Changes in the Gasometric and Hemodynamic Profile upon Graft Reperfusion in Living Donor Kidney Transplant Patients

Alfonso de Jesus Flores Rodriguez*[1], Enrique Morales Zepeda[2], Karla Joselyne Manrique Marines[3], Mariana Elisa Guillen Camacho[4], Javier Vázquez Falconi Justiniani[5], Ricardo Daniel Romero Morelos[6], Paulina Carpinteyro Espin[7]

[1]Critical Care Medicine and Anesthesiology Physician. Hospital Juarez de México, CDMX. Abdominal Organ Transplant Unit.

[2]Critical Care Medicine and Internal Medicine Physician. Hospital General de México "Eduardo Liceaga", CDMX. Obstetric Intensive Care Unit

[3]General Surgery and High specialty in Abdominal Organ Transplant Surgery. Hospital Juarez de México, CDMX. Abdominal Organ Transplant Unit.

[4]Transplant Anesthesiology Physician. Hospital Juarez de México, CDMX. Abdominal Organ Transplant Unit.

[5]Third year Anesthesiology Resident. Hospital Juarez de México, CDMX.

[6]General Surgery and High specialty in Abdominal Organ Transplant Surgery. Hospital Juarez de México, CDMX. Abdominal Organ Transplant Unit.

[7]General Surgery and High specialty in Abdominal Organ Transplant Surgery. Hospital Juarez de México, CDMX. Abdominal Organ Transplant Unit.

ABSTRACT

Introduction: In the field of medicine, the study of hemodynamics is fundamental to understanding the functioning of the cardiovascular system and its impact on tissue oxygenation. Within this context, crucial parameters such as mean systemic filling pressure (MSFP), cardiac output (CO) and oxygen extraction rate (EO2) play essential roles in regulating tissue perfusion and oxygen delivery to the lungs the vital organs. In the case of kidney transplant recipient patients, the interaction between these parameters acquires unique relevance, given the intimate relationship between cardiovascular function and the new implanted kidney.

Material And Methods: Material And Methods: A descriptive, observational, single-center, and retrospective study was carried out undergoing living donor kidney transplant patients between June 2022-2023 at the Hospital Juarez de Mexico.

Results: 54 patient records were examined, 24 with exclusion criteria. Remaining 30, 18 were female (60%) and 12 were male (40%); Receiver: Age 30.46±11.68 years; Weight: 59.93±11.87kg; Size: 156.20±18.74cm; Hemoglobin: 9.87±1.62g/dl. Prior hemodynamic values to reperfusion: CaO2 (arterial content) 13.44 ± 2.22, CvO2 (venous content) 9.07 ± 2.20, Da-vO2 (arterio-venous difference) 3.67 ± 1.48, CO (cardiac output) 5.65 ± 3.13, VS (stroke volume) 76.05±41.73, SVR(systemic vascular resistance) 852.61±396.72, DO2(oxygen delivery) 770.89±444.07, VO2(oxygen consumption) 239.33±29.38, EO2(oxygen extraction) 26.83±9.82, PMSF (mean systemic filling pressure) 17.47±3.09 mm Hg with Student's T con values of p<0.05 statistically significant; Hemodynamic values after reperfusion: CaO2 13.3±2.18, CvO2 8.11±1.61, Da-vO2 5.03±1.04, CO 4.57±1.26, VS 62.32±17.41, SVR 1265.97±419.41, DO2 621.66±150.83, VO2 239.33±29.38, EO2 37.62 ±5.28, PMSF 14.27±2.44 mm Hg with Student's T statistically significant values of p<0.05.

Conclusions: Undergoing kidney transplantation patients strategies that include the optimization of PMSF, CO and EO2 based on their determinants can be implemented to improve perfusion and

ARTICLE DETAILS

Changes in the Gasometric and Hemodynamic Profile upon Graft Reperfusion in Living Donor Kidney Transplant Patients

oxygenation of kidney transplanted prior kidney graft reperfusion, which in turn contributes to longterm viability and improved quality of life for the transplant recipient.

