



Case Report

# From Normal Renal Function to Renal Replacement Therapy after Liver Transplantation: A Case Report

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**Abstract:** Postoperative renal failure significantly impacts long-term renal function and the overall survival of patients receiving liver transplantation (LT), being a crucial factor in their morbidity and mortality. It is difficult to define whether the causes of renal failure are solely related to surgery or anaesthesia during liver transplantation (LT). Indeed, liver disease requiring liver transplantation is often the cause of preoperative renal failure. We report a case of a 62-year-old patient undergoing LT for cholangiocarcinoma that led to acute kidney injury postoperatively while his preoperative renal function was normal. This report highlights the major influence that the surgical and anaesthetic procedure can have on renal function and identifies the factors that may have led to renal replacement therapy being required for this patient.

**Keywords:** liver transplantation; acute kidney injury; veno-venous bypass; piggyback technique; anaesthesia



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## 1. Introduction

Acute kidney injury (AKI) after liver transplantation (LT) is a common problem and sometimes a complex perioperative challenge. The prevalence of AKI after orthotopic liver transplantation varies from 12% to 70%, depending on the definition [1]. We present here the case of a patient with post-LT end-stage renal failure that occurred in the absence of any recognisable preoperative risk factors, without major intraoperative complications or postoperative graft dysfunction. Several risk factors have been identified for post-LT AKI such as the Model for End-Stage Liver Disease (MELD) score, baseline renal function, hemodynamic status, red blood cell requirements, surgical technique, the use of nephrotoxic drugs, graft dysfunction, and sepsis [2]. This article sheds light on the intra- and postoperative factors that may have led to end-stage AKI and explains why the prevalence of renal failure tends to be so high after this particular operation. Finally, we discuss the decisions that could have improved renal prognosis.

The CARE Checklist has been completed by the authors for this case report, and is attached as Supplementary Materials.

## 2. Case Report

We present the case of a 62-year-old patient with end-stage renal failure following liver transplantation. The indication for liver transplantation was cholangiocarcinoma related to sclerosing cholangitis. In 2018, the patient presented two episodes of sepsis requiring antibiotic treatment. At that time, an ERCP was performed, which revealed a diagnosis of sclerosing cholangitis. At the beginning of 2020, the patient presented an increase in transaminases, and ultrasound showed a hyperechogenic parenchymal nodule in the right liver. Radiological investigation by means of magnetic resonance imaging (MRI) showed a periductal tumoral lesion originating in segment IV of the liver,

extending to the biliary convergence, with evidence of deep invasion. A diagnosis of right Bismuth III cholangiocarcinoma obstruction was retained. One month after the diagnosis of cholangiocarcinoma, an exploratory laparoscopy to exclude carcinomatosis was performed, followed by a laparotomy to identify lymph nodes as well as a liver biopsy of segment IV. At that time, hepatectomy was not performed due to inflammation, signs of cholestasis, and macroscopic liver appearance, suggesting the presence of relatively advanced sclerosing cholangitis. Due to the advanced pathology and the anatomopathological results of invasive cholangiocarcinoma, a neoadjuvant radio-chemotherapy treatment was prescribed with Gemcitabine intravenous (iv) 2500 mg/m<sup>2</sup>/j, which was stopped after 14 days due to ileitis. A pre-assessment was carried out during hospitalization in order to discuss LT.

Apart from the liver disease, the patient was otherwise in good health, and there were no associated comorbidities. Hematologic and biological analysis revealed no abnormalities, creatinine level was around 80 µmol/L, and eGFR was 92 mL/min/1.73 m<sup>2</sup>. The patient was not taking any chronic medication, was not a smoker, had a negligible alcohol consumption, and maintained a high level of daily physical activity.

Four months later, in August 2020, the patient underwent liver transplantation from a donation after circulatory death (DCD). Warm ischemia time was 22 min and cold ischemia time was 9 h 29 min. The donor was a 45-year-old Caucasian male known for smoking and epilepsy, who died after a severe traumatic brain injury.

