ISSN: 2755-0133

Journal of Medical & Clinical Nursing

SCIENTIFIC Research and Community

Research Article

Open Access

Post Reperfusion Syndrome In Liver Transplant After Normothermic Perfusion

Luciano Beltrão Pereira[1,2,3*], Juliana FH Bezerra Pereira[1], Ilka de Fátima Santana F Boin[3], Leila MMB Pereira[4], Nigel Heaton[1], Hector Vilca-Melendez[1], Wayel Jassem[1] and Zoka Milan[1]

[1]Liver Transplant Service, Institute of Liver Studies. King's College Hospital, London, UK

[2]Liver Institute of Pernambuco (IFP), Recife, Brazil

[3]Universidade Estadual de Campinas – Campinas (SP) – Brazil

[4]Universidade de Pernambuco (UPE) - Recife (PE)- Brazil

ABSTRACT

Introduction. Post reperfusion syndrome (PRS) is defined as severe haemodynamic instability, with a greater than 30% drop below the anhepatic mean arterial blood pressure (MAP) within 5 minutes of reperfusion sustained for at least 1 minute. It is an important factor in graft survival, recipient morbidity and mortality. The donor liver storage method may influence the incidence of PRS. Cooling the donor liver in cold storage (CS), is the conventional method. Normothermic Machine Perfusion (NMP) is a novel method that may reduce the incidence of PRS.

Patients and Methods. Data from a previously published randomized trial comparing donor livers preserved with CS or NMP, using Organox machine (OX), was used to perform a retrospective analysis on 22 patients (11 CS, 11 OX). Recipient and donor demographics were evenly matched between patients. The primary outcome was the incidence of PRS with secondary outcomes being inotrope, blood product and fluid requirements. Data was examined for normality, and compared using Fisher exact test or Mann-Whitney.

Results. PRS occurred in 54% (n=6) of the CS group compared to 18% (n=2) of the OX group, p=0.183. The percentage drop in MAP was significantly lower for the OX group (15.8%) compared with the CS group (40%) p= 0.007. Total amount of adrenaline used was lower in the OX group compared with the CS group, p=0.088. Total amount and number of patients on noradrenaline was similar between groups. Total fluids administered was less for the OX group (5090 mls) compared with in the CS group (7419 mls) p=0.365. The OX group received less blood products in the post reperfusion phase compared to the CS group, however this was not statistically significant.

Discussions and Conclusions: We observed a reduced incidence of PRS in patients receiving NMP compared to CS livers, with improved haemodynamics and lower adrenaline requirements.

***Corresponding author**
Luciano Beltrão Pereira, Universidade Estadual de Campinas Campinas (SP) Brazil.

Received: March 20, 2026; **Accepted:** March 24, 2026; **Published:** April 03, 2026

Introduction

Graft reperfusion during liver transplantation (LT) is a crutial phase which can be associated with major intraoperative haemodynamic and metabolic stresses. During reperfusion of the donor liver the presence of severe haemodynamic instability is commonly described as "post-reperfusion syndrome" (PRS). This syndrome is associated with a greater risk of graft injury and recipient mortality and morbidity [1]. PRS has been linked to postoperative renal failure, higher intraoperative mortality and lower early survival (<15 days after LT) [2].

The commonly accepted definition of PRS is a decrease in mean arterial pressure (MAP) of more than 30% from the value recorded during the anhepatic phase, lasting for more than 1 minute, within the first 5 minutes after reperfusion [3]. The incidence of PRS is reported between 25% and 50% [2-4] with some authors attempting to determine clinical predictors. These predictors include: graft/donor related factors, recipient factors and intraoperative factors. However, the predictive value of these factors on incidence of PRS remains controversial.

The pathophysiology of PRS is complex and poorly understood. It is thought to be influenced by liver ischemia-reperfusion injury (IRI), which is described as a 'global' event affecting the function of multiple organs including myocardium, kidney, lung, intestine and adrenal glands. Several mechanisms have been implicated in IRI pathophysiology, one of which includes activation of oxidative pathway and release of inflammatory mediators released by the ischemic liver. These mediators may produce myocardial and cellular damage resulting in acidosis, hyperkalemia, coagulation disorder and reperfusion hypothermia. This is thought to contribute to multi-organ dysfunction and haemodynamic instability which in turn impacts post-operative morbidity and mortality [5-6].

