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CASE REPORT
Coronary heart disease

Variant angina in chronic kidney disease: a case report of an unusual presentation of cardiac arrest following dialysis

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Abstract

Variant angina (VA) is described as a relatively benign syndrome of unprovoked chest pain and electrocardiographic (ECG) changes. Chronic kidney disease (CKD) may be associated with this syndrome. However, the incidence of severe manifestations of VA in this patient cohort with kidney disease has not been elucidated. Also, no description has been made of coronary vasospam in relation to dialysis sessions. Our patient, a young female with CKD, had an unusual history of angina following dialysis sessions and she suffered an acute coronary syndrome complicated by cardiac arrest. The diagnosis was made on the basis of her clinical features, dynamic ECG changes, and coronary angiogram findings, and the patient was managed medically. Severe manifestations of VA may occur in chronic kidney disease, and this should be kept in mind by the treating physician. The association of coronary vasospasm with dialysis needs further analysis.

Keywords Variant angina • Chronic kidney disease • Cardiac arrest • Haemodialysis • Case report

Learning points

- Chronic kidney disease (CKD) is known to be associated with coronary vasospasm, which may present with severe manifestations. The association of haemodialysis with variant angina needs further analysis.
- Severe localized coronary spasm can angiographically mimic organic stenosis and should thereby be kept in mind while performing primary percutaneous coronary intervention.

Introduction

Variant angina (VA) is an uncommon syndrome that is mostly benign but carries a serious risk of myocardial infarction (MI), syncope, ventricular arrhythmias, and sudden cardiac death.[1,2] Cardiovascular complications are well known in chronic kidney disease (CKD). Coronary vasospasm has been described as a cause of chest pain in kidney disease, but details of its incidence and manifestations have not been elucidated.[3,4] Also, various haemodynamic changes occur during haemodialysis, but its acute effect on coronary circulation is not well understood.

We report a rare case of VA in a young patient with CKD who presented with MI and cardiac arrest secondary to ventricular fibrillation (VF). We also describe the therapeutic dilemma we encountered during management.

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Timeline

Since few months before presentation	Angina after dialysis
At presentation	ST Elevation Myocardial Infarction
20 minutes after admission	Cardiac arrest
Angiogram done 1hour after admission	Vasospasm of LAD with complete resolution after intracoronary NTG
Day 1- Course in ICU after angiogram	Multiple episodes of ST elevation, resolving with sublingual NTG
Day 2	Calcium channel blocker therapy
Day 3 to Day 9	No clinical event, 2 uneventful dialysis sessions. Discharged on Day 9
Follow up at 1 month	Asymptomatic

Patient information and physical examination

A 46-year-old premenopausal female, non-smoker, hypertensive with CKD on maintenance haemodialysis, with known atrial fibrillation (AF), presented to our hospital with chest pain and was diagnosed as a case of acute coronary syndrome.

The patient had this episode of severe angina for 45 minutes, following a session of haemodialysis. On enquiry, she complained of recurrent episodes of angina at rest, following her haemodialysis sessions since few months which resolved with nitroglycerine spray.

She had an irregular pulse with a rate of approximately 110/minutes, with a normal blood pressure at presentation. Cardiovascular examination showed variable first heart sound, normal second heart sound, and no additional sounds or murmurs. Respiratory examination was unremarkable with equal air entry and no foreign sounds.

Diagnostic assessment and interventions

Electrocardiogram (ECG) revealed AF with ST-segment elevation in Leads I and aVL, suggestive of high lateral wall MI (*Figure 1*). The serum troponin I level was 10 ng/mL (normal <20 ng/mL), and the 2D echo examination was normal with no wall motion abnormality. Other blood investigations were normal, except for high serum creatinine of 4.85 mg/dL (normal 0.57–1.11 mg/dL). Approximately 20 minutes after admission, patient developed an episode of VF leading to cardiac arrest and was successfully resuscitated with a single DC shock of 200 J, a single intravenous bolus of 1 mg adrenaline, and

