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Contrast Induced Nephropathy can be Surpassed with Meticulous Attention Even in Patients with Severe Renal Dysfunction

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ABSTRACT

Contrast induced nephropathy (CIN) is one of the most important complications of the percutaneous coronary intervention (PCI) and may lead to dialysis especially in the presence of baseline high creatinine level. Here we describe coronary angiography and subsequent successful PCI of proximal left anterior descending (LAD) artery lesion without developing CIN in patient who has medically refractory angina pectoris and baseline very high creatinine level (6.3 mg/dl). This case report illustrates the potential role of invasive approach in patients with coronary artery disease even in the presence of severe renal dysfunction by using preventive measures and close monitoring.

Key words: Dialysis, contrast media; creatinine; nephropathy; percutaneous coronary intervention

KONTRAST NEFROPATİSİ CİDDİ BÖBREK YETERSİZLİĞİ OLAN BİR HASTADA BİLE DİKKATLİ BİR YAKLAŞIM İLE AŞILABİLİR

ÖZET

Kontrast nefropatisi perkütan koroner girişim (PKG) sırasında meydana gelebilecek en önemli komplikasyonlardan biri olup özellikle yüksek kreatinin seviyeleri varlığında dializ gereksinimine yol açabilir. Burada medikal tedaviye dirençli angina pektoris yakınması olan ve çok yüksek kreatinin seviyesi (6.3 mg/dl) olan bir hastaya koroner anjiografi ve sonrasında sol ön inen artere başarılı PKG yapılan bir hasta sunulmaktadır. Bu vaka koroner arter hastalığı olan bir hastada, ciddi renal yetersizlik varlığında dahi önleyici tedbirler ve yakın takip ile invaziv yaklaşımın potansiyel rolünü ortaya koymaktadır.

Anahtar sözcükler: Dializ; kontrast madde; kreatinin; nefropati; perkütan koroner girişim

Chronic renal disease (CRD) patients have often accompanying cardiovascular disease and therefore are referred frequently for angiography. However, CRD patients are often denied access to these potentially life-saving contrast studies for fear of worsening their nephrologic outcomes. To our knowledge this case has the highest baseline creatinine level in the literature, while undergoing coronary angiographic intervention without developing undergoing CIN and the requirement of dialysis.

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Case

A 63 year-old man with a history of diabetes mellitus, hypertension, CRD, and coronary artery disease presented to hospital with increased frequency and severity of angina pectoris (class III-IV according to the functional classification of the New York Heart Association) despite optimal medical therapy. CRD was secondary to long standing uncontrolled diabetes mellitus. The patient was considered as candidate for kidney transplantation and arterio-venous fistula was also prepared. He was on asetilsalisilic asit, statin, insulin, amlodipin, diltiazem and nitrate therapy. Although for two years his physical condition was good, with this treatment, in one month his complaints not only resurfaced but were also more severe. Physical

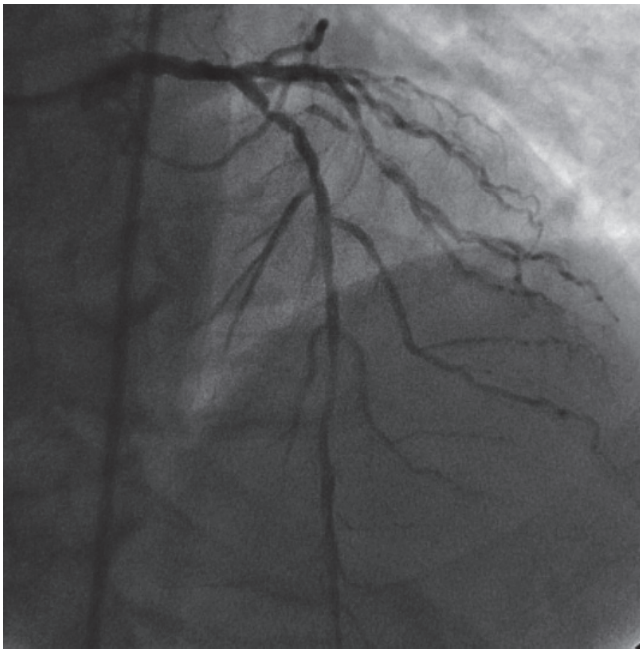

Figure 1. Critical proximal LAD stenosis

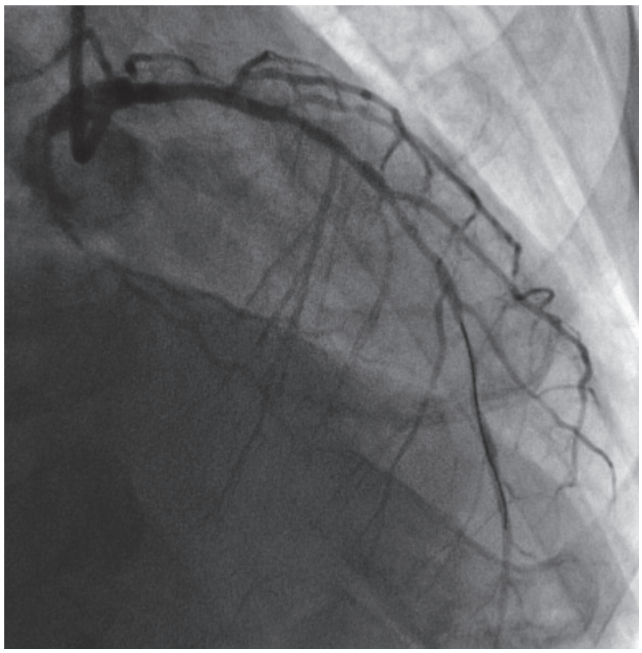

Figure 2. After successful percutaneous coronary intervention of proximal LAD stenosis

