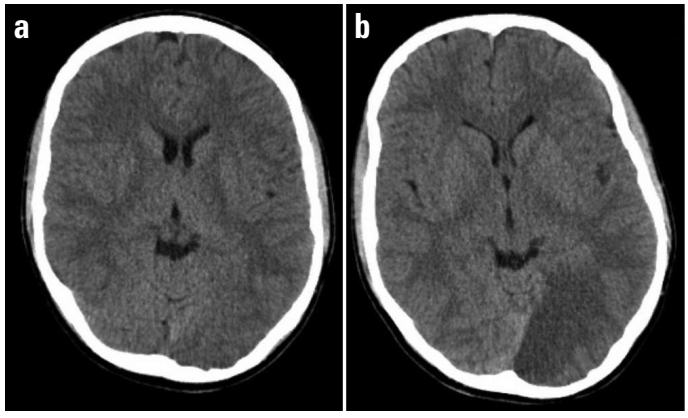

Figure 3. (a, b) Initial and control cranial MDCT

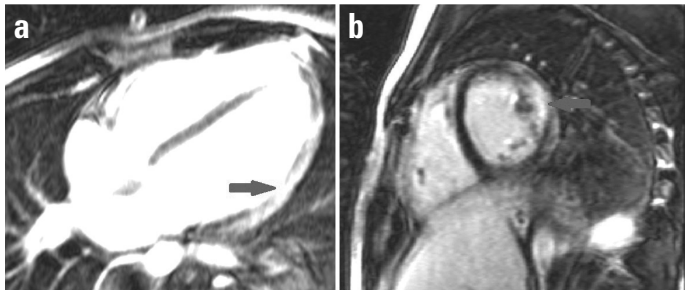

Figure 4. (a, b) Cardiac MRIs showing late gadolinium enhancement (LGE) in posterolateral portion of the heart (arrows), which is consistent with an old myocardial infarction

with little or no increase in blood pressure (2). There are different reports on the type of AMI associated with marijuana, including atherosclerotic coronary artery disease, coronary artery thrombosis, and coronary vasospasm (3, 4). Considering vasospasm, the present patient was administered diltiazem during hospitalization. Usually, cardiovascular and cerebrovascular ischemic mechanisms associated with marijuana are similar. Intense postural hypotension, cardio-embolism, vasospasm, and increased catecholamines and carboxyhemoglobin levels might be the responsible mechanisms. Paroxysmal atrial fibrillation (PAF) is accepted as the main reason for cardio-embolism (5–7). Albeit the sinus rhythm observed in the patient's admission ECG, SEC in the left chambers suggested a history of PAF. Resolution of symptoms with anticoagulants and disappearance of SEC indicates cardio-embolism as the responsible mechanism. Furthermore, in children and adolescents, following marijuana consumption, posterior cerebrovascular circulations have been reported as the regions of predilection (8). Our patient's stroke-related area was the left occipital lobe, which confirms the predilection.

Conclusion

Marijuana is a novel illicit drug used worldwide. There have been some reports about the side effects of marijuana on the cardiovascular and cerebrovascular systems. To the best of our knowledge and according to the literature, our patient was the youngest to present with stroke and myocardial infarction.

Video 1. TTE showing inferior and posterolateral wall hypokinesis with an EF of 40%.

Video 2. TTE showing +2 SEC in left chambers.

Video 3. TTE showing moderate mitral regurgitation.

Video 4. Control TTE showing normal left ventricular wall motion.

Video 5. Control TTE showing mild mitral regurgitation.

Video 6. Cardiac MRI showing mild posterolateral hypokinesia.

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Severe hypocalcemia and hypercalciuria due to contrast medium in the course of acute myocardial infarction

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Introduction

Contrast media-related nephropathy is one of the possible complications in myocardial infarction patients following primary percutaneous intervention (PCI). Contrast media-related nephropathy is mainly defined as a decrease in creatinine clearance and an increase in serum creatinine levels; however, contrast media may also cause electrolyte imbalances. Here we present a case report of severe electrolyte deficiency related with contrast media administration.