On the first postoperative day, the patient developed oliguric AKI KDIGO 3 with renal tubular epithelial cells in the urine. Over the next three days, the patient presented signs of fluid overload requiring the introduction of renal replacement therapy with a volume depletion regime of 2.5 L per day, which was well tolerated hemodynamically. The patient was discharged from the intensive care unit on the 7th postoperative day, and four continuous veno-venous hemodiafiltration (CVVHDF) sessions were required in the following two weeks. The patient was discharged from the hospital 43 days after LT, with a creatinine level at 126 µmol/L, eGFR 52 mL/min/1.73 m<sup>2</sup>. The patient did not receive any diuretic drugs during the ICU stay. No septic episodes were reported in the postoperative period.

Preoperative physical examination was unremarkable. The patient's weight was 78 kg for 183 cm. Pre-LT assessments including cardiac, pulmonary, and renal investigations were normal and did not show any cardiac, pulmonary vascular abnormalities or portal hypertension. Transthoracic echocardiography showed a left ventricular ejection fraction of 62%, discrete mitral regurgitation, and no segmental kinetic disorder.

Preoperative liver tests showed close to normal liver function with minor hepatic cytolysis (ASAT 87 U/L, ALAT 45 U/L), slight cholestasis (alkaline phosphatase 437 U/L, gamma glutamyl transferase 720 U/L), and a total bilirubin of 27 µmol/L. Renal function was normal, with serum creatinine at 78 µmol/L and eGFR at 92 mL/min/1.73 m<sup>2</sup>. Apart from insignificant anaemia, with a haemoglobin level of 123 G/L, the remaining laboratory tests were normal.

The anaesthesia equipment included a central venous line, a pulmonary artery Swan-Ganz catheter, a radial artery catheter, two peripheral venous lines, a nasogastric tube, a urinary catheter, a 7.5 size endotracheal tube, and two temperature probes.

At anaesthesia induction, the patient received intravenously Propofol 200 mg, Sufentanil 15 mcg, Suxamethonium 100 mg, and Lidocaine 30 mg. Endotracheal intubation was performed without any complications. An atracurium infusion was used for neuromuscular blockade, and anaesthesia was maintained with inhaled sevoflurane. During the pre-anhepatic phase, the patient remained hemodynamically stable. There were no particular surgical or anaesthetic events. Blood loss was minor and controlled. Cardiac output before the inferior vein cava (IVC) cross-clamping was 7 L/min. After IVC cross-clamping, a noradrenaline (0.14 mcg/kg/min) infusion was started to maintain mean arterial pressure at 65 mmHg during the whole anhepatic phase (79 min). Cardiac output after IVC cross-clamping was stable at 4.6 L/min. According to the institution's protocol, a shared decision was made by the surgical and anaesthesia teams not to use veno-venous bypass

(VVB) during the anhepatic phase. During the post-anhepatic phase (4 h 10 min after the start of surgery), relative hypotension (MAP 60–65 mmHg) persisted under noradrenaline 0.14 mcg/kg/min, and cardiac output averaged 8 L/min. Total blood loss was estimated to be 500 mL, no blood products were transfused, and the patient received 4900 mL iv crystalloids (575 mL/h). More aggressive filling could have compromised the anastomotic zones, and therefore small doses of noradrenaline were administered to maintain MAP between 60 and 65 during the procedure, as recommended in the literature [3]. Urine output during the surgery was 1 mL/kg/h, and 0.5 mL/kg/h during the first 24 h in the postoperative phase.

At the end of the surgery (512 min total duration), the patient was admitted to the intensive care unit (ICU), under sedation and mechanical ventilation, and remained hemodynamically stable over the first 24 h (continuous iv noradrenaline doses not exceeding 0.1 mcg/kg/min). Noradrenaline support could be weaned on the first day after surgery. The patient did not present any significant blood loss or transient hypotension during the postoperative period. No septic episode was identified.

On admission to the ICU, serum creatinine level was at 74  $\mu\text{mol/L}$ . On postoperative day 1, the serum creatinine was 183  $\mu\text{mol/L}$  and eGFR was 33 mL/min/1.73 m<sup>2</sup>, which increased to 450  $\mu\text{mol/L}$  and 11 mL/min/1.73 m<sup>2</sup>, respectively, on day 4. At this time and due to severe volume overload, CVVHDF sessions were applied until day 8, with the withdrawal of 2.5 L in total, which was well supported hemodynamically. On day 8, the patient was discharged from the ICU. Four more dialysis sessions were necessary after discharge from the ICU.