I. INTRODUCTION

End-stage renal disease (ESRD) represents a clinical condition of extreme severity, characterized by a significant kidney function loss, which leads to an accumulation of metabolic products and imbalances in the hydroelectrolyte balance. Hemodialysis, one of the main dialysis methods, emerges as a vital treatment for patients with ESRD, as it seeks to partially replace the function of the kidneys by filtering and purifying the blood through an extracorporeal circuit.

The hemodynamic profile in ESRD patients undergoing hemodialysis is a complex entity that involves a series of physiological changes and compensatory adaptations. These changes are intrinsically linked to underlying kidney dysfunction and the nature of the hemodialysis procedure itself.

One of the central aspects in the hemodynamic profile of these patients is high blood pressure, which occurs in a significant proportion of individuals with ERET. The alteration in the renin-angiotensin-aldosterone system and the increase in sodium and water retention are key factors in the development of hypertension. In addition, decreased nitric oxide secretion and imbalance in endothelial function also contribute to elevation of blood pressure.

Another crucial component in the hemodynamic profile of ESRD with hemodialysis is cardiovascular system dysfunction. Uremia, which is the accumulation of nitrogenous products in the blood due to renal failure, leads to a series of changes in the structure and function of the heart and blood vessels. This includes left ventricular hypertrophy, myocardial fibrosis, and endothelial dysfunction, increasing the risk of adverse cardiovascular events[1].

In addition volume overload is also a common characteristic in these patients, due to difficulty excreting water and sodium efficiently. Hemodialysis seeks to address this imbalance by eliminating excess fluids and metabolic products accumulated in the body, contributing to maintaining a state of euvolemia.

Furthermore, it is important to highlight that hemodialysis itself can have a significant impact on the patients hemodynamic profile with ESRD. During the procedure, a series of acute changes in blood pressure levels and fluid distribution occur, requiring constant monitoring and careful treatment adjustments.

The lack of validated and easy-to-apply methods to measure body fluid volume status likely contributes to the high prevalence of volume excess in hemodialysis (HD) patients.

In most centers, volume status is ordinally estimated based on clinical criteria, i.e., patient signs and symptoms, peridialytic blood pressure measurements, and intradialytic hemodynamic instability. However, previous evidence suggests that clinical evaluation of volume balance is a method with limited reliability[2].

Kidney Transplant

Kidney transplantation is now recognized as the best treatment for patients with end-stage kidney disease due to its superior outcomes in patient survival and quality of life. Due deceased people organ donations scarcity, living donor kidney transplants are now widely performed. Despite marked improvements in transplant techniques, the incidence of delayed graft function (DGF) in living donor kidney transplant has not decreased. The underlying pathophysiology of DGF could involve donor, recipient, or surgeon-related factors, as well as hemodynamic (ischemic reperfusion) or immunological processes (particularly T cells).

Several studies have suggested that on living donor kidney transplantation an intraoperative target mean arterial pressure (MAP) of >95 mm Hg and a central venous pressure (CVP) of >15 cm H2O (12 mm Hg) at the timing of reperfusion may result in immediate graft function. However, the appropriate intraoperative target for blood pressure and volume status is poorly understood.

Other research papers have demonstrated that intraoperative targeting of MAP >95 mm Hg and CVP >15 cm H2O at the time of reperfusion could lead to immediate graft function. A MAP of <70 mm Hg at reperfusion was associated with DGF; Achieving SAP 150 at the time of reperfusion and in the early postoperative period may be associated with early ATN recovery after living donor kidney transplantation. However, these data suggest that recipients with a long history of dialysis are less likely to achieve SAP 150 at the time of unclamping in living donor kidney transplantation[3].

II. MATERIAL AND METHODS

This is a retrospective, descriptive and analytic study. It was conducted in the Kidney Transplant Unit in Hospital Juarez de México from June 2022-2023. The sampling was non probabilistic by convenience. Inclusion criteria included kidney orthotopic adult patients with 7Fr central venous catheter. Those with decompensated kidney disease due to bleeding or cardiovascular dysrhythmias and those operated without 7Fr central venous catheter were excluded. The aim

Changes in the Gasometric and Hemodynamic Profile upon Graft Reperfusion in Living Donor Kidney Transplant Patients

was to correlate the geometric and hemodynamic variables before and after reperfusion in transplant patients.

