# Management of Pulmonary Hypertension, Acute Right Sided Congestive Cardiac Heart Failure, and Cranial Vena Cava Syndrome in a Dog undergoing Hemodialysis

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## ABSTRACT

The Objective of this case report was to describe the successful conservative management of life-threatening cardiopulmonary complications following renal replacement therapy (RRT) in a dog, attributable to hemodialysis central venous catheterization. An 8-year-old, 15-kg neutered male dog with overhydration, anuria, International Renal Interest Society Grade IV acute kidney injury (AKI) and *Ehrlichia canis* infection underwent continuous and prolonged intermittent renal replacement therapy (CRRT and PIRRT) respectively. Following the tearing and dislodgement of the initial right-sided hemodialysis catheter and its subsequent replacement with a catheter in the left jugular vein, the dog developed severe complications. These included cranial vena cava syndrome (CVCS), pulmonary hypertension (PH) leading to right-sided congestive heart failure (R-CHF), pleural and pericardial effusions with secondary cardiac tamponade. Concurrent pulmonary thromboembolism (PTE) was suspected but not confirmed. Echocardiography and clinical manifestations confirmed R-CHF and PH with a 49-mmHg systolic trans-tricuspid regurgitation pressure gradient and mild right atrial dilation, right ventricular hypertrophy, with reduced caudal vena cava collapsibility. Medical treatment including Rivaroxaban, Pimobendan, and Tadalafil was instituted. Finally, the removal of the hemodialysis central venous catheter (HCVC) was assumed to be the key factor leading to the resolution of the cardiac tamponade. Following this intervention and concurrent medical treatment for the R-CHF, PH and suspected PTE, the dog went on to make a full clinical recovery. To the best of the authors' knowledge, this is the first report of a successful dual therapeutic approach to severe HCVC complications, where catheter removal resolved the life-threatening cardiac tamponade while medical therapy addressed the concurrent PH and R-CHF.

**Key words:** Renal replacement therapy complications; Catheter-induced cardiopulmonary disease; Veterinary hemodialysis; Suspected iatrogenic acute pulmonary thromboembolism.

## INTRODUCTION

Although Renal Replacement Therapy (RRT) offers a life-saving intervention, it is not without significant risks. Various complications are associated with HCVC in humans, including catheter associated thrombosis, pulmonary thromboembolism (PTE), pericardial/pleural effusions, and superior vena cava syndrome (SVCS) (1-3). In dogs, case reports with HCVC complications include a report of fibrin sheath formation (4), as well as two cases with catheter associated thrombosis, chylopericardium, and pleural effusion (5). This report describes a case of transient CVCS, PH, and R-CHF in a dog with anuric AKI and concurrent ehrlichio-

sis undergoing RRT, which resolved with a combination of medical treatment and definitive HCVC removal.

## Renal Replacement Therapy Protocol

A dual-lumen hemodialysis catheter (11-Fr, 25-cm dual-lumen dialysis catheter, GAMCATH, Short-Term Catheters, Deerfield, IL, USA) was placed in the right jugular vein and an esophageal feeding tube was inserted on the left side of the neck (Esophageal feeding tube, Mila International, Inc., Covington, KY). Thoracic radiographs confirmed the catheter's correct placement, with its distal tip located in the right atrium, and revealed a mild diffuse interstitial pattern. The RRT was performed using a Prismaflex system (Prismaflex CRRT System, Baxter Healthcare Corp., Deerfield, IL, USA) in a Continuous Venovenous Hemodiafiltration (CVVHDF) mode with regional citrate anticoagulation (6) . Blood flow rates were kept at 30-40 mL/min, and fluid removal was titrated at up to 40 mL/hr. Between sessions, the catheter was locked with unfractionated heparinized saline (100 units/kg/ml) (Unfractionated heparin, Teva Medical, Ashdod, Israel).

The RRT included two CRRT sessions over the first 7 days (effluent dose: 37 mL/kg/hr; durations: 48 hours each; interdialytic periods: 24 and 48 hours), followed by four PIRRT sessions over 10 days (effluent dose: 290 mL/kg/hr; durations: 6, 8, 8, and 8 hours; interdialytic periods: 24-48 hours).

