ABO-incompatible living donor liver transplantation without graft local infusion and splenectomy

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Abstract

Background: Graft local infusion and splenectomy in ABO-incompatible (ABO-I) living donor liver transplantation (LDLT) are associated with high rates of operative complications.

Methods: Consecutive ABO-I LDLT patients treated at the National Cancer Centre between January 2012 and February 2013 were identified. The protocol for ABO-I LDLT at the study centre included the administration of rituximab (300 mg/m²) at 2 weeks preoperatively, followed by plasma exchanges (target isoagglutinin titre: ≤ 1 : 8), basiliximab (20 mg on the day of surgery and on postoperative day 4), and i.v. immunoglobulin (0.8 g/kg on postoperative days 1 and 4) without graft local infusion or splenectomy.

Results: Fifteen patients (11 men and four women) who underwent transplantation for liver cirrhosis (n = 3) or hepatocellular carcinoma (n = 12) were identified. These included 13 patients with hepatitis B virus infection, one with hepatitis C virus infection and one with alcoholic cirrhosis. The mean age, mean Model for End-stage Liver Disease (MELD) score and mean graft-to-recipient weight ratio (GRWR) of these patients was 51.8 years, 11.5 and 0.84, respectively. The median isoagglutinin titre before plasma exchange was 1:32 (range: 1:4 to 1:256). There were no hyperacute or antibody-mediated rejections. No bacterial or fungal infections were observed. Complications included herpes zoster viral infection in one patient, postoperative bleeding in one patient and extrahepatic biliary stricture in three patients.

Conclusions: This simplified ABO-I LDLT protocol showed good graft outcomes without immunologic failure or serious infections.

Received 14 October 2013; accepted 27 November 2013

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Introduction

In living donor liver transplantation (LDLT), ABO incompatibility (ABO-I) is regarded as a relative contraindication, except in urgent cases, because of the likelihood of severe rejection, hepatic arterial thrombosis and biliary complications.¹⁻³ As understanding of humoral rejection has improved, various strategies to overcome ABO-I in liver transplantation have been introduced. These include the use of plasma exchange, graft local infusion, splenectomy, aggressive immunosuppressive agents, and a monoclonal antibody such as rituximab.⁴⁻⁶

In particular, local graft infusion therapy, which involves the administration of protease inhibitors, prostaglandin and steroids through the hepatic artery or portal vein, has dramatically increased survival in ABO-I LDLT patients.⁶⁻⁸ This therapy has been considered as an inevitable step towards overcoming ABO blood type barriers. However, catheter-related complications associated with local infusion therapy have been reported to include vascular thrombosis, vascular injury, bleeding and infections.⁶ Splenectomy has been performed routinely in ABO-I LDLT in many centres.⁵⁻¹¹ The spleen is the body’s major antibody-producing organ, and contains large amounts of B cells and plasma cells. It also fulfils particular functions in blood filtration, phagocytosis, erythrocyte destruction, antigen uptake and potential haemopoiesis. However, splenectomy in ABO-I LDLT carries
risks for severe post-transplant infection and portal vein thrombosis.\textsuperscript{12,13} Splenectomy in cases of advanced liver cirrhosis is dangerous and is associated with intra- or postoperative bleeding as a result of splenomegaly. In the post-rituximab era, the clinical significance of splenectomy in ABO-I LDLT remains controversial.\textsuperscript{14}

In the present study, 15 ABO-I LDLT patients were treated using a simplified protocol involving rituximab, plasmapheresis, basiliximab and i.v. immunoglobulin (IG) without any additional surgical procedures, such as local graft infusion or splenectomy. The results indicate the potential value of an approach based on routine surgical procedures in LDLT and excluding splenectomy and local infusion therapy for ABO-I LDLT.

