

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 ms), 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 ms 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 neph-

ron 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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DOI:10.14744/AnatolJCardiol.2016.7130

