## **Annals of Case Reports**

Martelli LM, et al. Ann Case Rep: 7: 1005. www.doi.org/10.29011/2574-7754.101005 www.gavinpublishers.com

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## **Case Report**

## Resolution of Uremic Pericarditis after Living Donor Kidney Transplantation: A Case Report

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Citation: Martelli LM, Di Vico MC, Fava A, De Ferrari GM, Gallo E, et al. (2022) Resolution of Uremic Pericarditis after Living Donor Kidney Transplantation: A Case Report. Ann Case Report. 7: 1005. DOI: 10.29011/2574-7754.101005

Received Date: 19 October 2022; Accepted Date: 22 October 2022; Published Date: 25 October 2022

#### **Abstract**

**Introduction:** End-stage renal disease (ESRD) is a relevant metabolic cause for the onset of pericarditis and pericardial effusion. Uremic pericarditis has different therapeutic perspectives such as intensification of dialysis sessions, non-steroidal anti-inflammatory drugs, and corticosteroids (Systemic or Intrapericardial) or Pericardiocentesis. In patients with pericardial effusion and severe renal impairment colchicine, is contraindicated. Kidney transplantation is the treatment of choice for kidney failure and living donor kidney transplantation represents the best option in terms of graft function and longevity. **Case Presentation:** A 45-years-old Caucasian man with ESRD secondary to IgA glomerulonephritis, starting dialysis treatment in 2011. First kidney transplant in 07/04/2015. Following gradual worsening of kidney function. The echocardiographic checks were accompanied by a gradual increase in pericardial effusion, probably on uremic basis. Patient was admitted and started dialysis sessions and Pericardiocentesis, but the cardiac effusion reappeared. He underwent a living donor renal transplantation with good kidney function and at eight days following an echocardiogram was performed which showed the total disappearance of the effusion. **Discussion:** Our case appears to be the first report in literature of total and rapid resolution of uremic pericarditis with refractory pericardial effusion after living donor kidney transplantation, suggesting that the occurrence of uremic pericarditis should not delay transplantation when a living donor is available.

**Keywords**: Kidney transplantation; Living donor; Uremic pericarditis

#### Introduction

End-stage renal disease (ESRD) is a relevant metabolic cause for the onset of pericarditis and pericardial effusion. The term uremic pericarditis is used to describe patients who develop clinical manifestations of pericarditis before starting renal replacement therapy [1].

The uremic state and pericarditis associated with dialysis represent the same nosological entity caused by uremia / dialysis inadequacy [2].

The pericardium is the fibroelastic sac surrounding the heart composed by 2 layers separated by a space filled with about 50 ml of pericardial fluid [2].

Volume 7; Issue 05

Ann Case Rep, an open access journal

ISSN: 2574-7754

The term acute pericarditis refers to inflammation of this fibroelastic sac; even small increases in volume induce high intrapericardial pressure increases with consequent cardiac tamponade; the same phenomenon occurs in chronic conditions with a gradual accumulation of fluid [3].

Clinically, pericardial effusion may be asymptomatic or induce cardiac tamponade by inducing dyspnea, tachypnea, tachycardia, and symptoms and signs of hypoperfusion [3].

The diagnosis can be made using a transthoracic echocardiogram which allows you to assess the extent of the effusion and can also guide the pericardiocentesis procedure. The hemodynamic significance of the effusion does not strictly depend on the extent but also depends on the accumulation speed [3].

Uremic pericarditis has different therapeutic perspectives such as intensification of dialysis sessions with progressive weight reduction [2], non-steroidal anti-inflammatory drugs and corticosteroids (systemic or intrapericardial) that may be considered in case of intensive dialysis inefficacy [4] or Pericardiocentesis. This surgical procedure is performed in cases of non-response to previous treatments or in severe symptomatic cardiac tamponade according to the ESC guidelines [2]. In patients with pericardial effusion and severe renal impairment colchicine, is contraindicated [4].

Kidney transplantation is the treatment of choice for kidney failure and living donor kidney transplantation represents the best option in terms of graft function and longevity.