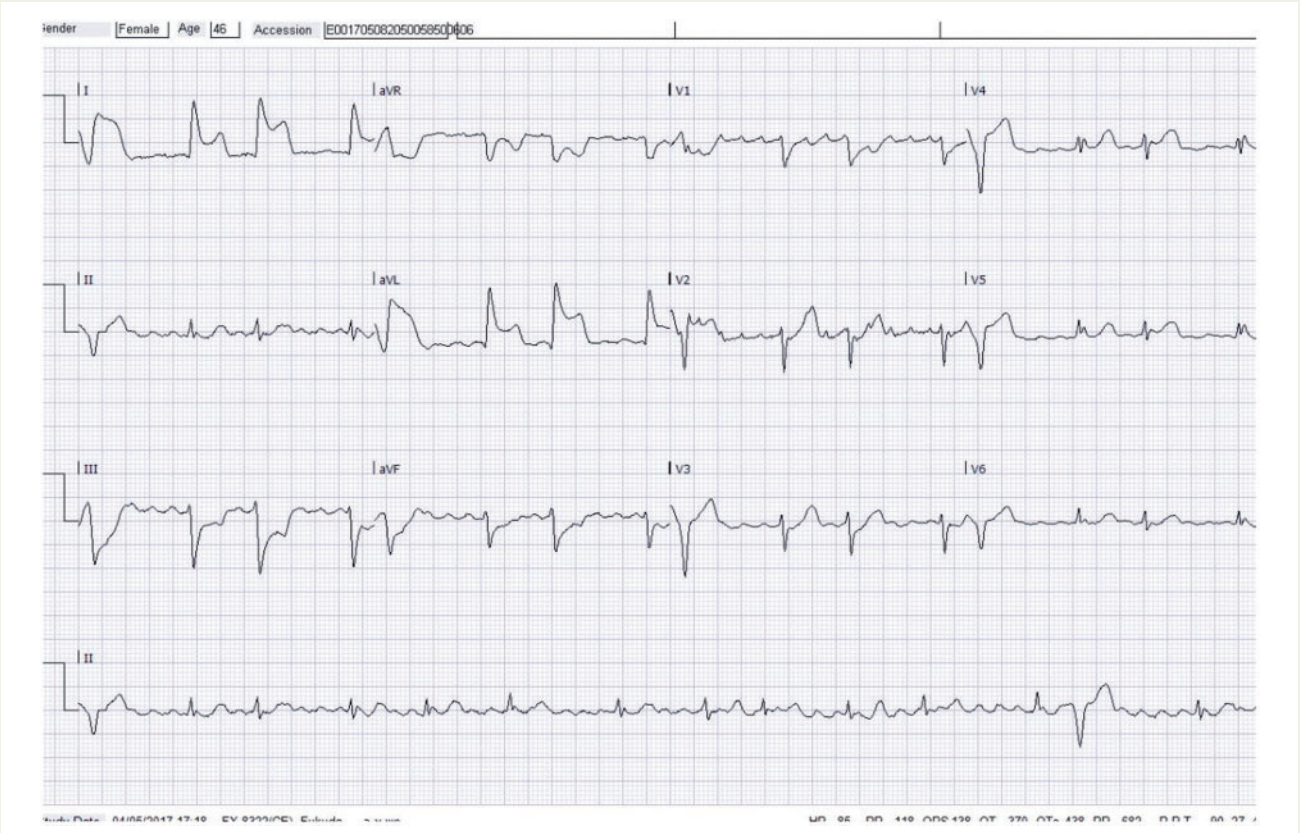

Figure 1 Baseline admission electrocardiogram recording showing ST-elevation in Leads I and aVL with an underlying rhythm of atrial fibrillation.

five cycles of cardiopulmonary resuscitation (CPR). Following the cardiac arrest, the patient was shifted for an emergency percutaneous coronary intervention (PCI) on inotropic support with dopamine injection.

Coronary angiogram revealed >90% stenosis in mid-left anterior descending artery (LAD) and non-significant disease in the left circumflex artery and the right coronary artery (*Figure 2*).

