Development of hyperacute high-output heart failure at access creation

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ABSTRACT

High-output heart failure can occur in patients undergoing hemodialysis via permanent access. We have described two cases of hyperacute high-output heart failure. Two patients with multiple previous failed access attempts presented for redo access. Each patient experienced high-output heart failure in the operating room during access placement. A delay in the diagnosis led to the first patient's death, although early recognition led to successful treatment of the second patient. Hyperacute heart failure during access creation is uncommon. Early recognition of this complication is important because timely intervention can be life-saving. (J Vasc Surg Cases and Innovative Techniques 2021;7:529-31.)

Keywords: Access-related complications; Access-related high-output heart failure; Hemodialysis access; High flow fistula; Hyperacute high-output heart failure

Cardiovascular disease is highly prevalent among patients with end-stage renal disease (ESRD), accounting for mortality of ≤40% in this patient population.[1-3] The physiologic changes induced by high flow through the hemodialysis access are suspected to contribute to the development of high-output heart failure in a small subset of hemodialysis patients over time.[1,3-7] However, the more acute changes of heart failure related to access have only been rarely described.[8,9] In the present report, we have described two cases of patients developing what we have termed "hyperacute" high-output heart failure in the operating room after following access creation. The living patient and the family of the deceased patient provided written informed consent for the report of the case details.

CASE REPORT

Patient 1. A 40-year-old man with ESRD secondary to type 1 diabetes and multiple previous bilateral upper extremity failed access attempts with bilateral central vein occlusion had presented for new permanent dialysis access placement. The findings from previous echocardiography and cardiac catheterization were notable for the presence of pulmonary hypertension with tricuspid regurgitation and a 70% stenosis of a small, nondominant right coronary artery without evidence of ventricular dysfunction. Creation of a left leg femoral loop graft with a 6-mm × 40-cm Vectra conduit (Bard Peripheral Vascular, Inc, Tempe, Ariz) was performed without complications. However, at completion of the procedure, the patient had developed transient bradycardia and hypotension. This had initially resolved with medical management and was attributed to protamine administration just before the event. However, the patient developed hypotension again and was admitted to the intensive care unit for ongoing pressor requirements, which persisted despite adequate resuscitation. The postoperative echocardiography findings again demonstrated severe pulmonary hypertension with normal ventricular function. The patient deteriorated further during the following days, and repeat echocardiography demonstrated new right ventricular dysfunction. The femoral graft was then ligated at the bedside, and postprocedure echocardiography was performed, which demonstrated normalization of right ventricular function. However, the delay in diagnosis led to the development of multisystem organ failure and ultimately resulted in the patient's death.

Patient 2. The second patient was a 55-year-old woman with a history of ESRD, human immunodeficiency virus, cardiac sarcoidosis, and severe pulmonary hypertension. Moderate to severe tricuspid regurgitation and moderate right ventricular hypokinesis was found on preoperative echocardiography. She had presented after failure of right arm autogenous access, the use of multiple subsequent right-sided catheters, and ligation of a left arm brachiocephalic fistula which had functioned for 2 years previously but had ultimately developed ulcerative changes related to pseudoaneurysm formation. A left arm, first-stage brachiobasilic fistula was attempted but had failed to mature adequately. Therefore, this was ligated, and a brachial loop graft was created under a regional block with a

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6-mm × 30-cm expanded polytetrafluoroethylene. During closure of the incision, the patient became acutely hypotensive with an altered mental status. Protamine had not been administered during this case. Several high doses of vasopressors were given with no hemodynamic response. The tunneling counter incision was emergently reopened, and the graft was clamped, with immediate resolution in hemodynamics and mental status. The graft was very briefly unoccluded, which again resulted in acute, profound hypotension. We found the response to be so severe we did not believe that interventions to plicate the graft would be successful or safe. Therefore, the graft was excised. The patient was monitored overnight in the intensive care unit with no further hemodynamic instability. Postoperative echocardiography demonstrated no changes in heart function, and the patient was discharged without further complications.