On the first postoperative day, ASAT was 5181 U/L, ALAT was 2501 U/L, and total bilirubin was 31  $\mu\text{mol/L}$ . On day two, ASAT was 1543 U/L, ALAT 1570 U/L, and total bilirubin 19  $\mu\text{mol/L}$ , and on D5, ASAT was 109 U/L, ALAT 405 U/L, and bilirubin 21  $\mu\text{mol/L}$ . On D12, all these liver parameters were normalized (Table 1).

**Table 1.** Postoperative parameters.

	Pre-Op	D1	D2	D5	D12
ALAT (U/L)	87	5181	1570	109	20
ASAT (U/L)	45	2501	1543	405	52
Bili tot (mcmol/L)	27	31	19	21	7
Creat (mcmol/L)	78	183	267	369	581
Hb (G/L)	113	84	74	76	78
MCV (fl)	100	103	104	99	97

During the 7 days in ICU, liver ultrasound was performed daily. All assessments showed normal parenchyma, arterial resistance indexes were within normal ranges, and no bile duct dilatation was identified. Thus, no acute rejection could be highlighted. Regarding immunosuppression, during the anhepatic phase, the patient received 500 mg of Methylprednisolone and 20 mg of Basiliximab. On the first day after LT, immunosuppression consisted of the combination of Mycophenolic Acid 1 g/12 h and Tacrolimus 3 mg/12 h. On the 4th day after LT, the patient received a second dose of 20 mg Basiliximab. Tacrolimus was stopped at this time for 10 days due to AKI and replaced by methylprednisolone 20 mg/day. The patient was given a dose of prednisone 20 mg on the second postoperative day and then from the 3rd day, he received 20 mg methylprednisolone daily. The Tacrolimus blood level was in a therapeutic range during the ICU stay.

Two years later, renal function remained stable, with a serum creatinine level of 120  $\mu\text{mol/L}$  and an eGFR of 58 mL/min/1.73 m<sup>2</sup>, and the kidneys showing a normal morphology and echo structure without urinary tract dilatation.

### 3. Discussion

Liver transplant patients often experience chronic kidney disease, but it is complex to pinpoint causes solely linked to the surgical or anaesthetic procedures. Indeed, several aeti-

ologies linked directly or indirectly to liver diseases can lead to renal failure, which can be immediate (in the first few hours postoperatively) or delayed (during the following days).

Risk factors for immediate renal failure after LT described in the literature are mainly associated with pre-existing chronic renal failure (hepato-renal syndrome, or a renal failure of other origin), perioperative poor renal perfusion due to hemodynamic instability, major blood loss, and decreased left cardiac preload related to vena cava and portal clamping procedures and an elevated Model for End-Stage Liver Disease (MELD) score (6 in our patient) [4,5]. In our case, we did not find any of these risk factors. Patients with portal hypertension develop hepato-renal syndrome and acute tubular necrosis [6]. Pre-LT hypovolemia can be related to renal or intestinal loss, bacterial infections, gastrointestinal bleeding, and excessive diuretic administration, which are common causes of renal failure [7]. Aminoglycosides may also be a cause of nephrotoxicity [7]. Glomerulonephritis associated with hepatitis B and C or alcoholic cirrhosis is a cause of renal failure that is not related to hemodynamic alterations [8].

Risk factors for delayed renal failure after LT described in the literature are female sex, overweight, the severity of Child-Pugh score, pre-existing diabetes, the number of red blood cell and/or fresh frozen plasma transfusions, and liver failure associated with non-alcoholic steatohepatitis [9]. Acute kidney injury or failure after LT affects not only the renal function but also the survival of the recipient [10,11]. Unexpectedly, preoperative creatinine was not a significant predisposing factor [9]. This can be explained by various factors potentially influencing the preoperative serum creatinine level in LT patients, including low muscle mass, ascites, hypervolemia, age, sex, ethnic origin, and cachexia, making the evaluation of kidney function in this setting difficult [10].

The interest of this clinical case lies in the fact that the patient had no preoperative renal function impairment and developed major renal failure within 24 h after surgery. Thus, it appears that surgical or anaesthetic factors may have had a significant impact on the perioperatively impaired renal function.

There is evidence that the number of fresh frozen plasma and packed red blood cells transfused during LT is directly correlated with the risk of developing postoperative renal failure [9]. In our case, blood loss was minor, no blood products were transfused, and crystalloid infusion was adapted to hemodynamic parameters.