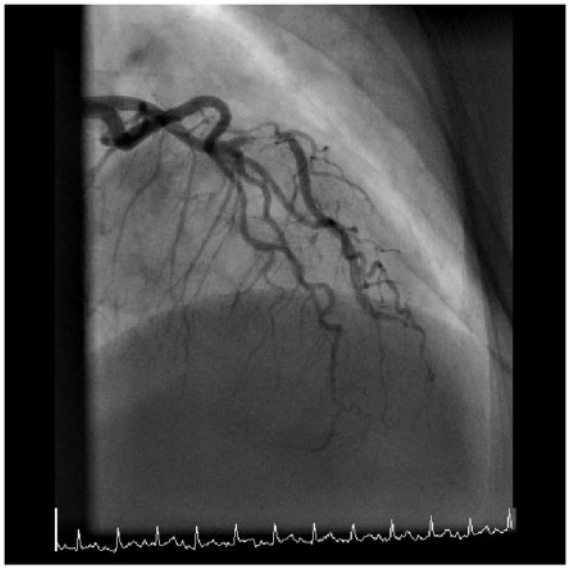

Figure 2 Baseline angiography showing no atherosclerosis and severe narrowing of the mid-left anterior descending artery.

As the patient was a premenopausal female with a history of non-exertional angina resolving with nitroglycerine and had no obvious thrombus or dissection in the culprit artery, vasospasm of the LAD was suspected to be the likely aetiology of her presentation. Although an ergonovine provocative test would have been ideal for diagnosis, we refrained from performing it as the patient had been resuscitated minutes ago. Instead, 200 µg of nitroglycerine was injected into the LAD, and patient's blood pressure paradoxically increased with complete resolution of ST elevation (*Figure 3*) and relief in chest pain.

Final angiogram revealed a patent LAD with improved calibre throughout its length (*Figure 4*), indicating focal and diffuse vasospasm in the LAD as aetiology for her complaints.

The patient's next 24 h in the intensive care unit were also dramatic with multiple episodes of similar ST elevation in Leads I, aVL, V4, and V5 (*Figure 5*), accompanied by chest pain which resolved with sublingual nitroglycerine. Ventricular arrhythmia did not recur with any of these events. These remarkable ECG changes further helped in confirming the diagnosis of VA.

High dose calcium channel blocker (CCB) therapy with oral verapamil (240 mg/day) was initiated the following day after stabilization of blood pressure. The patient had no further chest pain episode or any new ECG changes. She was monitored for another 7 days during which she underwent two haemodialysis sessions that were uneventful. The patient was discharged on CCB therapy and advised close follow-up.