**Materials and methods**

**Patients**

Between January 2012 and February 2013, data on consecutive patients submitted to ABO-I LDLT at the National Cancer Centre, Goyang-si, South Korea were collected in a liver transplant database. All patients were transplanted with a right lobe from a live donor and did not undergo simultaneous splenectomy and local infusion therapy. The same ABO-I LDLT protocol was used in all patients. The medical records of all patients were retrospectively reviewed for data on patient demographics, surgical procedures, postoperative complications and follow-up information. In addition, perioperative laboratory changes in total bilirubin, prothrombin time ( PT), aspartate transaminase ( AST) and alanine transaminase ( ALT) were analysed. The study protocol was approved by the Institutional Review Board at the National Cancer Centre.

**Protocol for ABO-I LDLT**

The current immunosuppressive regimen in ABO-compatible LDLT involves the administration of high-dose steroids during the operation, followed by tacrolimus and mycophenolate mofetil and a combination of corticosteroids after transplantation. In addition, basiliximab is administered as induction therapy (20 mg on the day of surgery and on postoperative day 4). Tacrolimus was started within 2 days after LDLT. The target tacrolimus level in the first month postoperatively was 10–12 ng/ml, which was titrated down to 8–10 ng/ml over the next few months. After 1 year, a tacrolimus trough of approximately 5 ng/ml was maintained. Mycophenolate mofetil was started 2 days after LDLT at a dose of 1.5 g/day and titrated according to white blood cell count and tapered off 12 months after LDLT. Steroids were tapered to discontinuation by 6 months after LDLT.

For ABO-I LDLT, the following three steps were added to the basic protocol for ABO-compatible LDLT (Fig. 1). First, a single dose of rituximab (300 mg/m\textsuperscript{2}) was given 2 weeks prior to LDLT. Second, several sessions of plasma exchange to decrease the performed anti-donor blood type isoagglutinin antibody titre to ≤1 : 8 were started at 1 week prior to LDLT. Third, a high dose of i.v. IG (0.8 g/kg) was administered on postoperative days 1 and 4. The immunosuppressant regimen was identical to that in ABO-compatible LDLT.

The prophylactic regimen for ABO-I LDLT consisted of broad-spectrum antibiotics (ticarcillin sodium/clavulanate potassium) for 7 days, an antifungal agent (fluconazole) for 1 month, and trimethoprim-sulfamethoxazole for 1 year. This prophylactic regimen has a longer duration of use than that used in ABO-compatible LDLT. Cytomegalovirus (CMV) prophylaxis was not routinely given. Instead, CMV antigenaemia was checked twice per week until discharge, once per week until 1 month postoperatively, and then every other week or once per month as preemptive therapy. To prevent a rapid increase in hepatitis B and C viral load as a result of desensitization, antiviral agents were administered before rituximab. The prophylaxis for hepatitis B virus (HBV) recurrence after LDLT consisted of entecavir and hepatitis B IG. For recurrent hepatitis C virus (HCV), pegylated-interferon and ribavirin were administered after confirmation by biopsy or if an abnormal liver function test showed elevated HCV RNA loads.

The routine protocol biopsy for the detection of rejection was not performed. Instead, if liver function tests showed serum levels of AST, ALT and total bilirubin after LDLT to be elevated two- or three-fold higher than the normal limit without abnormal imaging tests, a biopsy was performed. In particular instances in which the isoagglutinin antibody titre was simultaneously increased to over four times that on the day of surgery or over 1 : 32, and accompanied by an increased liver function test, antibody-mediated rejection was suspected and biopsy was performed. When histopathology showed portal tract oedema, ductular reaction, and a neutrophil-rich portal inflammatory cell infiltrate without significant acute cellular rejection, antibody-mediated rejection was suspected. In such instances, C4d immu-
no fluorescence staining was performed and specific linear deposition was assumed to suggest antibody-mediated rejection. In biopsy-proven antibody-mediated rejection, the treatment plan included high doses of i.v. IG (1 g/kg/day), steroid pulse therapy and plasma exchange.