Case Report

A 49-year-old male was admitted to the emergency department with the complaint of squeezing chest pain for 30 minutes. On 12-lead electrocardiography (ECG), anterior myocardial infarction was diagnosed, and we performed primary PCI for the left anterior descending (LAD) coronary artery. Successful reperfusion was achieved according to angiographical, clinical, ECG, and laboratory parameters. We used approximately 100 mL ioversol, a low-osmolar contrast medium. During the follow-up period, at 36th hour after the primary PCI, ventricular fibrillation occurred, and after a successful resuscitation, heart rhythm was stabilized without any neurological deficit. On 12-lead ECG, QTc interval was shown to be slightly prolonged (550 msn), and there was no new ST-segment elevation. Control coronary angiography was performed to rule out possible stent thrombosis, but LAD stent was open with a TIMI III distal flow. We detected a severe electrolyte imbalance with deep hypocalcemia (serum ionized Ca^{++} was 2.5 mg/dL, reference level 4.5–5.3 mg/dL). Serum parathormone (PTH) level was 588 pg/mL (reference level 15–68 pg/mL). Blood urea nitrogen level was 24 mg/dL, and serum creatinine was 0.9 mg/dL. We collected a 24-h urine sample to determine the cause of hypocalcemia and detected an increased Ca^{++} output (347 mg/day, reference level 100–300 mg/day). Twenty-four hour urine volume was 3100 mL, and urine creatinine clearance was 122 mL/min. We did not detect any other serum or urine electrolyte deficiency. We also assessed serum vitamin D and albumin levels, and they were within normal reference limits. We initiated Ca^{++} replacement and calcitriol therapy for 3 weeks, and serum Ca^{++}/PTH measurement and 24-h urine collection was renewed. Blood urea nitrogen level was 22 mg/dL, and serum creatinine was 0.8 mg/dL. Serum ionized Ca^{++} was within normal reference limits (4.6 mg/dL), and serum PTH level was substantially decreased (82 pg/mL). We detected urine Ca^{++} level to be within normal reference limits (192 mg/dL). Twenty-four hour urine volume was 3200 mL, and urine creatinine clearance was 137 mL/min. QTc interval was 435 msn on 12-lead ECG.

Discussion

Primary PCI is the preferred reperfusion treatment in acute myocardial infarction (1). Large amounts of contrast media can be used during primary PCI. Contrast media have nephrotoxic effects via direct cellular toxicity. Administration of various contrast media may cause contrast media-related nephropathy in the presence of underlying risk factors (2). Contrast media-related nephropathy is generally defined as an increase in serum creatinine levels and a decrease in urine creatinine clearance (3). However, contrast media may have different nephrotoxic effects other than affecting the urine creatinine clearance.

Calcium (Ca^{++}) is a multivalent cation, which plays an important role in cellular functions. Ca^{++} metabolism is greatly regulated by renal tubular cells. Proximal convoluted tubule of nephron is the main site of Ca^{++} reabsorption. Ca^{++} reabsorption is mainly passive and occurs along with sodium (Na^{+}) and water reabsorption; however, approximately 15% of Ca^{++} reabsorption is under the active control of PTH and calcitonin. As a response to disturbances in serum or urine Ca^{++} levels, PTH levels may change rapidly for the hormonal control of Ca^{++} reabsorption (4).

In various clinical studies, several contrast media were compared for their nephrotoxic effects. In these studies, renal tubular cell death was shown to be related with contrast media administration. Ludwig et al. (5) stated that low-osmolar contrast medium is more nephrotoxic than iso-osmolar contrast medium. Contrast media-related nephropathy is generally expressed as serum creatinine levels and urine creatinine clearance, but contrast media can have different renal side effects other than these two. Serum electrolyte metabolism is closely related with renal tubular cells, and contrast media may have some toxic effects on the renal tubular cells via cellular apoptosis (6).

In the present case, severe hypocalcemia was closely related with increased urine Ca^{++} output. Increase in urine Ca^{++} output was possibly due to toxic effects of contrast media on the renal tubular cells. We assumed that the first serum PTH level increase was a reactive response to hypocalcemia. As serum Ca^{++} levels returned to normal, the transient serum PTH increase reversed.

Conclusion

In our patient, severe hypocalcemia, apparent as low serum ionized Ca^{++}, was related with increased urine Ca^{++} excretion. This was proved by 24-h urine collection. We also detected an increase in serum PTH levels as a response to hypocalcemia. We believe that reversible hypocalcemia and increased PTH levels were related with toxic effects of low-osmolar contrast medium, ioversol, possibly on the proximal convoluted tubule of nephron.

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Coronary-subclavian steal syndrome in a hemodialysis patient with ipsilateral subclavian artery occlusion and contralateral vertebral artery stenosis "Case Report"

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Introduction

Subclavian artery (SCA) stenosis proximal to the internal mammary artery (IMA) may result in a condition termed as "coronary-subclavian steal syndrome," in which the left IMA (LIMA) bypasses the left anterior descending artery (LAD) (1). We present the case of a patient having end-stage renal failure (ESRF), with a history of coronary artery bypass graft (CABG) surgery, who developed angina during hemodialysis because of an arteriovenous fistula (AVF) in his left forearm. Clinical signs, physical examination findings, and recovery of symptoms after intervention are described.