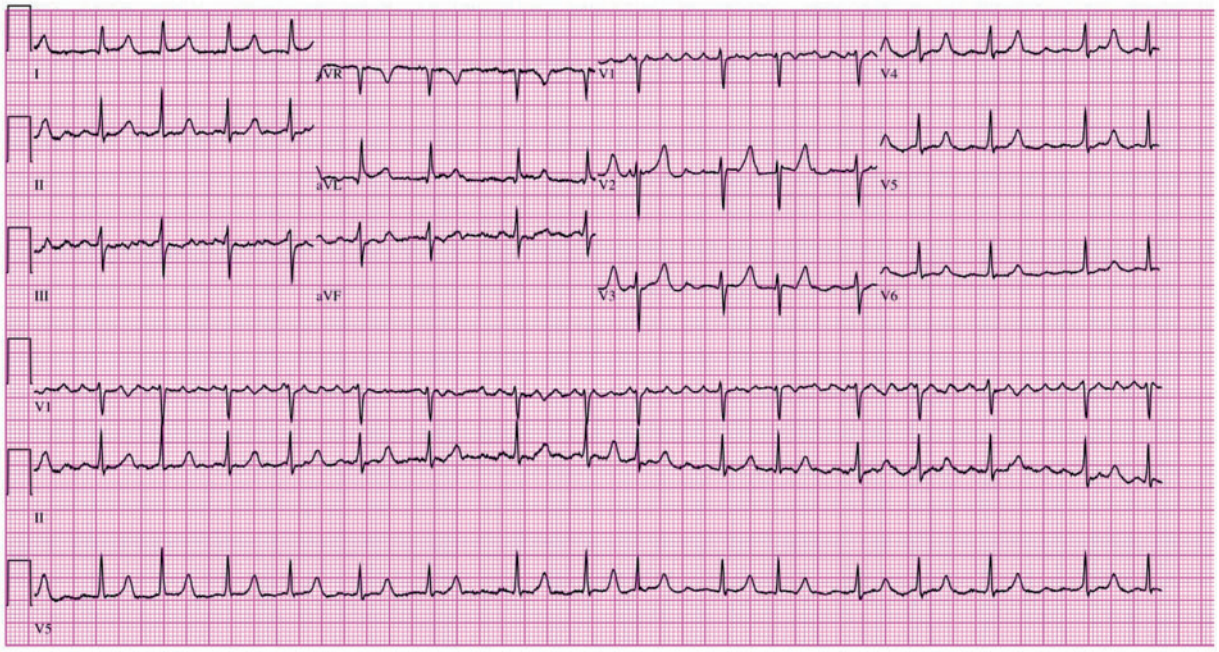

Figure 3 Electrocardiogram of complete ST resolution obtained after patient was shifted back to the intensive care unit from the catheter lab.

Follow-up and outcomes

On follow-up at 1 month, the patient was asymptomatic, tolerating her medications well and was compliant to therapy. Following discharge, she underwent eight haemodialysis sessions without any clinical event. Baseline ECG showed AF and no ST-T changes.

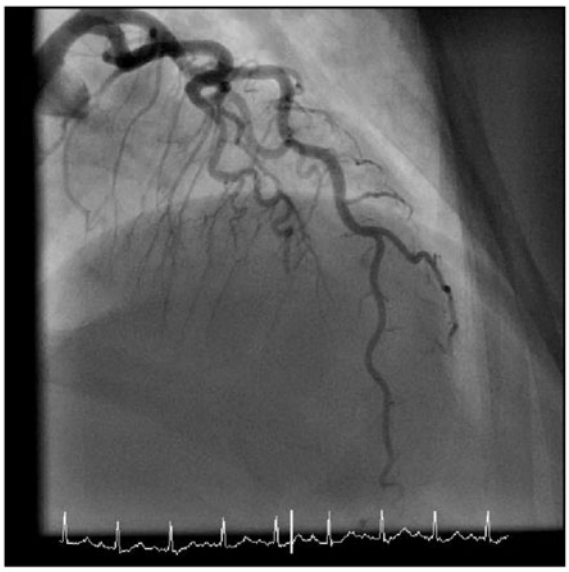

Figure 4 Angiogram showing normal calibre of the affected left anterior descending artery throughout its length.

Discussion

To our knowledge, this is the first reported case of a patient with CKD having VA with complication of VF leading to cardiac arrest. The association of angina following haemodialysis presumably due to coronary vasospasm has also not been described.

Pathophysiology and association with chronic kidney disease

The association of coronary vasospasm with renal impairment has been shown to be high in certain study populations.[3,4] Endothelial dysfunction[3] and chronic inflammation[4] are probable factors that play a role in the pathogenesis of VA in these patients. An association of VA with elevated levels of C-reactive protein (CRP) in patients with CKD has been shown.[4] Our patient had very high-sensitivity CRP levels of 15.1 mg/dL (normal <0.5 mg/dL). Few uraemic toxins are also associated with endothelial dysfunction and immune dysregulation.[5]

Although peripheral vasoconstriction is known following haemodialysis,[6] no description could be found associating coronary vasospasm to dialysis. Low levels of nitric oxide end products and elevated endothelin-1 levels have been isolated in patients who develop hypertension during and after dialysis.[7] These mechanisms might have played a role in the pathophysiology of coronary vasospasm in our patient.