**Anti-blood type isoagglutinin titres and peripheral blood CD19+ cells**
The preformed anti-blood type isoagglutinin antibody titre was tested with the immediate spin technique. The isoagglutinin titre was checked before the administration of rituximab and serially measured after the initiation of plasma exchanges. After LDLT, isoagglutinin titre was checked daily before discharge, weekly until 1 month postoperatively, and every other week or monthly thereafter. The prevalence of peripheral blood CD19+ cells was checked before the administration of rituximab by flow cytometry, and immediately before LDLT to identify the effect of rituximab. After LDLT, the lymphocyte subset panel including CD19+ B cells was followed up twice per week until discharge, weekly until 1 month postoperatively, and monthly thereafter.

**Perioperative transfusion**
In major incompatibility, transfusions of recipient blood type or type O packed red blood cells (PRBC) and donor blood type or type AB fresh frozen plasma (FFP) and platelets were administered. In mixed major and minor incompatibility, transfusion of blood type O PRBC and blood type AB FFP and platelets were administered (Table S1, online).

**Statistical analysis**
Non-normally distributed quantitative variables were expressed as the median (centile 25; centile 75). Statistical analysis was performed using SAS Version 9.1.3 (SAS Institute, Inc., Cary, NC, USA).

**Results**

**Donor and recipient characteristics**
Fifteen patients (11 men, four women) underwent elective ABO-I LDLT at the study centre according to the centre’s protocol. Patient characteristics are summarized in Table 1. The aetiology of liver disease included HBV infection with cirrhosis (n = 2), hepatocellular carcinoma (HCC) with HBV infection (n = 11), HCV cirrhosis (n = 1), and HCC with alcoholic cirrhosis (n = 1). Of the 12 patients with HCC, pathological findings showed seven (58.3%) to be within the Milan criteria. The mean ± standard deviation (SD) recipient age and Model for End-stage Liver Disease (MELD) score were 51.8 ± 9.4 years and 11.3 ± 3.8, respectively. Living donors consisted of the respective patient’s wife (n = 5), son (n = 4), brother-in-law (n = 2), husband (n = 1), sister-in-law (n = 1), sister (n = 1) and nephew (n = 1). The mean ± SD donor age was 40.3 ± 15.0 years. The mean ± SD graft-to-recipient weight ratio (GRWR) was 0.83 ± 0.16. The mean ± SD operative time was 452.7 ± 67.0 min, mean ± SD PRBC transfusion was 4.1 ± 6.1 packs, mean ± SD total ischaemic time was 133.4 ± 37.8 min, and mean ± SD postoperative hospital stay was 13.1 ± 3.0 days. Follow-up after LDLT ranged from 3 months to 16 months.

**Isoagglutinin titre and CD19+ lymphocytes**
In all patients, several sessions of plasma exchange effectively lowered the isoagglutinin titre to ≤1:8 before LDLT. Two patients (Patients 1 and 5) displayed an elevated isoagglutinin titre of ≥1:32 at 5 days postoperatively without changes in laboratory liver function tests. These patients were treated with one cycle of plasma exchange to prevent further elevation. Titres were lowered to ≤1:32 and maintained thereafter. Patient 12 showed a four-fold increase in isoagglutinin titre and elevated liver function test findings at postoperative day 5 compared with that on the day of surgery. This patient was submitted to biopsy, two sessions of plasma exchange, and steroid pulse therapy to differentiate acute rejection. The titre and laboratory changes were controlled with the therapy, and the biopsy did not reveal acute rejection.

CD19+ lymphocytes were effectively suppressed to ≤1% of all lymphocytes with the administration of rituximab (300 mg/m²) 2 weeks before LDLT. The suppression of CD19+ lymphocytes was continued for ≥6 months in these patients.

