Pigment nephropathy associated with percutaneous hemodynamic support during ventricular tachycardia ablation

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Introduction

The Impella mechanical assist devices (Abiomed Inc, Danvers, MA) are percutaneously inserted left ventricular assist devices developed to provide temporary circulatory support in patients in cardiogenic shock after myocardial infarction and during high-risk percutaneous coronary interventions.[1,2] These devices are increasingly used in the treatment of ventricular tachycardia (VT) storm and for mechanical support during VT ablation.[3–5] Induction of VT during ablation, fluid administration for catheter irrigation, and anesthesia use may lead to decompensated heart failure and worsen procedural outcomes.[6] Prophylactic Impella placement provides hemodynamic support to help complete these procedures. However, the benefit of this strategy has been difficult to prove, probably owing to a very high-risk cohort of patients.[3,4]

One of the known risks of Impella use is hemolysis, which is associated with longer indwelling times.[1,7–9] Two cases of severe hemolysis leading to acute renal failure have been reported.[10,11] Here we describe a third case of pigment-induced nephropathy resulting from an Impella CP mechanical assist device used for procedural hemodynamic support during VT ablation, which is unique as it occurred despite early withdrawal of the therapy in an effort to reduce complications.

Case report

A 33-year-old man with history of arrhythmogenic right ventricular cardiomyopathy presented with palpitations, tachycardia, and multiple shocks from his implanted dual-chamber implantable cardioverter-defibrillator. The VT had a left bundle branch block morphology with a rightward


KEY TEACHING POINTS

- Temporary mechanical assist devices are used during ventricular tachycardia ablation when rhythm induction is not tolerated and hemodynamic support is required.

- Impella device (Abiomed Inc, Danvers, MA) placement carries a risk of hemolysis and renal injury or failure. The treatment is device removal. The rate of recovery of renal function is unknown.

- These potential risks should be considered and discussed with patients, as should alternative strategies, such as substrate mapping during sinus rhythm and monitored sedation when appropriate.

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inferior axis, V_4 precordial transition, and a cycle length of 375 ms (Figure 1). Despite treatment with amiodarone and lidocaine infusions, spontaneous VT recurred with hypotension and progressive signs of volume overload. His cardiomyopathy was characterized by biventricular dysfunction with a left ventricular ejection fraction of 14% by transthoracic echocardiogram and a severely enlarged right ventricle with reduced systolic function.