The success of liver transplantation increased the demand for donor livers without a reciprocal increase in organ pool. The waiting list for grafts increased so recently liver transplantation is increasingly dependent on the use of extended criteria donors (ECD) to include marginal quality or deemed 'high-risk' owing to donor characteristics. Typically, from older, higher body mass index (BMI), medically co-morbid donors and donors after circulatory death (DCD)[7-8].

There is evidence that marginal organs have greater vulnerability to ischemia reperfusion injury (IRI) caused by traditional preservation in cold storage (CS)[9-10]. Hence there has been increasing interest in more physiological way of perfusion marginal liver and increasing liver donation pool.

Normothermic Machine Perfusion (NMP) aims to recapitulate an environment that mimics the human body, providing the liver with oxygen and nutrition at 37 °C, it may have a role in reducing IRI and in "resuscitating" marginal organs [11-12]. Some studies have reported that NMP may have a better outcome than CS in graft preservation [13-15]. This new technique may help to attenuate the ischemic injury in marginal livers and improve intraoperative haemodynamics, however only a small number of trials on human subjects exist. Data from the first randomised controlled trial (OrganOx trial) [16] on the feasibility and safety of NMP in transplanted human liver grafts was used to characterise the haemodynamic changes related to PRS. The aim of this study was to compare PRS incidence between cold storage (CS) and warm preservation of donor liver using OrganOx® machine (OX). Secondary outcomes were to compare use of blood products, fluids and need for inotropes during the post reperfusion phase.

Methodology

Study design

This was a retrospective case controlled study, using data from the previously published OrganOX trial at the author's institution. Patients were recruited between January 2011 and December 2013 non consecutively. A total of 22 patients were included; 11 patients received a NMP OrganOx perfused liver (OX), and 11 patients formed a control group, who received liver grafts from Cold Storage (CS). Both groups received liver transplant at the same institution following contemporary transplant methods.

Both groups were matched for recipient and graft characteristics. Matching criteria for the recipient was: Age (+/- 10), MELD score, and BMI. And the matching criteria for the graft was: donor age (+/-5) and type of donation DCD Vs DBD.

Data was collected from electronic anaesthetic charts, Allscripts Electronic Patient Record (EPR) system, picture archiving and communication system (PACS) and liver donation local data base.

The following definitions were used: mean arterial pressure was defined as "the average pressure in the radial artery during 1 cardiac cycle by invasive arterial monitoring". Postreperfusion syndrome was defined as "a decrease in mean arterial pressure (MAP) of more than 30% from the value recorded during the anhepatic phase, lasting for more than 1 minute, within the first 5 minutes after reperfusion". The anhepatic phase was defined as "the duration of time from the removal of the recipient's liver to the graft reperfusion" [3].

Anaesthesia Protocol

Anaesthesia was induced with intravenous Thiopental (4-5 mg/kg) and Fentanyl (1-1.5 μg/kg) and maintained with volatile anaesthesia (isoflurane), opiate (fentanyl) and muscle relaxant (atracurium) infusions. Mechanical ventilation was characterized by a tidal volume of 6 to 10 mL/kg and a respiratory rate appropriate to achieve an end-tidal CO2 of 4 to 5.5 kPa. Invasive arterial and central venous monitoring was used. The MAP was measured by invasive BP traces during the entire operation. A continuous infusion of norepinephrine (NE) was started if required with the aim of maintaining a MAP greater than 60 to 65 mm Hg. Continuous venovenous hemofiltration was used in cases with significantly impaired renal function. At reperfusion, the rate of NE infusion was increased to maintain the MAP above 65 mm Hg, and boluses of adrenaline of 10 to 30 μg were used if the hypotension was severe or arterial pressure did not recover promptly. During surgery patients received intravenous fluids (such as crystalloids and colloids) for volume replacement, and packed RBCs to maintain a blood hemoglobin level above 70 g/L. Fresh-frozen plasma and platelet administration was guided by thromboelastography parameters.

