ATRIAL FIBRILLATION

COMPLEX CASE STUDY

Unusual Pulmonary Vein Isolation Ablation Complication Due to Contrast-Induced Nephropathy Facilitated by a Three-Dimensional Computed Tomogram

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ABSTRACT. The pulmonary vein antral circumferential ablation (PVAC) procedure is routinely utilized as an effective treatment option for patients with atrial fibrillation. Prior to the PVAC, detailed anatomical images are obtained, usually with the aid of transesophageal echocardiography and/or contrast-enhanced computed tomography or magnetic resonance imaging. We present the case of a 62-year-old male undergoing PVAC, complicated by contrast-induced nephropathy (CIN). Along with a discussion of the pathophysiology, diagnosis and treatment of CIN and risk factors associated with the development of this condition, we review strategies to avoid such a complication with future procedures.

KEYWORDS. atrial fibrillation, catheter ablation, contrast-induced nephropathy, imaging techniques prior to ablation.

Introduction

Over the last 20 years, our understanding of the pathophysiology of atrial fibrillation (AF) has led the way for ablation to be utilized as an effective treatment option. With the goal of facilitating more effective ablation through a better understanding of patient anatomy, multiple imaging modalities are employed, both before and during the ablation procedure. In the case of pulmonary vein antral circumferential ablation (PVAC) for AF, patients usually undergo transesophageal echocardiography (TEE), contrast-enhanced CT (MDCT), or magnetic resonance imaging (MRI) for establishing detailed left atrial anatomy and for left atrial appendage (LAA) thrombus identification. Specifically, MDCT has been shown to be efficacious in both excluding and detecting LAA filling defects. In the following report, we present and discuss a case of contrast-induced nephropathy (CIN) complicating a PVAC in a patient with persistent AF undergoing routine pre-procedural anatomical assessment by computed tomography (CT).

Case description

Our patient is a 62-year-old male with hypertension and type two diabetes mellitus complicated by peripheral neuropathy, who presented with severe dyspnea and palpitations, and was found to be in AF with rapid ventricular rates. He presented with symptoms of fatigue, lightheadedness, and shortness of breath, and with documented ventricular rates of up to 140 beats per minute (bpm). Cardiac evaluation was performed and was notable for a normal calcium scan, the electrocardiogram showed AF with rapid ventricular rate, and transthoracic echocardiography (TTE) demonstrated preserved systolic left ventricular (LV) function. The patient...
underwent a TEE, which excluded the presence of an LAA thrombus, and he was subsequently cardioverted to sinus rhythm. He was discharged on amiodarone 200 mg twice daily, diltiazem HCl CR 240 mg daily and rivaroxaban 20 mg daily. The patient was asymptomatic in sinus rhythm and remained very active. Four weeks later, he presented again with recurrence of his symptoms and was found to be in AF with rapid ventricular rate, despite compliance with the above medical regimen. At that point and after a comprehensive discussion regarding the ablation procedure, he decided to proceed to PVAC using radiofrequency (RF) ablation.

In accordance with our institutional protocol, the patient had pre-procedural blood work that demonstrated normal renal function, with a baseline creatinine of 0.83 mg/dl and calculated glomerular filtration rate (GFR) of 164.5 ml/min. Two days prior to the procedure, he underwent a diagnostic, volumetric, contrast-enhanced, high-resolution CT chest angiogram, receiving 100 cc of Ultravist 300 (iopromide) injected at 4 cc/s (Figure 1a). This scan was then uploaded into our three-dimensional electro-anatomic mapping system (Carto-3, Biosense Webster, Inc., Diamond Bar, CA) to improve the accuracy of RF lesion placement.

The ablation procedure was carried out in a standardized manner by performing a double transseptal puncture, in order to perform PVAC using a wide circumferential antral ablation technique, remaining safely away from the pulmonary veins. A Lasso NAV eco variable catheter (Biosense Webster) was used with Carto-3 Multi-Electrode Mapping (MEM) software to perform fast anatomical mapping (FAM) of the left atrium, the result of which was then merged with the images obtained from the CT scan. A 4-mm NaviStar RMT ThermoCool catheter (Biosense Webster), directed by remote navigation with Niobe Technology (Stereotaxis, St. Louis, MO), was used for ablation. The left pulmonary veins were mapped and extensive pulmonary vein potentials were seen. The left pulmonary veins were circumferentially isolated at the antrum, as confirmed by entrance and exit block (Figure 2). The right superior pulmonary vein was also mapped and isolated. Upon initiating ablation around the right inferior pulmonary vein, the patient was noted to be transiently hypotensive, with a systolic blood pressure of 70 mmHg (Figure 3). Intracardiac ultrasound (ICUS) using the CartoSound Module with SoundStar Catheter (Biosense Webster) demonstrated a small pericardial effusion without tamponade physiology, which was too small to explain the drop in the blood pressure. It is worth noting that the patient’s urine output during the procedure until this point was only 50 ml while he was receiving about 5 liters of fluid throughout the procedure (anesthesia-related, ThermaCool, and heparin drip). Due to the hypotensive episode and the noted oliguria, the procedure was terminated. The heparin infusion was stopped and protamine was given to reverse anticoagulation. The patient was then cardioverted at 150 joules to sinus rhythm. Of note, the effusion had not been present 1 month prior; nor had it appeared on the chest CT performed in the days prior to the PVAC (Figure 1a).