He was referred for VT ablation, which was performed under general anesthesia with propofol infusion and fentanyl boluses. After induction anesthesia, phenylephrine and epinephrine infusions were required to maintain an adequate blood pressure. With double extrastimulation (S1: 500 ms, S2: 250 ms, S3: 240 ms), VT was induced with similar morphology to the clinical tachycardia, a cycle length of 410 ms, and a calculated maximum deflection index of 0.58, suggesting epicardial origin (Figure 1). The slower cycle length was likely due to use of amiodarone and lidocaine infusions. The RV endocardium was then mapped during sinus rhythm with notation of local abnormal ventricular activation (LAVA) and late potentials (LP) within the right ventricular outflow tract (RVOT) and inferolateral tricuspid valve annulus (TVA), further evaluated by pace mapping.
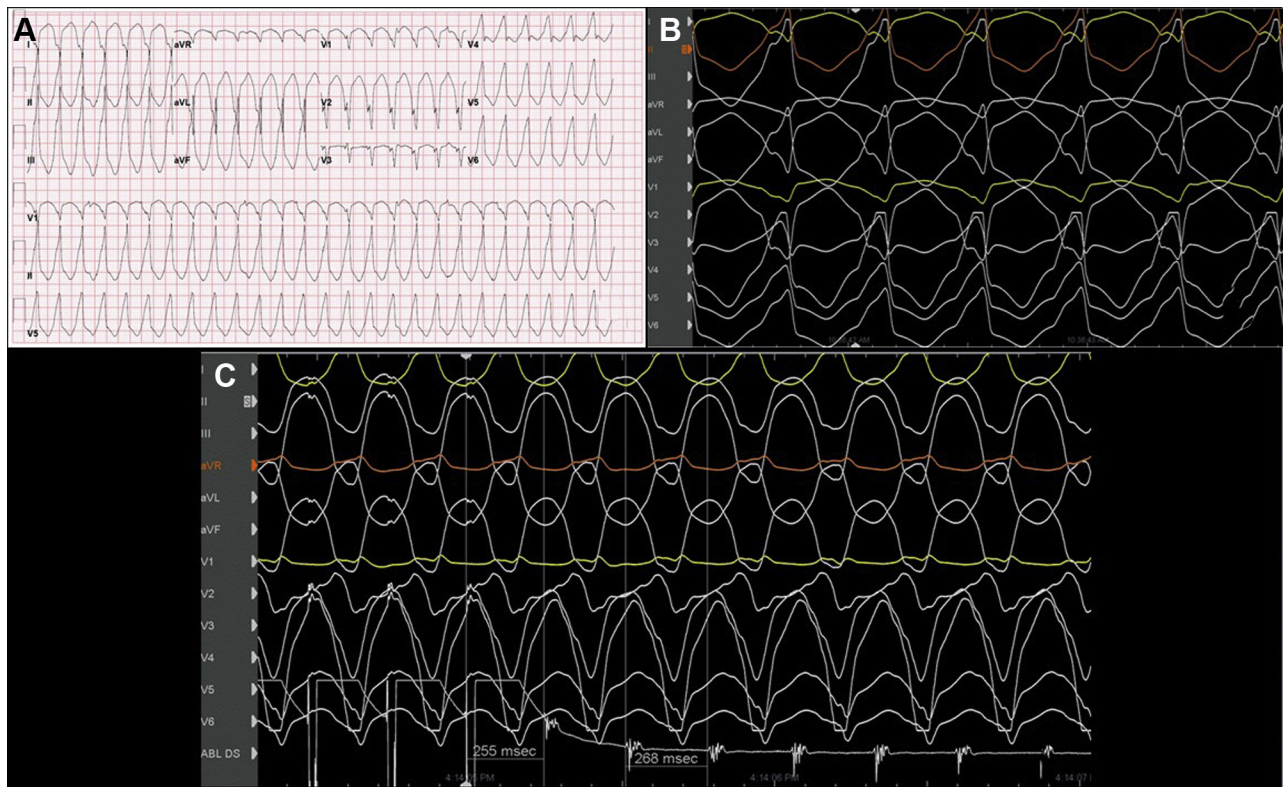
Figure 1   **A:** Electrocardiogram showing ventricular tachycardia (VT) at presentation with a rate of 158 beats per minute. **B:** VT1 observed in the laboratory with a slower cycle length of 410 ms (146 beats/min). **C:** Entrainment along the lateral tricuspid valve annulus with concealed fusion, a postpacing interval–tachycardia cycle length of 13 ms, and a stim-to-QRS/tachycardia cycle length of <30%, suggesting an exit site.

Activation mapping was also performed after induction of VT; however, the rhythm was not hemodynamically tolerated despite inotropic support, requiring pace termination.

After epicardial access was obtained, substrate mapping was performed on the epicardial surface during sinus rhythm, with LP and LAVA noted on the epicardial RVOT anterior free wall and infundibulum, extending toward the TVA (Figure 2). This was consistent with the VT morphology observed on electrocardiography as well as known arrhythmogenic right ventricular cardiomyopathy substrate. At this point, an Impella CP was placed for hemodynamic support in anticipation of mapping in VT. This was used for the remainder of the case at maximum support. A heparin bolus of 5000 units was administered after epicardial access and Impella placement with activated clotting times ranging from 170 to 190 seconds for the remainder of the case.

