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Discussion: defining and diagnosing hepatorenal syndrome

Asked by Professor Frederik Nevens (UZ-Gasthuisberg-KUL, Leuven, Belgium) if he advocated kidney biopsies in cirrhotics Professor Lebrec confirmed that his unit undertake transjugular renal biopsy before liver transplantation in patients who have renal failure without obvious precipitating causes. In his view, the transjugular approach is quite safe and may reveal abnormalities in the kidneys.

Professor Andrew Burroughs (Royal Free Hospital, London, UK) added that in his unit they perform biopsies transfemorally. Interestingly, renal biopsy may reveal more structural abnormalities than might be expected, indicating that cirrhotics who develop renal problems may actually have some intrinsic renal disease.

Professor Faouzi Saliba (Hôpital Paul Brousse, Villejuif, France) stated that his unit has experience of renal biopsy prior to liver transplantation in patients who are not yet at an end-stage of their cirrhosis. In patients with Child's C cirrhosis with ascites the renal biopsy can be performed at the same time as transjugular liver biopsy and provides a great deal of information, mainly on prognostic issues relating to the kidneys. This might indicate whether a double kidney and liver transplant is required or whether liver transplantation alone will suffice. In patients with cirrhosis, with normal creatinine and normal ultrasound, Professor Saliba does not perform renal biopsy. However, if there are some abnormalities, either of renal function or kidney size, he performs a biopsy. Professor Burroughs stressed that the message is that renal biopsies can be performed safely by the transvenous route and can help in the management of patients.

Asked whether the work-up of the patient with renal impairment and chronic liver disease should always include blood cultures Professor Lebrec agreed that in severe liver disease it should. Professor Burroughs speculated whether patients should be treated with antibiotics anyway, without waiting for the results of blood cultures, and asked the audience how many would treat with antibiotics empirically, faced with renal impairment in cirrhosis (i.e. in a patient who is admitted to hospital feeling unwell, with oliguria, and raised creatinine). Around half the delegates indicated that they would give antibiotics, while the remaining 50% would wait for the blood cultures to establish the situation and treat the next day if necessary. Professor Burroughs stated that his own approach is pragmatic: treat what is treatable in terms of precipitating causes and then 'worry' later.

Dr Veil Gülberg (Medizinische Klinik II, Munich, Germany) stated that it is well known that terlipressin (Glypressin®) is effective in hepatorenal syndrom (HRS) but asked about the diagnostic value of terlipressin testing for establishing the diagnosis of HRS. While agreeing that this is a good idea, Professor Lebrec pointed out that patients with acute tubular necrosis will not respond. Professor Burroughs agreed, his own impression being that terlipressin is a good drug to raise blood pressure empirically (obviously combined with fluid replacement) and speculated whether the crucial point is to manage renal impairment in cirrhosis rather than attempting to make a diagnosis of HRS.

Professor Gin-Ho Lo (Kaohsiung Veteran General Hospital, Taiwan) pointed out that diabetes mellitus is very common in cirrhotic patients and asked whether diabetic nephropathy can be differentiated from type 2 HRS. Professor Burroughs agreed that he sees a lot of NASH (nonalcoholic steatohepatitis) in diabetics. Professor Pere Ginès (Hospital Clínic, Barcelona, Spain) commented that his unit is undertaking a prospective study of different causes of renal failure in patients which cirrhosis. In this study he is using renal biopsy to diagnose the cause of renal failure in those patients in whom there is some data suggestive of parenchymal renal failure (such as haematuria or proteinuria).

Professor Saliba pointed out that Professor Lebrec did not mention that in HRS one of the main issues is patients who have jaundice or cholestatic disease, with high bilirubin levels (e.g. 200 or 300 μmol/L). These patients are difficult to treat. Professor Lebrec stated that he would not use the term HRS in cases such as this, but

rather 'acute renal failure in patients with cirrhosis', or perhaps 'prerenal failure in patients with cirrhosis'.

Professor Burroughs asked the audience what proportion of cirrhotics with renal failure (rising creatinine and oliguria) they diagnose as solely having HRS. The majority of the audience indicated that they would diagnose 25–50% of such patients as having solely HRS. Professor Burroughs then asked what are the principal criteria delegates use to diagnose HRS. Asked to choose between urine volume response to intravascular filling, urine volume response to vasoconstrictive therapy, and absence of precipitating factors causing acute tubular necrosis, no delegates would rely solely on one of these criteria alone, the majority using at least two of these criteria, and some requiring them all to be present before diagnosing HRS. Professor Burroughs believed that this response highlighted the problems that sometimes occur in separating acute tubular necrosis from HRS.

He went on to question how effective urine testing is in clinical practice and whether testing the urine once only is sufficient, or if the urine should be tested again, for example 6 h after intravascular filling, or the next day. Professor Ginès stated that several studies show that, at least in patients with cirrhosis, urine sodium may be high but the patient may still have HRS. However there are no data on whether repeating urine tests is better than doing just one.

Professor Burroughs raised the issue of using ultrasonography or other methods to assess kidney size in cirrhotic patients with worsening oliguria – assessing kidney size is a good indicator of whether the patient has intrinsic renal disease and also excludes obstruction. Interestingly, however, obstruction is an extremely rare diagnosis, perhaps because of the age frame of the patients.

Asked about the commonest precipitating cause in cirrhotics developing renal failure in their unit, the majority of the audience pointed to sepsis, with a few indicating nephrotoxic drugs. The audience generally felt that X-ray contrast media are not a precipitating cause.