**Patient outcomes and postoperative laboratory changes**
There were no hyperacute and antibody-mediated rejections, and no hepatic artery and portal vein thrombosis. Three patients (Patients 2, 3 and 5) were treated with percutaneous biliary drainage or endoscopic retrograde biliary drainage as a result of extrahepatic biliary stenosis. Among 13 patients with HBV infection, 10 patients showed an undetectable viral load by polymerase chain reaction (PCR) test under medication with entecavir or tenofovir. However, three patients (Patients 5, 10 and 11) showed loads of 412 234 IU/ml, 2 986 521 IU/ml and 286 274 IU/ml, respectively, before they were submitted to a desensitization protocol. These patients were started on antiviral agents before the administration of rituximab. There was no HBV recurrence after transplantation. Patient 6 showed HCV recurrence 1 month after LDLT with an elevated liver function test. The patient was successfully treated with a combination of pegylated interferon and ribavirin. There was one case of positive CMV antigenaemia (defined as antigen detection of ≥5 in 200 000 white blood cells; Patient 10) and one disseminated herpes zoster infection (Patient 13) after LDLT. However, no patient experienced bacterial or fungal infection after LDLT.

In this population, 12 patients underwent LDLT for HCC. After transplantation, two patients (Patients 3 and 5) experienced recurrent HCC with intra- and extrahepatic metastases (Patient 3 fulfilled and Patient 5 exceeded the Milan criteria). These patients were treated with multidisciplinary approaches. However, Patient 5 died of multiple tumour recurrence at 6 months after transplantation.
Table 1 Demographic and clinical data for recipients and donors in 15 cases of ABO-incompatible living donor liver transplantation

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<tr>
<th>Patient</th>
<th>Recipients</th>
<th>Donors</th>
<th>Clinical data</th>
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<td>Disease</td>
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MELD, Model for End-stage Liver Disease score (measured before plasma exchange); CTP, Child–Turcotte–Pugh score; GRWR, graft-to-recipient weight ratio; Op, operation; M, male; F, female; HBV, hepatitis B virus; LC, liver cirrhosis; HCC, hepatocellular carcinoma; HCV, hepatitis C virus.
Laboratory changes including total bilirubin, PT, AST and ALT after LDLT were normalized within 1 month postoperatively without graft dysfunction.

**Discussion**

The demand for ABO-I LDLT is increasing among patients who require immediate treatment when ABO-compatible donors are unavailable. As the safety of ABO-I LDLT has been verified in several current reports, this technique has been explored in contexts in which donor pools have been limited. However, additional procedures and acute antibody-mediated rejection have made transplant surgeons hesitant about the use of ABO-I LDLT. Of particular concern is the fact that invasive procedures, such as local infusion therapy through the hepatic artery or portal vein, and splenectomy, can induce fatal complications including vascular thrombosis, massive bleeding and severe infections. Nonetheless, dramatic improvements in patient survival since the early period of ABO-I LDLT have been noted. In the present series, the medical process in routine LDLT was modified by including the administration of monoclonal antibodies to B and T lymphocytes, plasmapheresis and i.v. IG to overcome antibody-mediated rejection in ABO-I LDLT, which allowed the procedure to be performed successfully in 15 consecutive patients without local infusion therapy or splenectomy.

Local infusion therapy was reported in 1998, and involved methylprednisolone, prostaglandin E1, and gabexate mesilate administered via catheter through the portal vein. The theoretical basis of these local infusion agents is that they inhibit different key reactions in single-organ disseminated intravascular coagulation triggered by preformed antibodies against the donor antigen. Meanwhile, hepatic artery infusion therapy was introduced to avoid portal vein thrombosis by portal vein infusion therapy. These local infusion methods have resulted in an increase in graft survival after ABO-I LDLT from approximately 40% to 60% since 2002. However, catheter-related complications, including vascular thrombosis, infection, bleeding and dislocation, have been reported to occur in 37% of patients submitted to portal vein infusion therapy, 22% of patients submitted to portal vein and hepatic artery infusion therapy, and 16% of patients submitted to hepatic artery infusion therapy. These complications can be life-threatening. Recently, Kim et al. showed no difference in liver function tests between patients undergoing local infusion therapy and systemic infusion, respectively.