Repeat programmed stimulation yielded a second VT that had right bundle branch morphology, with a rightward superior axis, V_2 transition, and a cycle length of 266 ms. Activation mapping was performed during VT on both the endocardial and epicardial surfaces (Figure 2). Mid-diastolic potentials were observed on the endocardial surface along the inferolateral TVA. Entrainment was consistent with an exit site (Figure 1). Ablation at this site slowed and terminated tachycardia. No ventricular arrhythmias were induced upon repeat ventricular programmed stimulation. Further substrate modification was performed targeting LAVA and LP within the in RVOT on both endocardial and epicardial

surfaces. The Impella device was removed in the procedure room with an indwelling time of 4 hours. No alarms occurred throughout the case, nor other indication that it was not working properly. Dark-tinged urine was noted at procedure end.

Shortly after the procedure, the patient became anuric with laboratory results concerning for acute renal failure despite diuresis with intravenous loop diuretics. Serum creatinine increased from 1.0 mg/dL preprocedure to 3.8 mg/dL with a development of metabolic acidosis (pH 7.17). Acute anemia was observed with a reduction in hemoglobin from 13.5 g/dL to 11 d/dL. Hemolysis was evident with a lactate dehydrogenase peak of 2689 U/L, bilirubin peak of 5.3 g/dL, and creatinine kinase peak of 2017 U/L.

Continuous renal replacement therapy was initiated and his renal function slowly improved, as did hematologic markers of hemolysis. He was transitioned to intermittent hemodialysis, which was discontinued 2 weeks post discharge with return of renal function to near baseline. He was evaluated and listed for heart transplant 1 month post discharge because of severe heart failure. He has had no recurrent sustained VT in 6 months of follow-up.

Discussion

Left ventricular assist devices, including the Impella, have been associated with hemolysis due to continuous mechanical shear stress on red blood cells flowing through the microaxial pump. The circulating heme pigment may then cause tubular obstruction, vasoconstriction, and direct
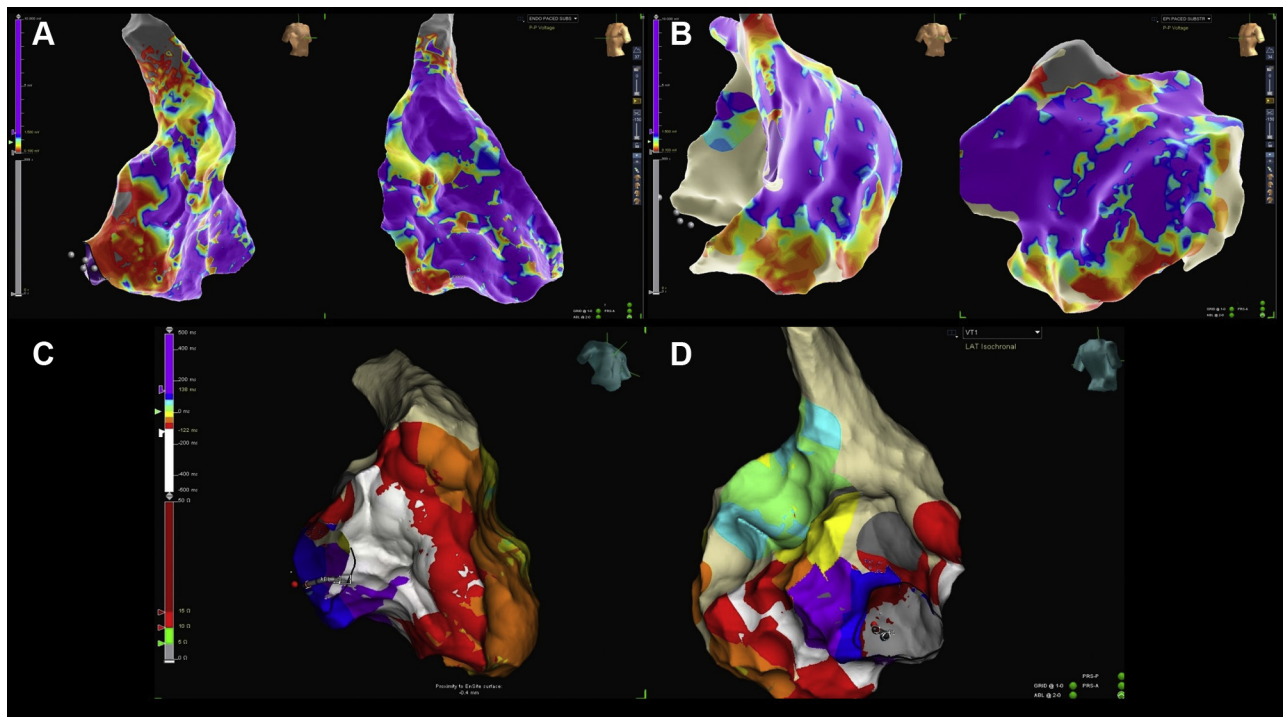
Figure 2   **A:** Endocardial voltage map. **B:** Epicardial voltage map. **C, D:** Endocardial activation map with site of termination marked by the ablation catheter location.

