had a mildly raised amylase. He was thought to have alcohol induced pancreatitis and was advised to stop alcohol at that time. In June 2001 he was noted to have abnormal liver function tests when he was investigated for the swelling of his ankles and consequently was referred to Glasgow Royal Infirmary. He was found to be hepatitis C positive and the only source of infection was a plasma transfusion which he had when he sustained burns on his hands in the 1980's. He was advised to stop alcohol in view of his abnormal liver function tests. In November 2001 he was seen by Dr Stanley at Glasgow Royal Infirmary and was noticed to have a raised AFP. He was referred to the Edinburgh Royal Infirmary after repeated imaging, namely two ultrasounds, one contrast enhanced CT scans and one MRI scan which did not reveal the presence of any tumour as a cause of his rising AFP.

In February 2002 Mr Tamburrini underwent the formal transplant assessment process. At that time his AFP was 359. During the week he underwent contrast enhanced CT scan with Lipiodol injection. The scan did not reveal the presence of any tumour. He underwent laparoscopic ultrasound which revealed a cirrhotic tumour and liver biopsy done at the time of laparoscopy revealed cirrhosis with mildly active viral hepatitis C. In view of the history of alcohol excess in the past he was reviewed by the psychiatrist who felt that he had started to understand the importance of alcohol and his liver disease and was willing to be abstinent from alcohol and was willing to be reviewed regularly by the alcohol liaison services.

Through March 2002 until July 2002 his AFP progressively increased to 732 in July this year. A triple face CT scan was performed at the end of June 2002 which was negative for tumour. The liver biopsy done the week prior to the assessment week did not reveal any evidence of tumour either.

As far as his symptoms due to liver disease are concerned, Mr Tamburrini does not have major symptoms. He only complained of increasing swelling of ankles and feeling tired and lethargic all the time for a week prior to being admitted to the Liver Unit. He already felt that with the introduction of diuretics, he had started to feel better. There was no history to suggest haematemesis or melaena. No history of encephalopathy or history of any other problems due to liver disease. There is no other significant past medical history. In view of the previous history of alcohol excess he was reviewed by Dr Masterton while he was an in-patient. Dr Masterton felt that he had managed to stay abstinent and did not feel that alcohol was an issue now. He did not feel that Mr Tamburrini required any further psychiatric input from that point of view.

Mr Tamburrini underwent multiple investigations as part of the transplant assessment process. His haemoglobin was 126, white cell count 7.2, platelets 75, MCV 104, PT 18, sodium 134, potassium 3.8, bicarbonate 22, urea 4.5, creatinine 96, bilirubin 70, ALT 84, ALP 193, GGT 56 and his AFP level had come down to 136. His albumin was 21. Normal blood gases. His creatinine clearance was 113 mls/min. Normal chest xray. Ultrasound of the abdomen showed patent portal veins and hepatic veins. He did not have any focal lesions in his liver. His ECG was normal and his lung function tests were normal. He was found to be CMV IgG antibody positive, hepatitis C virus positive and negative for HIV and hepatitis B surface antigen. His hepatitis C PCR revealed copies more than 150 per ml. He was found to be varicella zoster virus IgG antibody positive and toxoplasma

antibody negative. He has been known to be negative for autoantibodies. On upper GI endoscopy he was found to have 4 grade I varices and portal hypertensive gastropathy which did not require banding. His dentition was found to be good.

While he was an in-patient Mr Tamburrini was informed about the transplant assessment process. After careful consideration, Mr Tamburrini was to delay the liver transplant as long as possible in view of the fact that he is entirely asymptomatic from the liver disease point of view. His history, investigations and his views about transplantation were discussed at the transplant assessment meeting. It was felt that despite the poor synthetic function of his liver, that the process of transplantation should be deferred as Mr Tamburrini did not want it at this time. The decision was made to defer listing him for the time being and to review him regularly in the out-patient clinic. He will be reviewed in 6 weeks time.

Medications on discharge:

Spironolactone 100mg bd
Quinine Sulphate 300mg once daily

Discharged to Home.

Narendra Kochar
Specialist Registrar (LAT) to Dr Alastair MacGilchrist

Copy to: Dr A Stanley
Consultant Physician
Royal Infirmary
Glasgow