DISCUSSION

The development of high-output heart failure in patients with ESRD requiring hemodialysis is a multifactorial physiologic process that might be driven, in part, by the blood flow through an existing dialysis access.[1,3,5-7,10] In the days immediately after access creation, cardiovascular changes occur. These include a decrease in peripheral resistance; an increase in cardiac output; increases in cardiac contractility, heart rate, and stroke volume driven by increased sympathetic nervous system activity; changes in overall blood volume and cardiac markers; and increases in pulmonary arterial flow and pressure.[1] In the present report, we have described the development of high-output heart failure within minutes after access placement in two patients. The prompt recognition and successful management of this complication in the second patient was a direct result of our previous experience with the first patient, who had experienced a delayed diagnosis and resultant multisystem organ failure. Rare studies have reported the development of high-output heart failure in the days after access creation.[8,9] To the best of our knowledge, the present report is the first description of the development of hyperacute heart failure in the operating room at access creation.

The development and management of high-output heart failure in patients with ESRD after access creation, in general, has remained a subject of debate. Ligation of hemodialysis access in the setting of symptomatic heart failure, especially for patients who no longer require access after successful renal transplantation, is known to be safe and effective.[2,11,12] However, arteriovenous access ligation has not been associated with a reduction in all-cause mortality in patients who have undergone transplantation.[13] Additionally, pulmonary hypertension is believed to be present in 40% to 50% of patients initiating hemodialysis treatment.[10,14] This is important, because pulmonary hypertension is closely related to the development of heart failure. Pulmonary hypertension in patients with ESRD and its relationship to hemodynamic changes incurred by hemodialysis access is also unclear. Although the mechanistic changes in the systemic circulation in the presence of hemodialysis access have logically been proposed to potentially lead to pulmonary hypertension in this patient population, several studies have suggested that the development of pulmonary hypertension in patients with ESRD is related to changes in the cardiovascular system secondary to the presence of ESRD itself and not associated with the presence of a functional dialysis access.[14-17]

The etiology of the development of the hyperacute high-output heart failure in the two presented patients is unclear. A previously reported prospective study assessed various physiologic changes after access creation.[10] They demonstrated an immediate decrease in subendocardial perfusion at 30 minutes postoperatively, measured by pulse wave analysis.[10] It is possible that these perfusion changes, which lead to imbalances in the supply and demand of myocardial oxygen, played a role in the clinical decompensation of the two presented patients. In both cases, the patients had had known severe pulmonary hypertension with tricuspid regurgitation before access creation. However, as stated, the high incidence of pulmonary hypertension in the ESRD population at baseline suggests it is unlikely this was the sole driver of the unusual presentations we have described.

Without a clear etiology, providing meaningful recommendations for the prevention of this complication through pre- and/or intraoperative planning and decision making remains challenging. The patient population treated by the single surgeon involved in the two presented cases mainly comprises patients with highly complex access requirements who had been referred from outside institutions after multiple failures to achieve access. This is especially evident in the history of the first patient described in our report. In such patients with multiple failed accesses, our goal is to create a robust access that is likely to maintain patency. Therefore, we have generally used straight grafts, rather than tapered, to optimize flow through the graft when a prosthetic conduit is necessary. Both of these patients had had previous accesses without the complication of access-related high-output heart failure. Therefore, we had not anticipated this to be an issue preoperatively. However, if suspicion exists that a patient might have a high risk of high-output heart failure in the future, the use of a tapered graft would perhaps temporize the flow through the access and, potentially, prevent this acute complication when all autogenous access options have been exhausted. In the case of the second patient, we have deemed her to be catheter dependent for life, because her cardiovascular collapse was severe and profound, and she has a high risk of recurrence with subsequent permanent access attempts.

CONCLUSION

We have presented the case of two patients who had developed hemodynamically significant hyperacute high-output heart failure in the operating room during hemodialysis access creation. In both cases, ligation of the access was necessary to restore hemodynamic stability. Although the cause of these incidents is unclear, we share these two cases to bring awareness to this rare complication, because prompt recognition is important for timely intervention and can be life-saving.

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