Vascular exclusion has many consequences that can negatively impact renal function because renal perfusion pressure is the key to maintain renal function [10,12]. Indeed, during clamping, there is an initial decrease in cardiac output of about 40–50%. This is followed by a decrease in venous return while the mean arterial pressure drops by about 15%. Finally, the renal venous pressure gradually increases [4,5,13]. In our case, cardiac output decreased from 7 to 4.6 L/min during the anhepatic phase (79 min), whereas the mean arterial pressure (MAP) decreased from 85 mmHg to 65 mmHg under continuous iv noradrenaline infusion. As renal venous pressure is not routinely measurable intraoperatively but is a determining factor in renal perfusion, anaesthetists focus on urinary output, cardiac preload, and cardiac output homeostasis.

Following surgery, the administration of immunosuppressive medication (received intra- and postoperatively) might also play a role in the development of renal failure. The tacrolimus level from initiation to cessation on D4 due to AKI never exceeded 7 mcg/L, so a microangiopathic cause due to the treatment is highly unlikely, as this tends to occur at levels above 11.4 mcg/L (Table 2) [14].

**Table 2.** Tacrolimus level.

	D1	D2	D3	D4	D5
Tacrolimus (mcg/L)	1.4	2.5	3.0	6.8	Stop: AKI

In this case, renal function was most likely modified by the hemodynamic changes during the procedure, such as the decrease in mean arterial pressure and cardiac output as well as the increase in renal venous pressure.

During the past decade, to compensate for the lack of organs available for transplantation, DCD liver transplantations have been increasingly used. A decrease in the frequency of postoperative renal failure occurred in recipients of donated livers following brainstem death transplantation (DBD). However, an increased frequency was found in recipients of DCD livers, which was the case in the present situation [15].

In our case, it was decided not to implement a VVB because the patient did not meet the criteria according to the institution's protocol, which focus on systemic oxygen delivery and consumption assessed by central venous oxygen saturation (to be maintained over 65%). VVB diverts blood from the femoral vein and carries it to the axillary vein, thereby reducing venous congestion in the lower body, intestines, and kidneys. Bypass flow control allows the adaptation of venous return, in order to improve hemodynamic stability.

The evidence is not strong enough to assume that VVB directly improves renal prognosis after transplantation. Its implementation can lead to postoperative complications, as well as an increase in hospitalization costs and length of stay [16,17]. The requirement of VVB is therefore selective and always involves a multidisciplinary perioperative judgement.

The use of VVB improves hemodynamic stability and thus could have avoided the use of noradrenaline, which can have a negative impact on renal function. However, noradrenaline was infused for less than 24 h at a low dose (0.14 mcg/kg/min), making vasopressors an unlikely cause of AKI [1].

A final key point needs to be discussed. Preserving the vena cava flow during liver transplantation using the piggyback technique can lead to better hemodynamics (better renal arterial perfusion) [18]. Improved hemodynamics using this technique significantly reduce the need for VVB. However, the impact of this technique on postoperative AKI remains controversial. In our case, the piggyback technique would have avoided the use of noradrenaline, which could have adversely affected our patient's renal function [2,19].

Ten years after LT, the rate of chronic kidney disease is about 18% and represents a long-term poor prognosis factor. The knowledge and treatment of issues that can lead to renal failure in LT influence the short- and long-term survival of recipients [9]. It is difficult to know whether VVB could have improved renal prognosis because there is no such evidence.

#### 4. Conclusions

Renal failure after liver transplantation is common. The main concern of this clinical case lies in the absence of perioperative complications such as poorly controlled hypotension, uncontrolled decrease in cardiac output, or major bleeding. This case illustrates that the origin of renal failure is therefore more likely related to the portal and vena cava clamping procedures. As a case report, this article does not claim to offer scientific conclusions. It raises questions in an area where the evidence remains poor and opens the prospect of research into multicentre cohort studies of postoperative AKI in complex transplantation procedures. Would a VVB or piggyback have changed the renal prognosis? This question remains pending.

**Supplementary Materials:** The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/transplantology5020007/s1>, CARE Checklist of information to include when writing a case report.

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**Conflicts of Interest:** The authors declare no conflicts of interest.

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