ISSN: 2572-3286

Sheta et al. J Clin Nephrol Ren Care 2019, 5:042
DOI: 10.23937/2572-3286.1510042
Volume 5 | Issue 1
Open Access

Journal of
Clinical Nephrology and Renal Care

CASE REPORT

Hyper-Acute Presentation of Access Based Cardiac Disease

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Introduction

Fifty years after the creation of the first arteriovenous access, little is known about the deleterious cardiac effects of this pathological circuit. Understanding the pathophysiology of Access Based Cardiac Disease (ABCD) is critical, given the fact that cardiac abnormalities are the leading cause of morbidity and mortality in end stage kidney disease patients on dialysis [1,2]. However, this has been a challenge, as many of hemodialysis patients have preexisting cardiac disease, they invariably have volume overload from salt and water retention, and many heart failure symptoms can be masked by regular efficient dialysis [3]. ABCD can be hyper-acute (minutes to hours), acute (days to weeks) [4,5], or chronic (months to years) [6]. In this manuscript, authors will focus on the hyper-acute form.

Case Presentation

An 82-year-old African American female developed severe hypotension after successful creation of left upper extremity loop graft. Past medical history includes type 2 diabetes mellitus, hypertension, chronic atrial fibrillation, hypothyroidism and end stage kidney disease on hemodialysis using right IJ cuffed dialysis catheter. No history of coronary artery disease. Medications include Metoprolol Succinate 25 mg daily, Furosemide 40 mg twice daily, Levothyroxine 50 mcg daily, Warfarin 5 mg daily and Glimepiride 1 mg daily. Preoperatively, BP was 147/77 and HR of 65 bpm. Rest of the examination was essentially normal. 2D Echocardiography showed EF of 70%, RVSP of 33 mmHg, normal ventricles but severely dilated atria, and severe tricuspid and pulmonary valves regurgitation. Within thirty minutes from access creation, BP dropped to 67/35 mmHg with HR of 65 bpm. Apart from altered sensorium, examination was again normal. BP improved to 120/70 after manual occlusion of the graft. Unfortunately, BP dropped again after releasing the pressure from the graft. The surgical team decided to surgically ligate the graft. Urgent 2D echocardiography showed EF of 70%, mild left ventricular hypertrophy, normal right ventricle, severely dilated atria and mild tricuspid regurgitation. In general, the study was not different from preoperative 2D echocardiography. Patient declined stress test. She was discharged home in a stable condition.

Discussion

The main function of the *Cardiovascular system* is maintaining adequate tissue perfusion. This can be achieved by contracting the cardiac muscle around a closed chamber ejecting small amount of blood (i.e. stroke volume) several times per minute (i.e. Heart rate). So, the amount of blood pumped per minute (Cardiac output) equals SV multiplies by HR. To maintain homeostasis, cardiac output should be enough to sustain adequate tissue perfusion. This simple concept is of extreme importance.

Stroke volume and heart rate are well regulated by multiple systems. The primary mechanisms controlling stroke volume are pre-load, after-load and inotropy. Sympathetic nervous system and hormonal signals control HR. Now a second critical concept appears, adequate tissue perfusion depends not only on cardiac but also extra-cardiac factors. Variations of any of the aforementioned factors is compensated by one or more of the others to maintain adequate tissue perfusion. These regulatory mechanisms are not equal in their potency or momentum. Generally speaking, increasing

Citation: Sheta MA, Patel A, Ross JR (2019) Hyper-Acute Presentation of Access Based Cardiac Disease. J Clin Nephrol Ren Care 5:042. doi.org/10.23937/2572-3286.1510042

Accepted: February 21, 2019: **Published:** February 23, 2019

heart rate is more effective when urgent increase in tissue perfusion is needed (e.g. exercise) [7].

After arteriovenous access creation, total peripheral vascular resistance drops [8]. According to the equation (Mean arterial pressure = cardiac output × total peripheral vascular resistance) [9], cardiac output should increase to maintain stable mean arterial pressure (index of tissue perfusion). This can be achieved by increasing the stroke volume (Frank-Starling law). In cases of arteriovenous graft, the sudden drop in total peripheral vascular resistance may outweigh the increase in stroke volume and increasing heart rate is necessary to maintain tissue perfusion. This is typically followed by plasma volume expansion and therefore increase in pre-load and further increase in the stroke volume. At this particular state, heart rate declines to baseline.

In rare occasions, where the patients are not able to increase heart rate especially if associated with decreased myocardial compliance (therefore not able to increase stroke volume) sudden drop in cardiac output, mean arterial pressure and tissue perfusion will occur. This is exactly what happens in the hyper-acute form of ABCD. As seen in our patient, clinical presentation includes hypotension with preserved heart rate which happens few minutes after creating the anastomosis.

Given the severity of the presentation, the authors suggest surgical ligation of the arteriovenous graft. We did not try to re-open the access few days-weeks after patient stabilization as we thought that there is a high chance the patient will re-develop cardiac decompensation. This could be hyper-acute, acute or at least chronic.

Conclusion

Hyper-acute form of ABCD, however rare, but could be fatal complication of arteriovenous access creation. Risk factors include decreased cardiac compliance (secondary to left ventricular hypertrophy), inability of the heart to increase heart rate (negative chronotropic medications, e.g. beta blockers), and arteriovenous graft creation. Suggested treatment is permanent arteriovenous access ligation.

References

1. (1998) Cardiovascular disease - An ESRD Epidemic. Am J Kid Dis 32.
2. MacRae J, Levin A, Belenkie I (2006) The cardiovascular effects of arteriovenous fistulas in chronic kidney disease: A cause for concern? Semin Dial 19: 349-352.
3. Alkhouli M, Sandhu P, Boobes K, Hatahet K, Raza F, et al. (2015) Cardiac complications of arteriovenous fistulas in patients with end-stage renal disease. Nefrologia 35: 234-245.
4. Alkhouli M, Alasfar S, Armando Samuels L (2013) Valvular heart disease and dialysis access: A case of cardiac decompensation after fistula creation. J Vasc Access 14: 96.
5. Martínez-Gallardo R, Ferreira-Morong F, García-Pino G, Cerezo-Arias I, Hernández-Gallego R, et al. (2012) Congestive heart failure in patients with advanced chronic kidney disease: Association with pre-emptive vascular access placement. Nefrologia 32: 206-212.
6. Vajdic Trampuz B, Ponikvar R, Kandus A, Buturović-Ponikvar J (2013) Hemodialysis arteriovenous fistula-related complications and surgery in kidney graft recipients. Ther Apher Dial 17: 444-447.
7. Vincent J (2008) Understanding cardiac output. Crit Care 12: 174.
8. Guyton AC, Sagawa K (1961) Compensations of cardiac output and other circulatory functions in areflex dogs with large A-V fistulas. Am J Physiol 200: 1157-1163.
9. Guyton AC, Lindsey AW, Kaufmann BN (1955) Effect of mean circulatory filling pressure and other peripheral circulatory factors on cardiac output. Am J Physiol 180: 463-468.