Kidney records for the period from June 2022-June 2023 were reviewed according to selection criteria. Demographic data were obtained, and gasometric and hemodynamic variables including the following: central venous pressure (CVP), mean arterial pressure (MAP), arterial oxygen content (CaO2), venous oxygen content (CvO2), arterio-venous oxygen difference (DA-VO2), cardiac output (CO), stroke volume (SV), systemic vascular resistance (SVR), O2 delivery (DO2), O2 consumption (VO2), O2 extraction (EO2), mean systemic filling pressure (MSFP) and central venous oxygen saturation (ScVO2) were recorded before (1 minute) and after reperfusion (3 minutes). To obtain the variable MSPF the formula was used: RAP (CVP) x 0.96 + MAP x 0.04 + CO x0.5; Cardiac output was obtained through the formula: SV x Heart Rate.

Procedure

By standardized anaesthetic procedure at the Hospital Juarez de México, general anaesthesia was administered to all patients with intravenous Fentanyl at a dose of 2mcg/kg, Propofol at 2mg/kg and Rocuronium at a dose of 0.60 mg/kg with a latency of 4 minutes and ventilation with Intermittent manual positive pressure with FIO2 100%. All patients underwent atraumatic laryngoscopy with endotracheal tubes that varied in the case of women (6.5-7.5 DI) and in the case of men (7.5-8.5 DI). They were connected to an anaesthetic circuit with mechanical ventilation in airway protective parameters in PCV-VG mode VTE 6-8 ml/kg, RR 10-17 rpm, I:E 1:2.5, PEEP 6-8 according to PEEP/ARDSnet. For anaesthetic maintenance, Desflurane of 0.8-1.2 MAC with Sedline (30-50 PSI) is used. The left or right jugular vein was cannulated with a 7FR central catheter according to the exhaustion of the patient's central venous access and a 20G arterial line was placed in the left or right radial according to the presence of arteriovenous fistula.

All patients were administered 3 boluses of 100 ml/hr each to dilute medications (antibiotic therapy, glucocorticoid, antihistamine) and are subsequently maintained at a dose of 10-15 ml/kg/hr and additional boluses of intravenous fluid. 250 ml of Hartman solution in case of hypotension (defined with SBP <90 mm Hg or MAP <60 mm Hg). Type II monitoring of heart rate, continuous blood pressure and central venous pressure (CVP) is maintained. In case of having a CVP greater than 15 mm Hg and hypotension, multiple boluses of Ephedrine 5 mg IV (boluses) are administered until the episode resolves.

Statistical analysis

The data were integrated into an Excel database and statistical processing was performed in SPSS or STATSm software. Descriptive statistics were used to obtain measures of central tendency (median, standard deviation and range

from discrete variables, mean and frequencies for nominal variables. The Kolmogorov-Smirnov test was performed.

Unpaired Student's t test was calculated to compare pre-reperfusion and post-reperfusion means. Non-normal data were analysed using Mann-Whitney U. All statistical analyses were performed with a value of p<0.05, considering these significantly.

This work was performed with the approval of the institutional research and ethics committees. The present study is considered without research risk in accordance with the regulations of the general health law on health research.

III. RESULTS

54 files of patients who underwent kidney transplantation between June 2022 and June 2023 were reviewed and only 30 met the selection criteria.

Eighteen female (60%) and twelve male (40%) patients were including with a median age of 30.46±11.68 years with a weight of 59.93±11.87 kg, height 156.20±18.74 cm. Initial hemoglobin was 9.87±1.62 g/dL.

Hemodynamic values prior to reperfusion (table 1)

At the alveolar level, the CaO2 (arterial oxygen content) was found to be 13.44±2.22 ml/O2 with a CvO2 (venous oxygen content) of 9.07±2.20 ml/O2, which results in a DA-VO2 (arterial-venous difference of oxygen) of 3.67±1.48 ml/O2 with the formula: DA-VO2 = CaO2-CvO2 (fig1).

Table 1. Mean Hemodynamic Variables before and after reperfusion in kidney transplant patients.