cytotoxicity. This pathology has been described in patients with rhabdomyolysis and hemolysis.[12] Progression of kidney injury to failure can be rapid and irreversible.

Although there is a clear associated risk of hemolysis with Impella use (7.5%–62.5%), the incidence of acute kidney injury, specifically injury caused directly by hemolysis, is unknown and presumably rare.[1,7–9] In 1 retrospective study of 69 patients, the development of hemolysis was not associated with an increased risk of renal dysfunction.[9] There have been 2 previously reported cases of pigment-induced nephropathy from Impella use. The first was a patient with advanced heart failure on home dobutamine therapy who presented with ventricular fibrillation and multiple implantable cardioverter-defibrillator shocks. VF ablation was performed with Impella 2.5 support and removed approximately 24 hours later after the development of hemolysis. Hemolysis resolved after device removal, but renal dysfunction did not improve and he required continued hemodialysis.[11] A second case described a patient who presented with ST-segment elevation myocardial infarction and acute cardiogenic shock. Oliguria with dark urine developed after Impella placement with laboratory markers of hemolysis. Hemolysis resolved after Impella removal at 12 hours, with slow recovery in renal function.[13]

Our case is unique in that we observed the development of pigment nephropathy in a short course of 4 hours. The nephropathy evolved rapidly in the immediate postprocedure period despite device removal at the end of the case. The etiology of acute renal failure is likely multifactorial, with low cardiac output and decreased renal perfusion exacerbated by vasodilation from general anesthesia. However, there was

a clear temporal relationship of hemolysis to worsening renal function. The differential diagnosis also included disseminated intravascular coagulation and heparin-induced thrombocytopenia; however, these were unlikely causes of renal injury, as the baseline thrombocytopenia was relatively unchanged during his hospital course, with no signs of bleeding or thrombosis.

The Impella device remains an important tool for VT ablation, providing hemodynamic support during the procedure and facilitating mapping of unstable VT. Our standard for hemodynamic support when needed is the Impella CP, which provides up to 4.3 L/min of augmented cardiac output, sufficient for most VT ablation procedures with relative ease of vascular access. Larger Impella devices with up to 5.5 L/min flow are available, but require larger-diameter vascular access, typically by surgical-requiring cutdown. Extracorporeal membrane oxygenation use is also reported, also requiring larger-diameter vascular access.

While being less invasive than more durable left ventricular support systems, the Impella CP device placement has risk, including pigment nephropathy. Albeit rare, this is a serious complication that may lead to end-stage renal disease, requiring long-term hemodialysis. This potential risk should be considered when deciding whether to use mechanical hemodynamic support.

Although activation mapping with entrainment remains the standard for VT ablation, the hemodynamic consequences are a common reason to use mechanical support. Our case highlights a potential advantage of substrate modification alone without VT induction. This can be performed during sinus rhythm or pacing with or without

general anesthesia. Emerging techniques including cardiac imaging and isochronal mapping have shown promise as adjunctive modalities to guide substrate-based modification.[14,15] If validated, these techniques may help further limit the need for VT induction during ablation procedures, reducing procedural risk and circumventing the need for hemodynamic support, while potentially improving procedural outcomes.

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