Surgical Techniques

Only liver allografts from DBD were included. All organs were retrieved by senior surgeons and were perfused with cold University of Wisconsin solution.

For the OX Group, immediately after the liver was retrieved from the donor, the back-table preparation of the graft was performed in the donor's hospital at 4°C. The liver was connected to NMP device (OrganOx) following manufacturer protocol. For the CS group, the donor liver was preserved following national guidelines for Cold storage in the UK [17].

Analysis

Categorical variables are expressed as frequency and were analysed using Fischer's exact test. Continuous variables are expressed as medians (interquartile range) and analysed using the Student's t test or Mann-Whitney U test (as appropriate). Tests were performed using GraphPad Prism 9.0 (GraphPad Software, San Diego, California, USA) and a p value of < 0.05 was considered significant.

Results

Recipients and donors and demographics

A total number of 22 patients were retrospectively reviewed. This included 11 in the OrganOx group (OX) and 11 in cold storage control group (CS). Groups were comparable in terms of recipient demographics including age, Sex, BMI, MELD, and United Kingdom MELD score, and aetiology of liver disease (Table 1 & 2).

Table 1: Recipient Demographics

Recipient parameters	OX	CS	p Value
No. patients	11	11	
Sex (M/F)	5/6	6/5	0.9
Age	53.3(39-62)	51.8(17-66)	0.67
Recipient BMI	25.7(18.4-29.9)	24.9(17.1-31.4)	0.61
MELD score	12.8 (6-27)	12.4 (3-19)	0.88
UKELD score	50.3 (45-61)	50.2 (40-59)	0.45

UKELD, United Kingdom MELD; BML, Body Mass Index; M, male; F, female

Table 2: Indication for Orthotopic Liver Transplant (OLT)

Indication for OLT:	OX	CS
Primary sclerosing cholangitis	1	1
Primary Biliary Cirrhosis	2	1
Alcoholic cirrhosis	3	4
HCV-related cirrhosis	3	1
NASH	0	1
Autoimmune hepatitis	0	1
Other :	Hepatic cholestasis Redo OLT	Haemochromatosis Acute Wilson

HCV, hepatitis C virus; NASH, nonalcoholic steatohepatitis

Similarly, the donor profile was also comparable in the 2 groups (age, sex, BMI, inotropic support, and liver steatosis) (Table 3).

Table 3: Donor Demographics and Characteristics

Donor parameters		OX	CS	p Value
Donor Sex M/F		7/4	5/6	0.9
Donor age		60.7	57.4	0.63
Donor BMI		25 (22-28.6)	24.6 (16.6-32)	0.57
Inotropic Support		7/11	9/11	0.66
Liver Steatosis	normal	7	6	0.9
	mild	3	4	0.73
	Mild - moderate	1	1	1
	moderate	0	0	1
	Sever	0	0	1

BMI, Body Mass Index; M, male, F, female.

Grafts preserved in the OX group were perfused on the machine for a median time of 687 (324-1110) minutes before transplantation. And CS group had a median storage time of 563 (375-834) minutes (p= 0.17).

PRS and MAP Drop:

Intraoperative data showed that PRS occurred in 18% (n=2) of OX group, whereas it occurred in 54% (n=6) of the patients in the CS group (p= 0.183) - Figure 1.

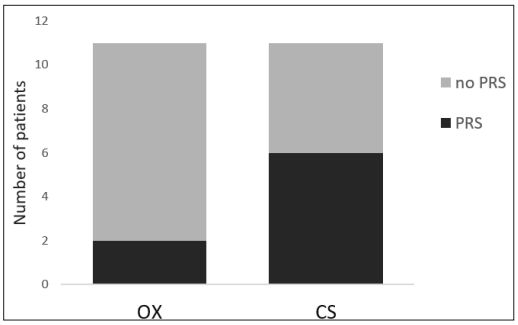

Figure 1: Incidence of Prs Observed Between Ox and Cs Group

The percentage drop in MAP in the first 5 min after reperfusion was significantly lower for the OX group, 15.8% (5-27%) compared with the CS group, 40% (26-54%) p= 0.007 - Figure.2.