The patient was stabilized and observed in the electrophysiology laboratory for 45 minutes without further increase in the effusion. Subsequently, he was transferred to the cardiac critical care unit for further monitoring. In the unit, the patient continued to be oliguric and was noted to be anasaric. His creatinine increased, reaching a peak value of 2.08 mg/dl with a corresponding decrease in GFR to 65.6 ml/min. The presence of a new pericardial effusion was confirmed by repeat TTE. A non-contrast CT of the chest and abdomen further demonstrated evidence of fluid third-spacing within the mesentery, bilateral pleural effusions, and trace abdominal subcutaneous edema (Figure 1b, c). The renal service was consulted and urinalysis revealed muddy brown casts, low urinary sodium, and a calculated fractional excretion of sodium suggestive of acute kidney injury (AKI). Renal ultrasound showed normal-sized kidneys. His total urine output for the first 24 hours was 350 ml. The patient was deemed to have AKI secondary to CIN and with a possible small contribution from the transient hypotensive episode during

Figure 1: CT imaging prior to and after the PVAC. a) CT image with contrast prior to PVAC. The heart is borderline enlarged without pericardial or pleural effusions. b) and c) CT images without contrast after PVAC. A small pericardial effusion and moderate bilateral pleural effusions are present (b), as well as trace mesenteric and subcutaneous edema (c).
Figure 2: CARTO-3 Map showing complete circumferential lesions around the left pulmonary veins as well as the right pulmonary superior vein. Key: left superior pulmonary vein (LSPV); left inferior pulmonary vein (LIPV); right superior pulmonary vein (RSPV); right inferior pulmonary vein (RIPV).

Figure 3: Tracing of vital signs during the PVAC. Hemodynamic instability lasting 10–15 minutes (red box). Y-axis represents arbitrary units and the black marks along the x-axis represent 30-minute increments. Key: black circles: oxygen saturation (SpO2); arrow with horizontal line: end-tidal CO2 (EtCO2); diamond: heart rate; ‘X’: mean arterial pressure; vertical line: range of systolic and diastolic blood pressures.
the PVAC. He was managed conservatively with strict monitoring of urine output and avoidance of nephrotoxic agents. His volume status, urine output, and renal function recovered within 48 hours and he was discharged.

Discussion

Our patient’s PVAC was prematurely terminated due to the development of acute volume overload and fluid third-spacing. Although the temporary hypotension during the PVAC likely contributed in part to the patient’s renal injury, the time course of his oliguria within 48 hours of radiocontrast administration, as well as his history of type two diabetes mellitus, increased his risk of AKI secondary to CIN. Third-spacing occurs when excess fluid moves from the intravascular space into the interstitial or “third” space – the non-functional area between cells. This can cause potentially serious problems such as edema, reduced cardiac output, and hypotension. It is useful to review the incidence and risk factors for developing oliguric CIN, as well as to consider possible aplanation protocol revisions to minimize such complications in the future.

CIN is the acute deterioration of renal function after parenteral administration of radiocontrast media. CIN is generally defined as an increase in serum creatinine concentration of >0.5 mg/dl (≥44 μmol/l) or 25% above baseline within 48 hours after contrast administration. The incidence and risk factors associated with CIN continue to be debated. While the switch to non-ionic low-osmolar or iso-osmolar contrast agents has reportedly decreased the occurrence of CIN, some authors still suggest that CIN may occur in over 2% of the general population and in 20–30% of high-risk patients, specifically those with diabetes, congestive heart failure, chronic renal disease, and advanced age. By contrast, other meta-analyses suggest no significant difference in the occurrence of kidney injury, dialysis, and death between patients receiving contrast versus non-contrast radiographic studies.

In addition to patient-related risk factors, there have been investigations to determine whether procedural characteristics, such as the dose of contrast used or mode of administration, are related to risk of CIN development. Lower doses are associated with reduced risk of CIN. In a unique study of patients who had received both intra-arterial and intravenous contrast injections within 1 year, the risk of developing CIN was found to be similar for the two modes of media administration.

There is unclear benefit of peri-procedural hydration for the prevention of CIN. An early retrospective study by Eisenberg et al showed that hydration with 550 ml normal saline plus 250 ml heparinized saline flush per hour prevented CIN in 537 patients undergoing cerebral, abdominal, or peripheral angiography with the use of high-osmolar contrast media. However, that study lacked randomization and an appropriate control group. Interestingly, another early study on 364 patients undergoing angiography did not find a preventive effect of hydration on CIN. While several prospective, randomized trials have shown that the administration of acetylcysteine along with hydration significantly reduces CIN in high-risk patients, other trials could not show a beneficial additional effect over hydration alone. As previously mentioned, our patient received 100 cc of Ultravist 300 (iopromide), a non-ionic, low-osmolar contrast medium 2 days prior to his procedure. The specific risk factors for our patient’s developing CIN were his diabetes, and the relatively large dose of contrast that he received in a short period of time. In an attempt to prevent CIN in the future, we would recommend performing the diagnostic CT study earlier, at least 1 week prior to the planned electrophysiology procedure, as CIN usually develops 48–72 hours after the administration of contrast. This would afford sufficient time to observe the patient prior to the PVAC and address any complications related to the pre-procedural work-up. In the event that this lead time were not available, we would advocate for the use of MRI pre-procedurally, which, although more costly, decreases risk of CIN in carefully selected patients with risk factors for CIN.

References