## Case Presentation

An 8-year-old, 15-kg, neutered male mixed-breed dog was referred for RRT due to anuria, IRIS Grade-4 AKI (serum creatinine concentration 8.5mg/dL; reference interval (RI) 0.4-1.30 mg/dL), 10% overhydration (based on physical examination and an acute increase in bodyweight) with concurrent pancreatitis and severe thrombocytopenia (12,000/μL; RI 148,000-484,000/μL) secondary to *Ehrlichia canis* diagnosed via polymerase chain reaction (PCR, Karnieli Laboratory, Kiryat Tiv'on, Isarel). Initial therapy included placement of a right-sided HCVC and initiation of RRT.

## In-Hospital Complications

On day seven of hospitalization, the dog developed subcutaneous (SQ) facial and neck pitting edema, most prominently on the right side where the HCVC was placed. A minimal volume abdominal effusion was also detected. On day eight, following the placement of a new HCVC in the left jugular vein, the SQ edema progressed to become severe, diffuse and generalized across the head and neck, consistent with CVCS.

On day 17, the dog collapsed due to high-pressure, low-volume cardiac tamponade. Emergency pericardiocentesis was performed, which had to be repeated four additional times over the next 24 hours. A new right-apical, grade 4/6 systolic murmur was subsequently auscultated. After the final RRT session, the HCVC was removed due to its suspected contributing role in causing CVCS, triple effusion, and worsening azotemia, all presumed to have developed secondary to recurrent cardiac tamponade. Although non-pericardial cavitary effusions persisted, no recurrent tamponade occurred. Worsening pleural effusion required repeated thoracocentesis (removing 400 mL on day 20 and 170 mL on day 21). Fluid analysis of pericardial and pleural effusate characterized them as transudates (Total Solids 1.4-1.6 g/dL) with low cellularity and a few non-degenerate neutrophils, reactive macrophages, and mesothelial cells. Significant clinical progress was made following HCVC removal, marked by the resolution of recurrent cardiac tamponade, SQ head and neck edema and improvement in azotemia. The dog was subsequently discharged on a conventional medical regimen for the at-home management of its post-dialysis acute kidney disease (Table 1).

## Post-Discharge Follow-up and Resolution

One week after discharge, deteriorating azotemia and a newly suspected cardiac arrhythmia prompted evaluation by a board-certified cardiologist. A 6-lead ECG tracing confirmed the presence of a complex arrhythmia. The findings included sinus tachycardia (170 bpm), a left axis deviation (60°) of the mean electrical axis of the ventricular depolarization process, and frequent ventricular premature complexes (VPCs) with recurrent right ventricular bigeminy at a fixed coupling interval as in re-entry, potentially indicating ventricular myocardial irritability. While the axis deviation may reflect a left bundle or a left fascicular conduction anomaly, which is often seen in healthy cats as well, the right-sided electrocardiographic changes were considered more important (Figure 1). Combined with the new loud right-sided murmur, right ventricular hypertrophy, and previous triple effusion, these findings raised concern for iatrogenic acute pulmonary thromboembolism (PTE), although this was not definitively diagnosed.