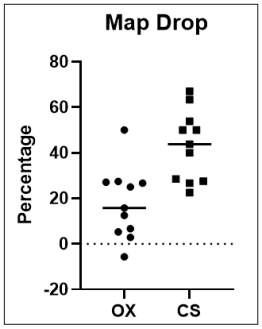

Figure 2: MAP Drop during the First 5 min of Reperfusion

Use of Fluids and blood products

There was no difference in the total operation time between the two groups (OX 4.8 hours vs CS 5.1 hours, p=0.63). The neohepatic phase (post reperfusion) was also similar in the two groups (OX 2.3 hours vs CS 2.6 hours p=0.45).

Comparing the blood products and fluids given throughout the liver transplant, the OX group received overall less packed red cells (PRC) 1391 (259- 2200) ml compared to 2053 (998-3500) ml given in the CS group, p= 0.365. Less Fresh Frozen Plasma (FFP) was given to the OX group, 1636 (1325-3508) ml vs 2712 (1831-2949) ml for the OX and CS groups respectively, p= 0.30. The total volume of crystalloid given was less for the OX group, 1500(1000- 2500) ml compared with the CS group 2000(1000-3000) ml, p= 0.898.

During the neohepatic phase, 400 (0-775) ml of PRC was transfused to the OX group compared with 600 (375-1000) ml in the CS group, p=0.223. The volume of FFP transfused to the OX group was 1100 (730-1897) ml compared with 1342 (1061-1412) ml in the CS group, p=0.705. Platelets transfused to the OX group was 0 (0-209) ml compared with 170 (0-233) ml in the CS group, p=0.223. The total volume of crystalloid given following reperfusion was 500ml in both groups, p=0.654. The total amount of blood products and fluids is described in Table 4.

Table 4: Comparison of volumes of blood products and fluids given between the OX and CS groups, during the entire operation (whole LT) and neohepatic phase. Values are quoted as median (IQR). PRC (packed red cells), FFP (fresh frozen plasma).

Whole LT procedure	OX	CS	p value
PRC (ml)	1391 (259- 2200)	2053 (998-3500)	0.365
FFP (ml)	1636 (1325-3508)	2712 (1831-2949)	0.30
Platelets (ml)	159 (0-273)	172 (0-365)	0.562
Crystalloid (ml)	1500 (1000- 2500	2000 (1000-3000)	0.898
Total(ml)	5090 (3541- 9977)	7419 (4787-9904)	0.365
Neohepatic phase			
PRC(ml)	400 (0-775)	600 (375-1000)	0.223
FFP(ml)	1100 (730-1897)	1342 (1061-1412)	0.705
Platelets(ml)	0 (0-209)	170 (0-233)	0.223
Crystalloid (ml)	500 (75-1000)	500 (0-1500)	0.654
Total(ml)	2216 (1300-3413)	2092 (1982-3451)	0.562

Inotropic Support

The total amount of adrenaline used was lower in the OX group compared with the CS group, 15 (0-20) mcg vs 20 (0-50) mcg respectively, p=0.088. There was similar number of patients requiring noradrenaline in both groups (81% and 72%) for CS group and OX group respectively. The total amount of noradrenaline administered for OX group was less 2275 (582-7050) µg compared to the CS group 1850 (860-5620) µg, p= 0.78.

Discussion

The main findings of this study was PRS occurred more frequently in donor livers preserved in CS (54%, n= 6) compared with NMP (18%, n=6), but this observation was not statistically significant. However, the degree of PRS observed between the groups was significantly less in the OX group. This was observed in the smaller reduction of % MAP drop seen within 5 minutes of reperfusion in the OX group (15%) compared with the CS group (40%), p=0.007. Blood products (PRC, FFP and Platelets) and crystalloids were given in larger volumes to patients in the CS group, this included volumes given after graft reperfusion. However, the differences observed were not statistically significant. Inotropic support was required for a similar number of patients in each group, 81% in CS group and 72% in OX group. The OX group required less adrenaline and NA compared with those in the CS group, however these differences were not statistically significant.