We propose a clinical case of a patient with uremic pericarditis with an effusion refractory to dialysis and pericardiocentesis who was promptly resolved after living donor renal transplantation.

#### **Case Presentation**

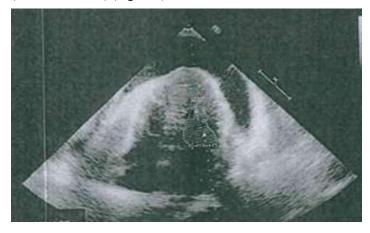
A 45-years-old Caucasian man with ESRD secondary to IgA glomerulonephritis, starting dialysis treatment in 2011. He then received a kidney transplant in 07/04/2015. Following gradual worsening of kidney function in 2018, he was submitted to a kidney biopsy that showed chronic antibody-mediated rejection with aspects of GSFS. Tocilizumab treatment was started [5].

Beginning in November 2018, episodes of severe hypertensive crisis with initial onset of mild pericardial effusion occurred.

Given the functional worsening and gradual central and peripheral overload, Tocilizumab was discontinued in 06/2019. At the same time, the echocardiographic checks were accompanied by a gradual increase in pericardial effusion, probably on uremic basis. On 15/10/2020 the echocardiogram showed a moderate circumferential pericardial effusion (lateral of 26 mm, apical of 19 mm, inferior-apical of 23 mm, close to the right atrium 26 mm).

In the meantime, his wife proposed a kidney donation. She was studied following the ERBP guidelines [6] and was considered eligible for donation.

The patient was admitted to our division in October 2020. Patient was asymptomatic but with objective central and peripheral overload with a blood pressure of 144/98 mmHg, body weight 80.5 Kg, and apyretic, serum creatinine of 5, 66 mg/dl and urea 235 mg/dl. Daily dialysis sessions were started after CVC placement in right jugular vein with weight loss of 10 Kg. After six dialysis sections, the echocardiogram showed a worsening of the effusion (max. about 3 cm) (Figure 1).



**Figure 1:** Circumferential pericardial effusion, on a uremic basis, of severe degree (max anteriorand posterior echo-covering space of about 3 cm), with "swinging heart" aspect and "flag" movement of the right atrium.

The cardiologist consultant indicated an evacuative pericardiocentesis with drainage of about 1000 ml performed. When the effusion completely disappeared, the drainage was removed.

From the dialysis point of view, the patient continued sessions at a four-weekly rate.

The result of the analysis of the liquid confirmed the suspicion of transudative effusion of uremic origin with negative cytology and cultures.

On rechecking 7 days after removal of the drainage, the reappearance of a thin layer of effusion anterior to the right ventricle by 1 mm was highlighted. A few days later the patient underwent a further pre-transplant echocardiogram which showed an increase in the effusion with circumferential flap (max 10 mm posterior to the left ventricle).

A few days after, he underwent a living donor renal transplantation that had immediate diuretic and functional response with rapid decrease of creatinine down to the minimum value of 1.2 mg/dl which subsequently stabilized around values of 1.5-1, 6

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mg/dl (Table 1), and contextual further weight loss down to 62.5-63 kg (Table 2). The induction therapy used was: Thymoglobuline plus high dose corticosteroids (cumulative dose of 900 mg) and maintenance with Tacrolimus, MMF and prednisone

Variables	Upon admission	Immediate pre-transplant	A week after the transplant	At the time of discharge
Hb (g/dl)	10,8	11,2	11,4	12,3
WBC (n/microL)	7040	6400	6430	7200
Neutrophils (%)	64,1	51,1	86,7	58,5
Lymphocytes (%)	21,2	36,4	5	33,3
Monocytes (%)	12,5	8,3	7,8	6,4
PLTs (n/microL)	168000	222000	143000	243000
Na+ (mmol/L)	139	136	138	140
K+ (mmol/L)	3,8	4,2	3,6	4,5
Uric acid (mg/dl)	4,4	2,7	6	4,1
Urea (mg/dl)	235	69	52	58
Creatinine (mg/dl)	5,66	3,28	1,36	1,45
Ph	7,29	7,37	7,31	7,37
HCO3-(mmol/L)	18	27,6	21,5	24,3
BE	-8,1	4,2	-3,6	-0,1
Ca2+ (mg/dl)	2,49	2,5	2,16	2,44
PO4- (mg/dl)	1,37	1,4	0,72	0,74
Total protein (g/dl)	6,2	6,2	5,3	6,3
Albumin (g/dl)	4,2	4,2	3,6	4,2
PCR (mg/L)	5,9	1	44,7	0,9
Proteinuria (g/die)	0,623	0,339	0,846	0,328
Clearance (ml/min)	13	13	62	63
FK (ng/ml)	4,1	6,6	11,5	9,2
PTH (pg/ml)	264	264	91	30