**Table 1:** Summary of Medical and Nutritional Management at Discharge and Following Cardiology Evaluation

| Medication | Dose | Route | Frequency |
|---|---|---|---|
| **Initial Discharge Medications** | | | |
| Maropitant (Maropitant, Zoetis, Israel) | 1 mg/kg | PO/ E-Tube | q 24h |
| Metoclopramide (Metoclopramide, Rafa Laboratories, Israel) | 0.5 mg/kg | PO/ E-Tube | q 12h |
| Mirtazapine (Mirtazapine, Unipharm LTD, Israel) | 0.7 mg/kg | PO/ E-Tube | q 24h |
| Amlodipine (Amlodipine, Teva-medical, Ashdod, Israel) | 0.2 mg/kg | PO/ E-Tube | q 12h |
| Doxycycline (Doxylin, DEXCEL, Or Akiva, Israel) | 10 mg/kg | PO/ E-Tube | q 24rh |
| Lactated Ringer's Solution (Lactated Ringer's solution, Teva-medical, Ashdod, Israel) | 10 ml/kg | SC | q 12h |
| Hill's Prescription Diet i/d (I/D low-fat Hill's, Hill's Pet Nutrition Israel) | 120 mL of slurry[a] per feeding | E-Tube | q 6d [b] |
| Vitamin B12 (vitamin B12, Raz Pharmaceutics LTD, Israel) | 50 µg/kg | SC | q7d |
| **Therapy Initiated Post-Cardiology Consultation** | | | |
| Pimobendan (Pimobendan, Vetmarket LTD, Israel) | 0.25 mg/kg | PO | q 12h |
| Tadalafil (Tadalafil, Inovamed Pharme LTD, Israel) | 0.75 mg/kg | PO | q 12h |
| Rivaroxaban (Rivaroxaban , BAYER LTD, Israel) | 0.5 mg/kg | PO | q 12h |

Abbreviations: PO, Per os; SC, Subcutaneous; q, quaque-every; d, day; h, hour; [a]1 can (370g, ~380 kcal) blended with 1/2 can (185 mL) water; [b]To meet 100% of RER.

Doppler echocardiography (General Electric, S70, Israel) confirmed tricuspid valve regurgitation with mild-to-moderate pulmonary hypertension (PH), with a peak systolic trans-tricuspid regurgitation pressure gradient of 49 mm Hg, mild right atrial dilation, right ventricular hypertrophy, and a congested caudal vena cava with diminished collapsibility. Notably, left ventricular systolic function remained preserved (fractional shortening: 54%; LA: 2.3 cm; LA/Ao: 1.53). The systolic trans-pulmonary valve pressure gradient was normal at 9.08 mm Hg, and tricuspid annular plane systolic excursion (TAPSE) was 1.6 cm (WNL for this dog's body weight) (7). Tricuspid annular systolic velocity was also normal at 0.24 m/s. Additional findings included residual pericardial effusion. No thrombi were seen in cardiac chambers or main pulmonary arteries. Based on these findings, the therapeutic regimen was expanded (Table 1).

At a two-month follow-up examination, the dog was clinically normal. The heart murmur had resolved, and a repeat echocardiogram confirmed the complete resolution of tricuspid regurgitation or signs of R-CHF with no direct or indirect evidence supportive of PH. The systolic pulmonary artery pressure gradient had decreased to 4.28 mm Hg, the triple effusion had resolved, and the caudal vena cava showed normal collapsibility (~40%). Renal function had also improved significantly (serum creatinine of, 1.7 mg/dl; IRIS CKD Stage 1). The Left Bundle Branch Block (L-BBB) which was initially documented, was absent but occasional VPCs persisted, warranting ongoing monitoring. The therapeutic regimen was adjusted accordingly (Table 1). At a six-month follow-up examination, the dog was thriving without any medications, exhibiting no arrhythmia on ECG, and maintaining stable renal function with a stable serum creatinine concentration of 1.7 mg/dL; IRIS CKD Stage 1.

## DISCUSSION

This case demonstrates that a severe iatrogenic HCVC complications can occur during RRT but may be fully reversible. The immediate, life-threatening complications of cardiac tamponade and CVCS were resolved promptly following the removal of the HCVC. This rapid improvement supported our clinical impression that the HCVC was the primary cause for CVCS and cardiac tamponade which, in turn, might have contributed to progressive azotemia as a tertiary complication resulting from the ensuing hemodynamic compromise. Had further RRT been required, peritoneal dialysis would have potentially been considered as an alternative strategy. As the number of veterinary hemodialysis centers continues to grow, it is crucial to document such complications, as well as the outcome of their attempted management.