The mechanisms involved in PRS are complex involving multiple pathways. Shortly after reperfusion there is a sudden load of cold, acidotic blood, rich in pro-inflammatory mediators from the donor liver into the right atrium. This creates an arterial-ventriculo decoupling that increases the pulmonary vascular resistance and decreases systemic vascular resistance (SVR) [18]. These mediators have a direct effect of the left ventricle, and to a lesser extent the right ventricle. [19] This causes a reduction in heart rate, cardiac index (CI) and arrhythmias [20- 21]. Moreover, cirrhotic cardiomyopathy, hyper dynamic circulation status in the recipient and electrolyte imbalance (hyperkalaemia and hypocalaemia) post reperfusion [22-24] have been suggested as possible mechanisms of PRS [20-25].

Reperfusion of the liver during LT is one of the most critical steps for anaesthesia, as it is frequently accompanied by haemodynamic instability. A decrease in systemic blood pressure, systemic vascular resistance, and cardiac output is usually seen with a moderate increase in pulmonary arterial pressure [3]. Culminating together these haemodynamic changes are interpreted as PRS.

Ischemia reperfusion injury (IRI) is thought to be a major contributor and correlate with the severity of PRS. The ischemic insult involved in IRI starts after liver retrieval and continues throughout cold/ warm perfusion. There is thought to be two phases involved in the pathophysiology of IRI, namely the ischemic and reperfusion phases [26-27]. The ischemic phase occurs during CS due to absence of oxygenation and tissue perfusion, resulting in a sump of metabolites. The main mechanism for this is activation of xanthine oxidase, NADPH oxidase and various lytic enzymes together with the production of reactive oxygen species (ROS), inflammation, tissue acidosis and oedema. The reperfusion phase, is characterized the release and delivery of ROS in the local and the systemic circulation, inducing Kupffer cells to produce cytokines. Post reperfusion, increases in tumour necrosis factor (TNF)-a, TNF-a receptor 1, interleukin (IL)-1β, IL-1 receptor antagonist, IL-6 and IL-8 are thought to contribute to a reduction in SVR, increased pulmonary vascular resistance and myocardial depression (from cooled blood returning to the heart) [20].

NMP as a method for solid organ preservation has been studied in laboratory models, animals and more recently in clinical trials. The ability to mitigate the cold ischemia and ischemia reperfusion injury, which is thought to contribute to early graft dysfunction from IRI is one of the main benefits of normothermic organ preservation [28-30]. In a recent study of normothermic machine perfusion, liver perfusate had lower number of interferon gamma (IFN-γ) and interleukin (IL)-17–producing T cells when compared to liver perfusate from a CS livers. Moreover, NMP liver tissues showed less necrosis and apoptosis in the parenchyma and fewer neutrophil infiltration compared to CS liver tissues [9].

Our study demonstrates that some of the detrimental physiological effects caused by IRI leading to PRS could be mitigated by the use of NMP. We found a reduced severity of PRS in the OX group, with less requirements for inotropes, suggesting the effects on reduction on SVR and myocardial depression were less pronounced in the OX group. This pattern has been observed in a similar study comparing the effects of PRS in NMP and CS transplanted livers [31].The authors found a significant drop in MAP post reperfusion in the CS group compared with the NMP livers, in addition to a significant reduction in inotrope and blood product use post reperfusion in the NMP group.

The main limitation of this study was its small sample size, which limited the overall statistical power. Differences observed between the groups may have shown statistical significance with a larger sample size. A further limitation was its retrospective design, using matched controls within the constraints of a phase 1 study. We could not retrieve data to analyse the SOFT, BAR and DRI scores which could be important tools to compare this two populations. We did not have any follow up data or measure of the pro-inflammatory mediators, both of which would be useful markers in studying the sequalae of PRS. However these limitations could not be avoided as the current study used data from a previous trial investigating the safety of NMP in humans for the first time. The data reviewed was also from a single institution, which affects the generalisability of the results. Despite these limitations, we have demonstrated that NMP may offer some benefit in reducing the cold ischemia component seen during reperfusion and degree of PRS encountered. This may go onto offer some benefit in reducing the pro inflammatory cascade and subsequent global organ damage seen in IRI.