VARIABLE	BEFORE REPERFUSION	AFTER REPERFUSION	p Value
Arterial Oxygen Content ml/O2 (CaO2)	13.44 ± 2.22	13.3 ± 2.18	p=0.0025
Venous Oxygen Content ml/O2 (CvO2)	9.07 ± 2.20	8.11 ± 1.61	p=0.0030
Arterio-Venous Oxygen Difference ml/O2 Da-V2)	3.67 ± 1.48	5.03 ± 1.04	p=0.0019
Cardiac Output Lt/min (CO)	5.65 ± 3.13	4.57 ± 1.26	p=0.0028

Changes in the Gasometric and Hemodynamic Profile upon Graft Reperfusion in Living Donor Kidney Transplant Patients

|			
Stroke Volume ml/beat (SV)	76.05 ± 41.73	62.32 ± 17.41	*p=0.0039*
Systemic Vascular Resistance dynas/cm^5 (SVR)	852.61 ± 396.72	1265.97 ± 419.41	*p=0.0033*
Mean Arterial Pressure mm Hg (MAP)	85.49 ± 11.22	87.74 ± 11.29	*p=0.0029*
O2 Delivery ml/min (DO2)	770.89 ± 444.07	621.66 ± 150.83	*p=0.0046*
O2 Consumption ml/min (VO2)	239.33 ± 29.38	239.33 ± 29.38	*p=0.0037*
Oxygen Extraction % (EO2)	26.83 ± 9.82	37.62 ± 5.28	*p=0.0028*
Central Venous Pressure mm Hg (CVP)	12.32 ± 2.97	9.51 ± 2.39	*p=0.0032*
Mean Systemic Filling Pressure mm Hg (MSFP)	17.47 ± 3.09	14.27 ± 2.44	*p=0.0023*
Central Venous Oxygen Saturation % (SCVO2)	66.5 ± 9.13	59.3 ± 4.84	*p=0.0037*
*Student T			
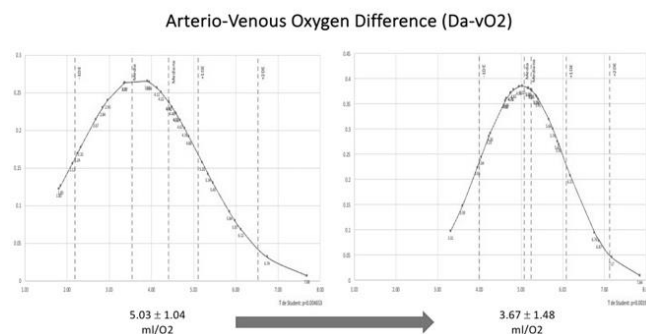
Figure 1. Left: Non normal distribution of values of arterio-venous oxygen difference before reperfusion; Right: Non normal distribution of values of arterio-venous oxygen difference after reperfusion in kidney transplant patients.

In the left heart, the CO (cardiac output) was 5.65 ± 3.13 Lt/min (fig 2) with the formula: CO = HR*SV; The SV (systolic volume) was 76.05±41.73 ml/beat and the SVR (systemic vascular resistance) was 852.61±396.72 dynes/cm5.
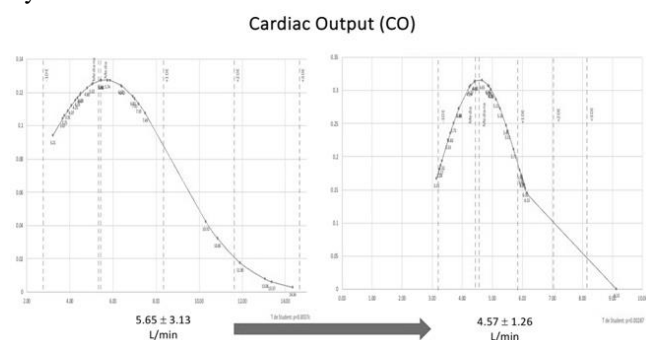

Figure 2. Left: Non normal distribution of values of cardiac output before reperfusion; Right: Non normal distribution of values of cardiac output after reperfusion in kidney transplant patients.