Complications from central venous catheter placement

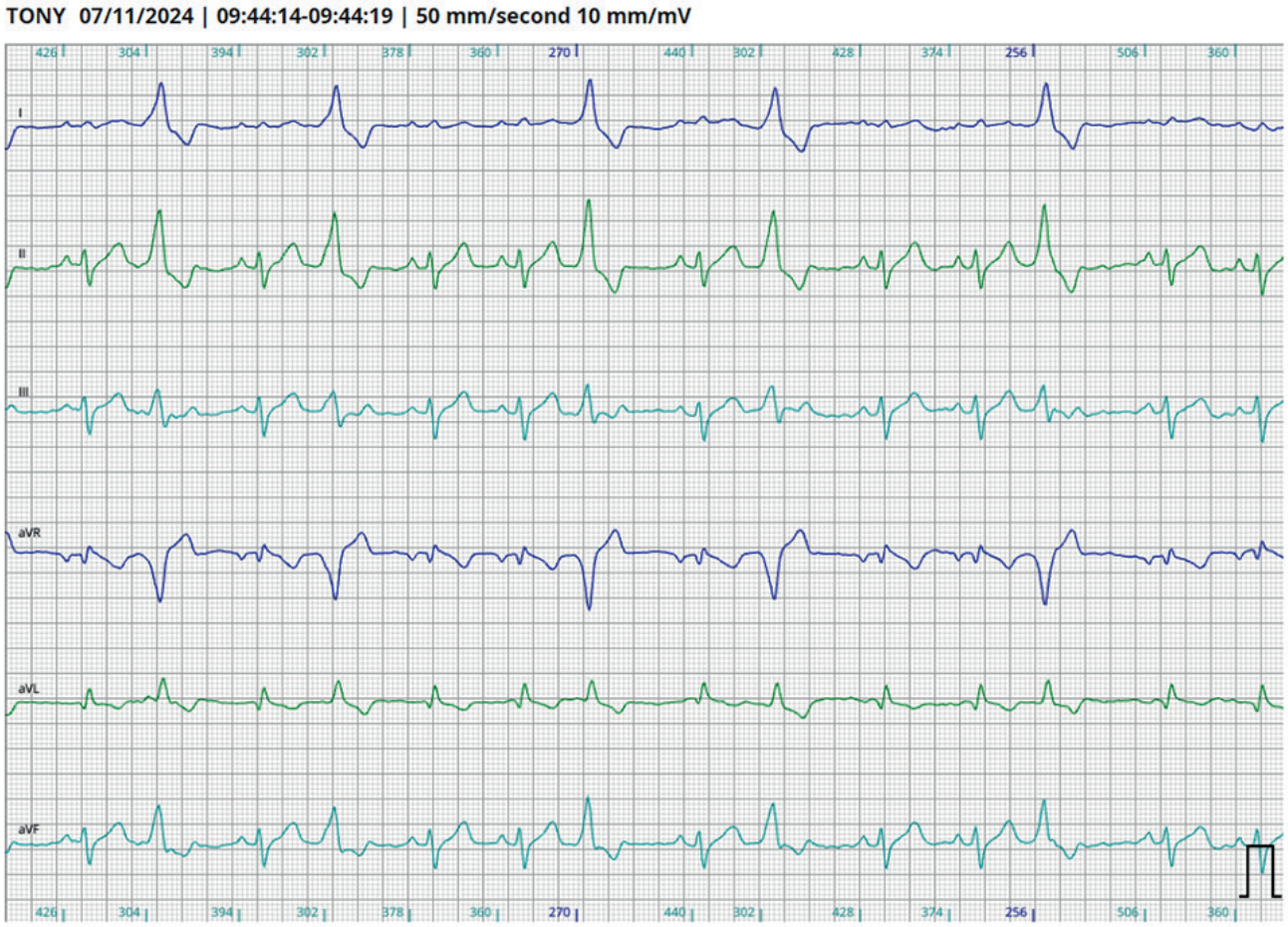


**Figure 1:** ECG shows sinus tachycardia with frequent ventricular premature complexes (VPCs), with frequent right ventricular bigeminy. This pattern is compatible with right ventricular enlargement with ventricular wall distention and frequent ventricular ectopy originating from the right ventricle (See text for details).