The use of NMP to facilitate more marginal graft use has gone some way to extend the donor pool. The detrimental effects of IRI and PRS are increased with the severity of liver disease, age, and graft steatosis [1,32]. As the use of marginal grafts become more common place, NMP may be a useful tool to reduce some of the physiological damage incurred during graft reperfusion. Larger multicentre trials examining the effect of PRS with NMP grafts and the effects on morbidity and mortality are needed to fully evaluate its role.

References

1. Ibtesam Hilmi, Charles N Horton, Raymond M Planinsic, Tetsuro Sakai, Ramona Nicolau-Raducu, et al.(2008) The impact of postreperfusion syndrome on short-term patient and liver allograft outcome in patients undergoing orthotopic liver transplantation. Liver Transpl 14: 504-8.
2. Catherine Paugam-Burtz , Juliette Kavafyan, Paul Merckx, Souhayl Dahmani, Daniel Sommacale et al. (2009) Postreperfusion syndrome during liver transplantation for cirrhosis: outcome and predictors. Liver Transpl 15: 522-529.
3. Aggarwal S, Kang Y, Freeman J A, Fortunato F L, Pinsky M (R1993) Postreperfusion syndrome: hypotension after reperfusion of the transplanted liver. J Crit Care 8: 154-160.
4. Ayanoglu HO, Ulukaya S, Tokat Y (2003)Causes of postreperfusion syndrome in living or cadaveric donor liver transplantations. Transplant Proc 35: 1442-1454.
5. Constantinos Nastos, Konstantinos Kalimeris, Nikolaos Papoutsidakis , Marios-Konstantinos Tasoulis , Panagis M Lykoudis, et al. (2014) Global consequences of liver ischemia/