For the kidney graft, the DO2 (oxygen delivery) was 770.89±444.07 ml/min while the VO2 (oxygen consumption) was 239.33±29.38 ml/min. Therefore the EO2 (oxygen extraction) 26.83±9.82% using the formula: EO2=DO2-VO2 (fig3).
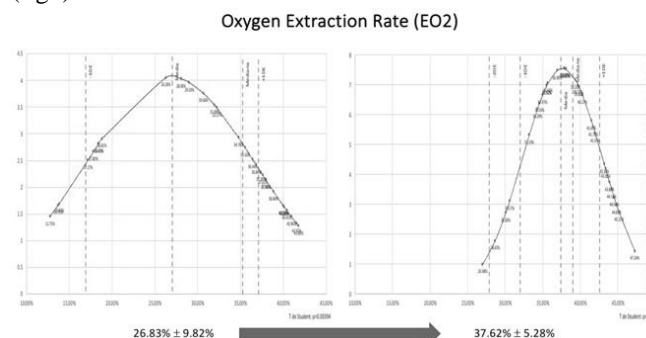

Figure 3. Left: Non normal distribution of values of oxygen extraction rate before reperfusion; Right: Non normal distribution of values of oxygen extraction rate after reperfusion in kidney transplant patients.

Changes in the Gasometric and Hemodynamic Profile upon Graft Reperfusion in Living Donor Kidney Transplant Patients

And at the end in the right heart, it is found with a CVP (central venous pressure) 12.32±2.97 mm Hg, MAP (mean arterial pressure) 85.49±11.22 mm Hg, which when integrated into the PMSF (mean systemic filling pressure) 17.47± 3.09 mm Hg from the formula: PMSF=(PVC*0.96)+(PAM*0.04)+(GC*0.3) (fig4). Likewise, SCVO2 (venous oxygen saturation) was 66.5±9.13% with p values <0.005 considered statistically significant.
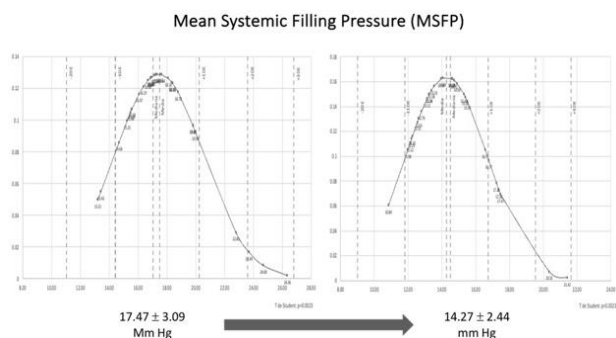

Figure 4. **Left: Non normal distribution of values of mean systemic filling pressure before reperfusion; Right: Non normal distribution of values of mean systemic filling pressure after reperfusion in kidney transplant patients.**

Hemodynamic values after reperfusion (Table 1):
At the alveolar level, CaO2 was found to be 13.3±2.18 ml/O2 with a CvO2 of 8.11±1.61 ml/O2, therefore a DA-VO2 of 5.03±1.04 ml/O2 is obtained from the same formula previously referenced (fig5).
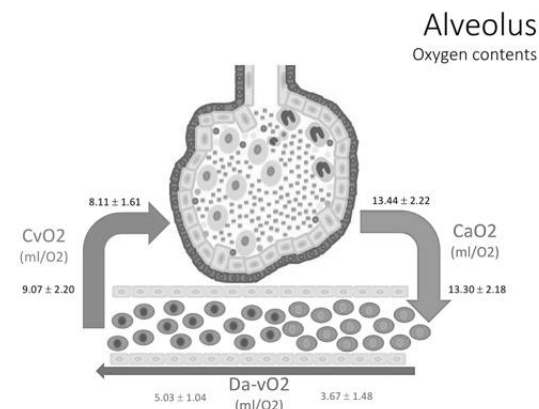

Figure 5. **Alveolus and Oxygen Contents before and after reperfusion in kidney transplant patients.**