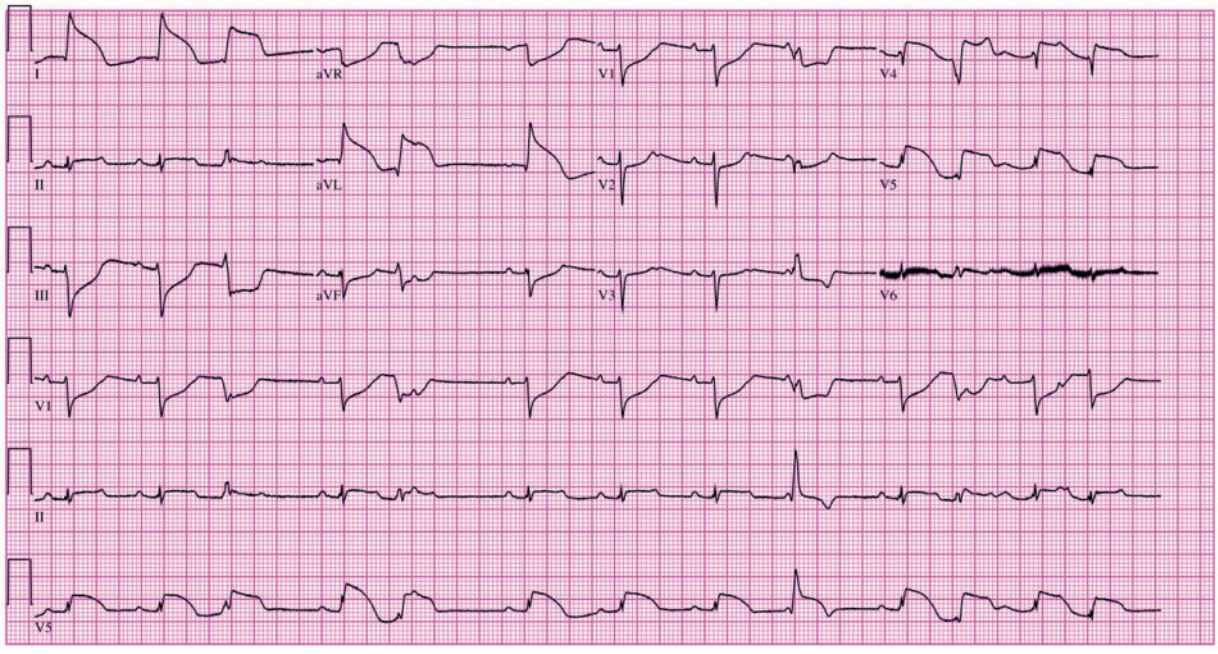

Figure 5 Electrocardiogram showing re-elevation of ST-segments in Leads I, aVL, V4, and V5 during the patient's intensive care unit stay.

Severe manifestations of variant angina

Lethal arrhythmias and high-degree atrioventricular block are known in VA.[1,2,8] Coronary artery spasm-induced cardiac death has been described in patients.[8] Our patient had an episode of VF leading to cardiac arrest.

Therapeutic dilemma

Management of cardiac arrest: Some case reports have described the use of nitrates during VA-induced cardiac arrest with successful outcome by avoiding adrenaline.[9,10] In our patient, we, however, gave adrenaline during CPR and did not find it detrimental.

Role of percutaneous coronary interventions: Percutaneous coronary intervention has been done in patients of VA with organic obstructive coronary artery stenosis[11] and even in patients with normal coronaries who were resistant to medical therapy.[12] However, larger studies are needed before recommending PCI as a treatment for VA.

Role of implantable cardioverter defibrillators: Variant angina and lethal arrhythmias have been reported in patients with silent ST-segment elevation.[13] Matsue et al.[14] recommended implanting ICD in every VA patient with lethal arrhythmia, irrespective of response to medical therapy. However, general consensus is that patients of VA with recurrent ventricular tachyarrhythmias, despite optimal medical therapy, should be considered for implantable cardioverter defibrillators (ICDs).[15] Hence, we decided not to implant an ICD in our patient.

Conclusion

(1) Variant angina (VA) can present with severe manifestations like myocardial infarction, ventricular arrhythmias, and even cardiac arrest.
(2) Chronic kidney disease may be associated with coronary vasospasm. The association of haemodialysis with VA needs further analysis.
(3) Severe localized coronary spasm can angiographically mimic organic stenosis and should thereby be kept in mind while performing primary percutaneous coronary intervention.
(4) Definite scientific evidence is lacking for several treatment issues of VA. Hence, management of every case should be individualized.

Patient perspective

Patient was satisfied with her treatment and her only concern was regarding the need for an ICD, for which she has been advised follow-up at present.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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