reperfusion injury. Oxid Med Cell Longev 906965.
6. Serracino-Inglott F, Habib NA, Mathie R T (2001)Hepatic ischemia-reperfusion injury. Am J Surg 181: 160-166.
7. Ceresa CDL, Nasralla D, Jassem W (2018) Normothermic Machine Preservation of the Liver: State of the Ar Curr Transplant Rep 5: 104-110.
8. Fungai Dengu, Syed Hussain Abbas , Georg Ebeling, David Nasralla (2020) Normothermic Machine Perfusion (NMP) of the Liver as a Platform for Therapeutic Interventions during Ex-Vivo Liver Preservation: A Review. J Clin Med 9.
9. Wayel Jassem, Emmanuel Xystrakis, Yasmeen G Ghnewa, Muhammed Yuksel, Oltin Pop, et al. (2019) Normothermic Machine Perfusion (NMP) Inhibits Proinflammatory Responses in the Liver and Promotes Regeneration. Hepatology 70: 682-695.
10. Xu J, Blayne Amir Sayed, Ana Maria Casas-Ferreira, Parthi Srinivasan, Nigel Heaton, et al. (2016) The Impact of Ischemia/Reperfusion Injury on Liver Allografts from Deceased after Cardiac Death versus Deceased after Brain Death Donors. PLoS One 11: e0148815.
11. Graham JA, Guarrera JV (2014) Resuscitation of marginal liver allografts for transplantation with machine perfusion technology. J Hepatol 61: 418-431.
12. Guarrera JV (2012)Assist devices: machine preservation of extended criteria donors. Liver Transpl 18: S31-S33.
13. Constantino Fondevila, Amelia J Hessheimer, Mark-Hugo J Maathuis, Javier Muñoz, Pilar Taurá, et al. (2011)Superior preservation of DCD livers with continuous normothermic perfusion. Ann Surg 254: 1000-1007.
14. Jens Brockmann, Srikanth Reddy, Constantin Coussios, David Pigott, Dino Guirriero, et al. (2009) Normothermic perfusion: a new paradigm for organ preservation. Ann Surg 50: 1-6.
15. Shiqi Bian , Zhijun Zhu, Liying Sun, Lin Wei, Wei Qu, et al. (2018) Normothermic Machine Perfusion versus Cold Storage of Liver in Pig Model: A Meta-Analysis. Ann Transplant 23: 197-206.
16. R Ravikumar, Jassem W, Mergental H, Heaton N, Mirza D et al. (2016) Liver Transplantation After Ex Vivo Normothermic Machine Preservation: A Phase 1 (First-in-Man) Clinical Trial. Am J Transplant 16: 1779-87.
17. Zalewska K (2018) National Standards for Organ Retrieval from Deceased Donors., NHS Blood and Transplant 50.
18. Acosta F, Reche T, Sansano RF, Contreras R, Beltran, et al. (1999) Effect of reperfusion on right ventriculoarterial coupling in liver transplantation. Transplant Proc 31: 2384-2395.
19. Christian Rosendal, Keneshbek Almamat Uulu, Raffaele De Simone, Ivo Wolf, Andreas Deckert, et al. (2012) Right ventricular function during orthotopic liver transplantation: three-dimensional transesophageal echocardiography and thermodilution. Ann Transplant 17: 21-30.
20. Antonio Siniscalchi, Lorenzo Gamberini, Cristiana Laici, Tommaso Bardi, Giorgio Ercolani, et al., (2016) Post reperfusion syndrome during liver transplantation: From pathophysiology to therapy and preventive strategies. World J Gastroenterol 22: 1551-1569.
21. Cristina Ripoll, María-Vega Catalina, Raquel Yotti, Luis Olmedilla, José Pérez-Peña et al. (2008) Cardiac dysfunction during liver transplantation: incidence and preoperative predictors. Transplantation 85: 1766-1772.
22. Zhen-Dong Xu, Hai-Tao Xu, Hong-Bin Yuan, Hao Zhang, Rui-Hua Ji, et al. (2012) Postreperfusion syndrome during orthotopic liver transplantation: a single-center experience. Hepatobiliary Pancreat Dis Int 11: 34-39.
23. Siniscalchi A, Aurini L, Spedicato S, Bernardi E, Zanoni A, et al. (2013)Hyperdynamic circulation in cirrhosis predictive factors and outcome following liver transplantation. Minerva Anestesiol 79: 15-23.
24. Chui AK, Shi L, Tanaka K, Rao A R, Wang L S (2000) et al, Postreperfusion syndrome in orthotopic liver transplantation. Transplant Proc 32: 2116-2127.
25. A Nanashima, Pillay P, Crawford M, Nakasuji M, Verran D J, et al. (2001) Analysis of postrevascularization syndrome after orthotopic liver transplantation: the experience of an Australian liver transplantation center. J Hepatobiliary Pancreat Surg 8: 557-563.
26. Jaeschke H (2003) Molecular mechanisms of hepatic ischemia-reperfusion injury and preconditioning. Am J Physiol Gastrointest Liver Physiol 284: G15-26.
27. Fondevila C, Busuttil RW, Kupiec-Weglinski JW (2003) Hepatic ischemia/reperfusion injury--a fresh look. Exp Mol Pathol 74: 86-93.
28. Thomas Vogel, Jens G Brockmann, Constantin Coussios, Peter J (2012) FriendThe role of normothermic extracorporeal perfusion in minimizing ischemia reperfusion injury. Transplant Rev (Orlando) 26: 156-162.
29. Sarah A Hosgood , Ernest van Heurn, Michael L Nicholson (2015) Normothermic machine perfusion of the kidney better conditioning and repair? Transpl Int 28: 657-664.
30. Herman Tolboom, Asya Makhro, Barbara A Rosser, Markus J Wilhelm, Anna Bogdanova, et al. (2015) Recovery of donor hearts after circulatory death with normothermic extracorporeal machine perfusion. Eur J Cardiothorac Surg 47: 173- 179.
31. Roberta Angelico, Thamara PR Perera M, Reena Ravikumar, David Holroyd, Constantin Coussioset al. (2016) Normothermic Machine Perfusion of Deceased Donor Liver Grafts Is Associated With Improved Postreperfusion Hemodynamics. Transplant Direct 2: p e97.
32. In S Chung, Ha Y Kim, Young H Shin, Justin S Ko, Mi S Gwak,et al. (2012)Incidence and predictors of post-reperfusion syndrome in living donor liver transplantation Clin Transplant 26: 539-543.