At the level of the left heart, the CO was 4.57±1.26 Lt/min (fig6) with an SV 62.32±17.41 ml/beat and SVR 1265.97±419.41 dynas/cm5.
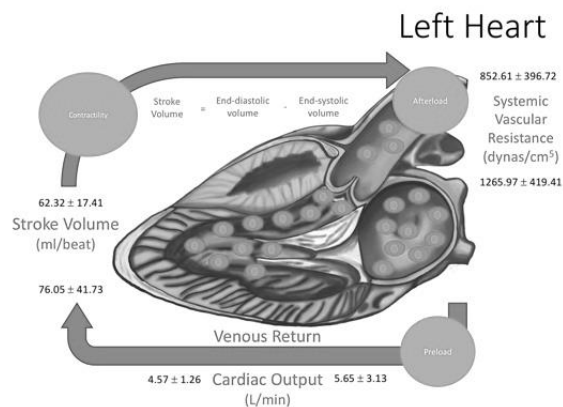

Figure 6. **Left Heart Hemodynamic Variables before and after reperfusion in kidney transplant patients.**

In the kidney graft the DO2 621.66±150.83 ml/min with a VO2 239.33±29.38 ml/min and an EO2 37.62±5.28% from the previously referenced formula (fig7).
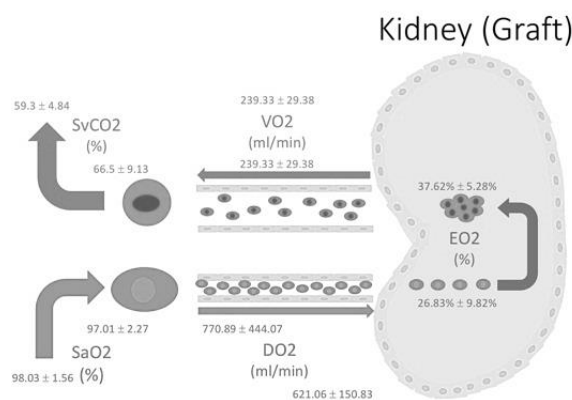

Figure 7. **Oxygen Delivery and Consumption Values in Kidney Graft before and after reperfusion in kideny transplant patients**

Finally, in the right heart a CVP of 9.51±2.39 mm Hg, a MAP of 87.74±11.29 mm Hg and a PMSF of 14.27±2.44 mm Hg were found (fig8). The SCVO2 was 59.3±4.84% with values of p<0.005 considered statistically significant.

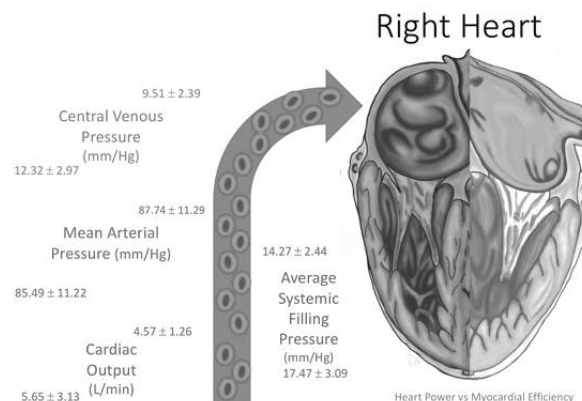Figure 8. **Right Heart Hemodynamic Variables before and after reperfusion in kidney transplant patients**

Corresponding Author: Alfonso de Jesus Flores Rodriguez

Changes in the Gasometric and Hemodynamic Profile upon Graft Reperfusion in Living Donor Kidney Transplant Patients

At fluid balance. The value of total input was 3850±1250 ml; Crystalloids: Hartman 2350±850 ml and Normal Saline 1150±650 ml. Albumin dosage of 0.5 mg/kg was found in only 40% (12 patients) and balanced solutions only in 30% (9 patients).

The total output value was 2650±1300 ml with a bleeding value of 270±150 ml. The ischemia values were: 5.6 ± 4.3 minutes while for cold ischemia it was 45.6 ± 18.2 minutes and for warm ischemia it was 40.4 ± 9.7 minutes.