Case Report

A 62-year-old man with ESRF, with a history of CABG surgery of a LIMA–LAD bypass, was referred to our cardiology department with the complaint of retrosternal angina happening in the course of each hemodialysis session for last 2 months. The patient had undergone bioprosthetic aortic valve replacement for severe degenerative aortic stenosis and single-vessel CABG surgery with a LIMA graft to LAD for 70% stenosis at the ostium of LAD 4 years ago. On physical examination, a low-flow thrill was palpated on his left forearm as a sign of AVF, and a difference of at least 70 mm Hg between systolic blood pressures of

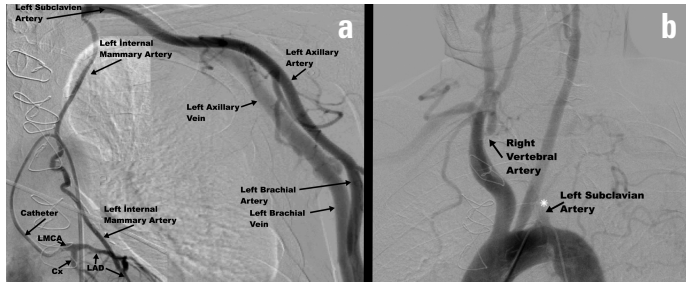

Figure 1. Left coronary angiography revealed the flow in the left brachial and subclavian vein via arteriovenous fistula in the forearm from the LIMA reversed flow (a). An aortography revealed a total occlusion in the left subclavian artery (asterisk) and severe stenosis on the ostium of the right vertebral artery (b)

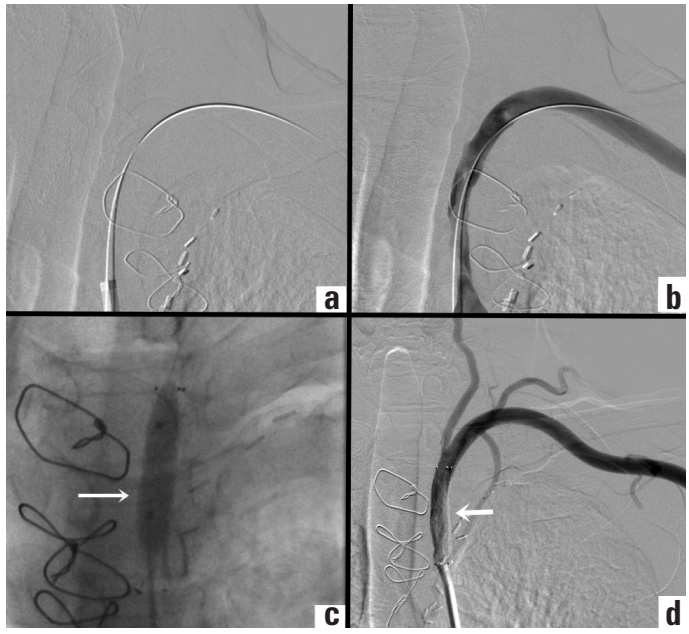

Figure 2. Totally occluded segment of the left subclavian artery was passed with the help of a guide-wire (a), and percutaneous treatment was performed (b, c). Angiography of SCA showed antegrade flow in LİMA after successful subclavian artery stenting (d)

the right and left upper extremities was observed (154/78 mm Hg and 84/55 mm Hg, respectively). Coronary angiography revealed moderate stenosis of LAD and no significant stenosis of the left circumflex or right coronary arteries. Left coronary angiography revealed a reverse flow in LIMA and a flow in the left SCA and brachial artery from LIMA (Fig. 1a). Aortography showed total occlusion in the left SCA and severe stenosis of the ostium of the right vertebral artery (VA) (Fig. 1b). For this reason, the patient underwent right VA and left SCA revascularization. The lesion at the ostium of the right VA was passed with a guide-wire (Guidewire®; Montmorency, France), and a 5x15 mm Powerflex balloon (PTA Dilatation Catheter®; Cordis Corporation, California, US) was used to dilate the stenotic segment before stent placement. Then a 5x15 mm Herculink stent (Vascular Stent®; Abbott Vascular, Diegem, Belgium) was placed, and reconstruction of VA was provided. Totally occluded segment of the left SCA was passed with a guide-wire and predilated with the help of a 7x20 mm