In addition, splenectomy has been an important part of ABO-I LDLT in many centres because the spleen is considered a site of B cell maturation and antibody production. The clinical significance of splenectomy in ABO-I organ transplantation in the post-rituximab era is controversial. In addition to antibody production, the spleen assumes haematopoiesis and antibody-dependent cell-mediated cytotoxicity. CD20 antibody that depletes B cells by complement-dependent cytotoxicity and antibody-dependent cell-mediated cytotoxicity. CD20 antigen is expressed on most B cells except stem cells, plasma cells and T lymphocytes. Therefore, a recent trend towards the omission of splenectomy in ABO-I LDLT has emerged with the prophylactic use of rituximab.

Rituximab is a monoclonal chimeric human-murine anti-CD20 antibody that depletes B cells by complement-dependent cytotoxicity and antibody-dependent cell-mediated cytotoxicity. CD20 antigen is expressed on most B cells except stem cells, plasma cells and cells. Previously, this group used CD19 as a surrogate marker in patients with circulating rituximab because CD19 is expressed from pro-B cells to memory B cells except stem cells and plasma cells, and mirrors CD20 expression. Following the introduction of prophylaxis in ABO-I LDLT in 2002, the systematic use of rituximab from the preoperative period has been emphasized to avoid antibody-mediated rejection. The current strategy for ABO-I LDLT at this institution is to administer a single dose of rituximab 300 mg/m² at 2 weeks prior to surgery. Since the early period of ABO-I LDLT, a large dose or multiple administrations of rituximab, such as in the treatment of B cell leukaemia, have come to be considered unnecessary in view of the risk for postoperative infection. Instead, a single administration of a small dose has been accepted since the kinetics of B cells in ABO-I LDLT became better understood. Furthermore, 2–3 weeks has been reported as sufficient to perceive the effect of rituximab. In the present study, B cell levels in all patients were lowered to ≤1% at surgery 2 weeks after the administration of rituximab. The effect of rituximab was sustained for at least 6 months after administration. Plasma exchange was performed to reduce circulating anti-ABO antibodies, and was started within 1 week prior to LDLT using blood type AB FFP and albumin. The target isoagglutinin level was ≤1:8. An isoagglutinin target of 1:32 is relatively safe. However, a strict target was set because the preoperative procedure did not include the provision of mycophenolate mofetil and splenectomy. When the titre was increased four times compared with the level on the day of surgery, a prophylactic plasma exchange was performed to prevent further elevation and antibody-mediated rejection (in Patients 1, 5 and 12). The titres of these patients were decreased after one or two sessions of plasma exchange without acute rejection. The catheter for plasma exchange was removed at postoperative day 7 in patients without immunological failure. No serious catheter-related infections occurred in this study.