are well-documented in dogs and include malposition, hematoma, dysrhythmias, mechanical obstruction, migration, infections, thrombus formation (leading to CVCS) and stricture (8). Studies on HCVC complications in dogs are scarce. One case report described a fibrin sheath formation that compromised catheter function (4). Another report documented the development of catheter associated venous thrombosis and chylopericardium in two dogs undergoing chronic hemodialysis: one dog developed right jugular and cranial vena cava thrombus along the HCVC with cranial mediastinal and proximal thoracic limb edema and chylopericardium with a modified transudate pleural effusion, while the other developed right jugular vein thrombus with both chylopericardium and chylothorax. In both cases, the complications were attributed to suspected catheter-associated venous thrombosis which proved refractory to medical and surgical intervention (5).

The development of both pleural and pericardial effusions in this and in the similar mentioned cases, can be explained by the pathophysiological principles of venous hypertension. In an experimental study, researchers inflated balloon catheters in the cranial and caudal vena cava and left atrium (raising retrograde right atrium pressure) of 32 dogs to induce systemic venous hypertension and high right atrial pressure, respectively, and then measured the amount of fluid that subsequently accumulated in the pleural and pericardial spaces (9). While elevated systemic venous pressure alone resulted in significant pleural fluid accumulation, a significant increase in pericardial fluid occurred only when this was combined with high right atrial pressure. This model closely mirrors the events in the present case: the CVCS created severe systemic

venous hypertension, providing a clear mechanism for the large-volume pleural effusion (9). Concurrently, the dog's PH and R-CHF created the required high right atrial pressure, likely contributing to the development of pericardial effusion. This is consistent with the leading hypothesis that high right atrial pressure compromises the normal reabsorption of pericardial fluid via its venous and lymphatic drainage channels to the right atrium, ultimately leading to its net accumulation. As a general consequence of such increased right atrial pressure, other signs of R-CHF like ascites, subcutaneous edema, hepatomegaly and splenomegaly can also develop (10).

Elevated pressure in the pulmonary vasculature, the hallmark of PH, can place significant strain on the right side of the heart (depending on severity and chronicity), ultimately leading to right ventricular dilation, hypertrophy, and/or R-CHF(11,12). While HCVC placement is not a direct cause of PH, several associated complications can lead to its development, necessitating a systematic investigation into the specific etiology. Therefore, using the ACVIM consensus statement for PH classification, a process of elimination was performed (11). Congenital shunts (Group 1) and PH due to left-sided heart disease (Group 2 and Goup 6 -mixed pre and post capillary) were ruled out based on Doppler echocardiography. The dog's history, with no travel to regions endemic to *Dirofilaria immitis* or *Angiostrongylus vasorum*, made parasitic disease (Group 5) improbable and persistent normoxemia made PH secondary to hypoxic pulmonary vasoconstriction (Group 3) an unlikely diagnosis. Furthermore, the complete resolution of the condition argued against irreversible causes like idiopathic or heritable PH (Group 1) and chronic obstructive airway disorders (Group 4), leaving acute massive (with R-CHF) PTE (Group 4) and parenchymal respiratory diseases (Group 3) the more likely etiologies.

Catheter-related thrombosis and pulmonary arterial embolization are frequent complications of central vein catheter placement, with an incidence of up to 40% in people (13). Advertent positioning the HCVC in the right atrium might further promote clot formation. Its insertion can trigger endothelial injury, blood stasis, and alterations in blood components, meeting Virchow's triad components, and leading to thrombus formation that, if dislodged, could trigger pulmonary arterial thromboembolism with or without more complications (including acute cardiac arrest due to a drop fall in cardiac output), depending on how large, proximal and diffuse the embolization is (3,14-16).

Interstitial pneumonia and vasculitis secondary to Ehrlichiosis represents another alternative differential diagnosis for the presently reported dog. Pathologically, this condition is characterized by mononuclear infiltration and endothelial damage, which is believed to be driven by a systemic cytokine-mediated immune response (14). Notably, this diagnosis could account for the severe PH and R-CHF, as this has been documented in two case reports (15,16) where the signs resolved with doxycycline therapy. According to ACVIM consensus statement on PH, canine monocytic Ehrlichiosis is categorized as Group 3 (11). Upon the dogs' presentation, thoracic radiographs demonstrated a mild, diffuse interstitial pattern, which may be explained by concurrent interstitial pneumonia.