IV. DISCUSION

The Arteriovenous Oxygen Difference (AD-VO2) is an indicator of the metabolic oxygen demand of the graft as it demonstrates the efficiency with which the transplanted kidney extracts oxygen from the blood at the time of reperfusion. In the case of our study, kidney transplant patients have pre-surgical anemia (9.87±1.62 g/dL), which is why there is no correct capacity for uptake and transport of O2 to the tissues, so as the metabolic demand for O2 increases, On the part of the kidney graft, the patient goes from a state of normo-hyperdynamic, with a DAVO2 of 3.67±1.48 ml/O2, to a state of hypodynamic with a DAVO2 of 5.03±1.04 ml/O2 post-reperfusion. This value reflects that the cardiovascular system may not adequately temporarily supply the high oxygen demand by the kidney graft.

Cardiac Output (CO) refers to the amount of blood that the heart pumps in a certain period of time. End-diastolic volume limits the cardiac function curve of the Fick principle. Thus, the hemodynamic profile presented by patients undergoing renal function replacement therapies exhibits normal or increased CO (5.65±3.13 Lt/min) with decreased SVR (852.61±396.72 dynes/cm5) and, as a consequence, SCVO2 (66.5±9.13%) is normal or increased due to a decrease in the rate of tissue oxygen extraction at the peripheral level and the increase in tissue oxygen delivery secondary to the elevated GC. Upon reperfusion with a decrease in preload due to greater extraction of oxygen and volume by the kidney graft, this distributive hemodynamic profile changes to a transient hypovolemic hemodynamic profile in which the CO decreased (4.57±1.26 Lt/min) causes a decrease in SCVO2 (59.3±4.84%) with a subsequent increase in SVR (1265.97±419.41 dynes/cm5) to maintain a MAP (87.74±11.29 mm Hg) and thus attempt to improve tissue perfusion.

From the pathophysiological point of view, the understanding between DO2 and VO2 is essential. DO2 refers to the amount of oxygen that the heart delivers to the tissues and is determined from the cardiac output and the blood oxygen content (CaO2). The determinants of CaO2 correspond to the concentration of hemoglobin, arterial oxygen saturation (SaO2) and the partial pressure of arterial oxygen (PaO2).

The difference between oxygen delivery vs. consumption corresponds to the amount of oxygen extracted by peripheral tissues during each cardiac cycle or also known as oxygen extraction (EO2). Upon reperfusion of the kidney graft, VO2 (239.33±29.38 ml/min) remains constant, however DO2 decreases (770.89±444.07 ml/min pre-reperfusion to 621.66±150.83 ml/min) linearly with each cardiac cycle due to the high oxygen demand at the tissue level (26.83±9.82% pre-reperfusion to 37.62±5.28%). This phenomenon occurs to a certain point where the critical oxygen extraction rate causes a decrease in both VO2 and DO2; Upon reaching this point, the anaerobic metabolism in the tissues begins and the resulting increase in the production of lactic acid and with these direct implications on the viability and functionality of the graft.

Central venous pressure is an indirect marker of intravascular volume status since it only estimates the preload of the right ventricle through the interaction of venous return to the right atrium and filling of the right ventricle. Initially, a pre-reperfusion CVP of 12.32±2.97 mm Hg indicates a state of volume overload; however, after reperfusion the CVP decreases to a value of 9.51±2.39 mm Hg. However, the CVP, being a static parameter, must be supported by a dynamic parameter that includes the average pressure of the circulatory average and that fully reflects the preload of the heart.

The mean systemic filling pressure pre-reperfusion was 17.47±3.09 mm Hg and post-reperfusion the value was 14.27±2.44 mm Hg indicating that the cardiac preload has decreased and therefore it passes from a state of overload only tolerated through treatments. substitutes for renal function where said state increases ventricular cardiac work and congestion, however, by reducing excess fluids, the pressure of the kidney vessels decreases and thus increases the effective flow from the patient to the graft, positively affecting the function and the filtration capacity of the new kidney.

V. CONCLUSIONS

Fluid overload is a potentially serious complication in kidney transplant patients at the time of graft reperfusion. Careful and accurate fluid management is essential to prevent complications and ensure a successful recovery.

Patients undergoing kidney transplantation, prior to reperfusion, experience a change in hemodynamic profile from a distributive type (increased CO, VS and decreased SVR) to a hypovolemic type (decreased CO, VS and increased SVR), which allows an increase in contractility to volume expense.