**Table 1.** Trend of blood chemistry tests.

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Variables	Upon admission	After intensive dialysis sessions	After pericardiocentesis	Immediate pre- transplant	A week after the transplant
Weight (Kg)	80,5	71	71	69	62,5-63
Circumferential spill (cm)	2,6	3	Absent	1	absent
EF (%)	70%	70%	70%	70%	62%
PAPs (mmHg)	20	20	32	unknown	22

Table 2. Weight and echocardiographic trend.

At eight days following transplant, an echocardiogram was performed which showed the total disappearance of the effusion (Figure 2).

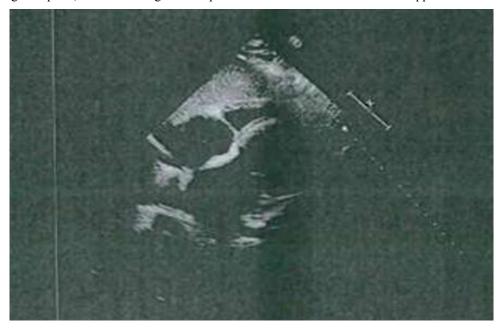


Figure 2: No pericardial effusion is detected.

#### **Discussion**

So far, no cases of resolution of pericardial uremic effusion in the post kidney transplant are described; in literature the strategy employed in ESRD patient is intensive dialysis.

Our case shows a persistence of pericardial effusion, despite intensive dialysis and weight loss of about 11 kg and in presence of purifying efficacy.

Transplantation from a living donor led to a total resolution of the pericardial effusion, as demonstrated by echocardiography performed at eight day following transplant.

From the laboratory point of view, it is interesting to highlight how with the same blood levels of uremic toxins dosed both during intensive dialysis treatment and in the post-transplant period, the efficacy on the resolution of the effusion only manifested itself after the kidney transplant.

Patients with ESRD have vascular phenotypic characteristics that are unique (presence of vascular calcifications, inflammation, oxidative stress, dyslipidemia) that seem to have a role in mediating an increased cardiovascular mortality and kidney transplantation provides a marked reduction of this risk compared to dialysis [7].

However, kidney transplantation does not eliminate the increased risk of CV disease, although it was associated with drastic changes in risk factors already in the early phases of follow-up. The most important CV risk factors which improved after the renal transplant were hypertension, uremic state, inflammation, anemia, left ventricular hypertrophy, which could justify the positive impact of transplantation on the development of CV disease [8].

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Often the Kidney transplant recipients from deceased donors had more time on dialysis therapy with a longer exposure to the uremic milieu, to inflammation, and to hypertension which are implicated in the greater prevalence of cardiovascular complications like uremic pericarditis compared to living donor. [9] In our case, the possibility of living donor allowed to reduce the time on dialysis which contributing to a more rapid resolution of pericardial effusion.

Immediate graft function, which is more common after living donor transplantation, may have played an important role in rapid resolution of uremic state and consequently disappearance of pericardial effusion.

In addition, the use high dose of corticosteroids for induction immunosuppressive treatment may have contributed to this effect. Indeed, glucocorticoids are considered for the therapy of uremic pericarditis in case of inefficacy of intensive dialysis [4].

In conclusion, our case appears to be the first report in literature of total and rapid resolution of uremic pericarditis with refractory pericardial effusion after living donor kidney transplantation, suggesting that the occurrence of uremic pericarditis should not delay transplantation when a living donor is available.

**Disclosure**: The author declared no competing interests

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