Iatrogenic Superior Vena Cava Syndrome (SVCS) has emerged as a critical diagnostic entity in human medicine, linked to the increased use of HCVC, pacemakers, and intra-cardiac implants and devices (17). Similarly, in this case, CVCS developed, evidenced by head and neck pitting edema that appeared 7 days after the right jugular HCVC insertion and worsened hours after insertion of the second HCVC in the left jugular vein. Obstruction, compression, or stenosis of major intrathoracic veins likely contributed to fluid accumulation, ultimately resulting in CVCS(18).

The potential for HCVC to cause multiple, severe cardiovascular complications has been documented in a related human case report. A woman undergoing hemodialysis developed catheter-related thrombosis that culminated in both SVCS and pulmonary embolism (19). Drawing a parallel to this, we propose that the development of both CVCS and PH in our case was also attributable to two, simultaneous complications from the HCVC: a primary obstruction causing the CVCS, and a concurrent right atrial thrombus that embolized to cause the PH. This propensity for extensive clot formation could have been further driven by a pre-existing hypercoagulable state. The pro-thrombotic state can be multifactorial, potentially caused by proteinuria secondary to renal disease or the hypercoagulable and hypofibrinolytic state associated with the dog's concurrent *Ehrlichia canis* infection(15,16,20-22). The large, indwelling HCVC also provides an ideal surface for thrombus initiation. The link between proteinuria and device-associated thrombosis is specifically supported by

a multicenter retrospective study, which found that proteinuria at the time of pacemaker implantation in dogs is associated with thrombosis at a reported incidence of 10.4% (23). Serval other case reports in dogs describe pacemaker-device-associated thrombosis and CVCS, (18,23-25) and thrombosis associated with central parenteral nutrition through a central venous catheter (26).

Volume expansion and PH are common among human patients with end-stage renal disease undergoing hemodialysis, with volume overload acting as an independent predictor of PH. Moreover, interventions that reduce excess volume led to decreased systolic pulmonary artery pressure values and a lower incidence of PH (27). The persistent overhydration in this dog might have acted as an additional significant contributor to PH and R-CHF.

With both PH and R-CHF, the right ventricle is subjected to increased wall stress, leading to structural and functional changes that might predispose to ventricular arrhythmia. Although supraventricular arrhythmia may result from catheter tip irritation of the atrial endocardium, in the present case no supraventricular arrhythmia was recorded. Such electrical disturbances, if frequent enough, might further worsen the ongoing compromise of heart function. Given the pivotal role of cardiac rhythm in preserving hemodynamic stability, the occurrence of arrhythmia could exacerbate the deterioration of the dog's AKI, as suggested by elevated creatinine concentrations coinciding with the detection of arrhythmic episodes, possibly compounding the adverse effects of PH and R-CHF.

Remarkably, the dog's renal function improved in tandem with the resolution of its cardiac pathology, marked by resolution of sinus rhythm, and the elimination of measurable tricuspid regurgitation and PH, a finding that was, in and of itself, unexpected, suggesting that a treatable pulmonary thromboembolism was an important contributing etiology (28).

## CONCLUSION

This case illustrates that severe HCVC-induced complications including CVCS, PH, R-CHF, ventricular arrhythmia, and cardiac tamponade can be reversible. The prompt resolution of the life-threatening cardiac tamponade and CVCS following catheter removal identifies this as the primary, life-saving intervention that reduced systemic venous pressure. Concurrently, medical therapy with Tadalafil (Tadalafil, Inovamed Pharme LTD., Israel), Pimobendan (Pimobendan, Vetmarket LTD., Israel), and Rivaroxaban (Rivaroxaban, BAYER LTD., Israel) likely contributed to achieving control of Ph and R-CHFTD.

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