The kidney graft displays an increase in energy demand (EO2 and SCVO2) and volume (PMSF and its determinants). This is when the patient goes from an independent preload zone due to a heterometric adaptation (non-responsive/non-volume tolerant) to a dependent preload zone with a homomeric adaptation (volume responder/tolerant).

The results presented in the study are consistent with the line of thought in world literature, which is why a randomized

Changes in the Gasometric and Hemodynamic Profile upon Graft Reperfusion in Living Donor Kidney Transplant Patients

controlled clinical trial of parallel groups is required to demonstrate these results prospectively.

SPONSORSHIP

The authors declare not having received support from any sponsor or resources outside those granted by the medical institution.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

REFERENCES

I. Wagener, G.; Bezinover, D.; Wang, C.; Kroepfl, E.; Diaz, G.; Giordano, C.; West, J.; Kindscher, J.D.; Moguilevitch, M.; Nicolau-Raducu, R.; et al. Fluid Management During Kidney Transplantation: A Consensus Statement of the Committee on Transplant Anesthesia of the American Society of Anesthesiologists. Transplantation 2021, 105, 1677–1684.

II. Halawa, A.; Rowe, S.; Roberts, F.; Nathan, C.; Hassan, A.; Kumar, A.; Suvakov, B.; Edwards, B.; Gray, C. A Better Journey for Patients, a Better Deal for the NHS: The Successful Implementation of an Enhanced Recovery Program after Renal Transplant Surgery. Exp. Clin. Transplant. 2018, 16, 127–132

III. Prionas, A.; Craddock, C.; Papalois, V. Feasibility, Safety and Efficacy of Enhanced Recovery After Living Donor Nephrectomy: Systematic Review and Meta-Analysis of Randomized Controlled Trials. J. Clin. Med. 2020, 10, 21

IV. Espino, K.A.; Narvaez, J.R.F.; Ott, M.C.; Kayler, L.K. Benefits of multimodal enhanced recovery pathway in patients undergoing kidney transplantation. Clin. Transplant. 2018, 32, e13173.

V. Sarnak, M.J.; Amann, K.; Bangalore, S.; Cavalcante, J.L.; Charytan, D.M.; Craig, J.C.; Gill, J.S.; Hlatky, M.A.; Jardine, A.G.; Landmesser, U.; et al. Chronic Kidney Disease and Coronary Artery Disease: JACC State-of-the-Art Review. J. Am. Coll. Cardiol. 2019, 74, 1823–1838.

VI. Chadban, S.J.; Ahn, C.; Axelrod, D.A.; Foster, B.J.; Kasiske, B.L.; Kher, V.; Kumar, D.; Oberbauer, R.; Pascual, J.; Pilmore, H.L.; et al. KDIGO Clinical Practice Guideline on the Evaluation and Management of Candidates for Kidney Transplantation. Transplantation 2020, 104, S1–S103.

VII. Jha, A.K.; Lata, S. Kidney transplantation in valvular heart disease and pulmonary hypertension: Consensus in waiting. Clin. Transplant. 2021, 35, e14116.

VIII. El-Boghdadly, K.; Cook, T.M.; Goodacre, T.; Kua, J.; Denmark, S.; McNally, S.; Mercer, N.; Moonesinghe, S.R.; Summerton, D.J. Timing of elective surgery and risk assessment after SARS-CoV-2 infection: An update: A multidisciplinary consensus statement on behalf of the Association of Anesthetists, Centre for Perioperative Care, Federation of Surgical Specialty Associations, Royal College of Anesthetists, Royal College of Surgeons of England. Anesthesia 2022, 77, 580–587.

IX. Ljungqvist, O.; de Boer, H.D.; Balfour, A.; Fawcett, W.J.; Lobo, D.N.; Nelson, G.; Scott, M.J.; Wainwright, T.W.; Demartines, N. Opportunities and Challenges for the Next Phase of Enhanced Recovery After Surgery: A Review. JAMA Surg. 2021, 156, 775–784.

X. Smudla, A.; Trimmel, D.; Szabo, G.; Fazakas, J. Systolic Blood Pressure Pattern: The Tick Mark Signal of Delayed Renal Graft Function. Transplant. Proc. 2019, 51, 1226–1230.