Basiliximab is a chimeric mouse-human monoclonal antibody to CD25 of the interleukin (IL)-2 receptor located on the surface of activated T lymphocytes. It inhibits T lymphocyte proliferation driven by IL-2 and prevents acute rejection in liver transplants. This monoclonal antibody acts by saturating the receptors and preventing T cells from replicating, and by preventing B cell activation that is necessary for the production of antibodies. In the present study, two doses of basiliximab 20 mg were administered on, respectively, the day of surgery and postoperative day 4. T cell kinetics after rituximab treatment are unclear. However, in the
present series, the proportion of helper T cells was increased for almost 1 month after ABO LDLT (data not shown). Basiliximab had been expected to restrict helper T cell activation and restrict the manufacture of antibodies because the B cells in the lymph node and spleen were not eliminated by rituximab. Furthermore, i.v. IG (0.8 g/kg) was administered on postoperative days 1 and 4. The mechanisms of action of i.v. IG have been suggested to include the blockade of Fc receptors on mononuclear phagocytes, the direct neutralization of alloantibodies, the inhibition of expression of CD19 on activated B cells, the inhibition of complement and the inhibition of alloreactive T cells. The successful use of i.v. IG without local graft infusion has been reported previously. However, these authors employed routine splenectomy in their ABO-I LDLT protocol. In ABO-I organ transplantation, i.v. IG is used in emergency transplant settings, such as in acute liver failure, in which the early administration of rituximab is not possible, and for rescue therapy for antibody-mediated rejection. In the present series, prophylactic i.v. IG administration synergistically improved outcomes in relation to the suppression of humoral reaction with rituximab, although splenectomy, local graft infusion and preoperative mycophenolate mofetil were not used. Moreover, i.v. IG has been used for the treatment of sepsis because neutralizing and opsonizing antibodies can inactivate bacterial endotoxins and exotoxins, stimulate leukocytes, and increase serum bactericidal activity. In the ABO-I LDLT setting, high-dose rituximab, plasmapheresis, splenectomy and aggressive immunosuppression markedly increase the risks for postoperative bacterial, fungal and viral infection. In the present study, no bacterial or fungal infections were evident after LDLT. Only one positive CMV antigenemia and one herpes zoster infection were detected among 15 patients without prophylactic antiviral therapy, and both were treated successfully. Intravenous IG might be helpful to prevent antibody-mediated rejection and postoperative infection in ABO-I LDLT without splenectomy and local graft infusion.

The recurrence of hepatitis virus after ABO-I LDLT was concerning in view of the aggressive immunosuppression of B lymphocytes. There was no recurrence of HBV in this study using the prophylactic therapy with a combination of hepatitis B IG and entecavir. However, the one patient enrolled with HCV infection did experience recurrence of HCV early after LDLT. In another recent study, all patients who underwent ABO-I LDLT for HCV infection experienced recurrence. In addition, patients in whom the hepatitis C viral load increased steeply early after LDLT experienced fluctuating liver function test results with a rapid increase in total bilirubin after LDLT. In the present study, the patient infected with HCV displayed a 20-fold increase in the viral RNA load at 1 month post-transplantation, with increased liver function findings, compared with the pre-transplant viral load. Therapy using pegylated interferon and ribavirin controlled the viral load. In the event of HCV recurrence, a prophylactic or pre-emptive treatment protocol might be needed in ABO-I LDLT. It is notable that the present series included a large proportion of patients with HCC (n = 12, 80.0%). Among these, two patients (Patients 3 and 5) experienced tumour recurrence at <6 months after LDLT, although the tumour in Patient 3 fell within the Milan criteria. The correlations supporting tumour recurrence in ABO-I LDLT are not well known; it is conceivable that severe immunosuppression may weaken the natural anti-tumour immune system and that tumour may be more likely to recur than in ABO-compatible LDLT. As cases accumulate, it is anticipated that the criteria for selecting ABO-I LDLT for HCC patients will become distinct from those for ABO-compatible LDLT.

Finally, the practical challenges in the application of ABO-I LDLT refer to its high cost. In the present study protocol, medications including a single dose of rituximab, several sessions of plasma exchange, two doses of basiliximab, and two doses of i.v. IG were used. Although the cost of these medications depends on the country and insurance system in place, the high costs of ABO-I LDLT place a heavy burden on both patients and clinicians. However, as the mechanism of antibody-mediated rejection becomes elucidated, it may become possible to minimize the desensitization protocol and reduce costs.

In conclusion, this simplified ABO-I LDLT protocol using rituximab, plasma exchange, basiliximab and i.v. IG without graft local infusion and splenectomy showed good graft outcomes without hyperacute or antibody-mediated rejection. This protocol describes a simple and effective mode of treatment for ABO-I LDLT.

Conflicts of interest
None declared.

References


Supporting information
Additional supporting information may be found in the online version of this article at the publisher’s website:
Table S1. Blood product selection in perioperative ABO-incompatible liver transplantation.