examination revealed a blood pressure of 110/80 mmHg, a pulse of 62 beats/min, respiration of 22 breaths/min. The cardiac sounds were normal and his lungs were clear on auscultation. Laboratory examination revealed high blood urea nitrogen (BUN) (89 mg/dl, normal range <20 mg/dl) and serum creatinine (6.3 mg/dl, normal range <1.3 mg/dl) levels. His glomerular filtration rate (GFR) was 11 ml/min according to the Cockcroft–Gault Formula. Other biochemical tests and complete blood count were all within the normal range. Electrocardiography revealed sinus rhythm with nonspecific ST-T changes. On transthoracic echocardiography, left ventricular function was normal and there were no segmental wall motion abnormalities or valvular dysfunctions.

According to this finding, we discussed possible alternatives and consequences with the patient and gave him detailed information. Finally, we decided to perform coronary angiography. He was hospitalized before the day of the procedure and started IV hydration with isotonic solution (1.5 ml/kg/h) and oral N-acetylcysteine (NAC) (600 mg, twice a day). Coronary angiography was performed using 40 cc non-ionic, low osmolar contrast media (CM) without performing ventriculography. After the procedure IV solution and oral NAC were continued, 24 and 48 hours respectively. Urine output and clinical course were followed closely. Control BUN and serum creatinine measurements were evaluated at 48 hours after the procedure and no increase was observed. Angiography showed diffuse three vessel disease but critical proximal LAD stenosis (Figure 1). We explained to the patient possible choices and risks. Because he preferred PCI we decided to perform PCI for proximal LAD lesion. Fifteen days after the coronary angiography, he was hospitalized and were checked BUN and serum creatinine levels. They were 80 mg/dl and 5.5 mg/dl, respectively. The same medical procedure was applied in order to prevent CIN. Intervention of the LAD proximal lesion was performed successfully with drug-eluting stent, using only 25 cc contrast media (Figure 2). Control values acquired again at 48 hours after the procedure and they were also less than the pre-procedure levels (BUN: 91 mg/dl and creatinine: 5.9 mg/dl). The patient was discharged with previous medical therapy and he had only class I-II anginal symptoms.

Discussion

CIN is a common complication of PCI.[1] Although there is no widely accepted definition for CIN, it is most commonly defined as an absolute (≥0.5 mg/dl) or relative (≥25%) increase in serum creatinine with respect to baseline within 48 hours contrast media administration in the absence of an alternate etiology.[2] Several predisposing risk factors for CIN have been identified, which include baseline renal impairment, diabetes mellitus, congestive heart failure, intravascular volume depletion, and the use of a large volume of contrast agent.[3] The incidence of CIN is low in

patients without risk factors, but is increased among patients with CRD, particularly those who also have diabetes mellitus (1,4). Currently, the most important and recognized risk factor for development of CIN is baseline renal impairment (5) and this is conveniently defined as serum creatinine level of ≥1.5 mg/dl (6).

CIN increases hospitalization period and in-hospital morbidity and mortality in the medium and long run. A large prospective study revealed a 0.44% incidence of CIN that required dialysis. The in-hospital mortality rate in this group of patients was 39% but only 1.4% of the subjects that did not present this complication died (7).

There is no agreed threshold change in renal function and the CM may not be the sole, but rather a contributory factor to the decline in renal function in a given patient. It may be more useful to think of CM as compounds with some nephrotoxic potential that becomes clinically important when combined with a suitable substrate (e.g., diabetic nephropathy), nevertheless contrast induced renal failure may not be reversible (8).

Many studies demonstrated that CIN can be prevented with some precautions and medications.

A meta-analysis showed that out of the 31 randomized studies, 22 favored the low osmolar CM (9) but the authors observed a statistically significant reduction in CIN incidence when low osmolar CM was administered only when serum creatinine level was above 1.35 mg/dl or when GFR was lower than 70 ml/min before CM was administered. According to this result, it is reasonable that low osmolar CM should be used in the presence of CRD or CRD plus DM. It can be estimated that if the amount of CM has increased, the CIN risk would also have increased. In light of these findings, the lowest possible volume of CM is recommended, as well as the ruling out of routine ventriculography in high-risk patients. Another known risk factor for CIN is dehydration (10). While no randomized controlled trial has studied the benefits of hydration alone, it seems plausible that adequate hydration may counteract some of the putative hemodynamic effects that may lead to CIN. Owing to antioxidant and vasodilatation effects, there have been many studies conducted considering the possible potential of NAC to prevent CIN. Despite the controversies regarding the efficacy of NAC, it is suggested to apply this prophylaxis, which is at least known to be inexpensive and harmless.

It is well known that patients with lower estimated GFRs at baseline are at the greatest risk for a significant loss in kidney function, or even dialysis after a contrast load. For this reason potentially life-saving procedures such as angiography are sometimes withheld or delayed. To our knowledge this case has the highest baseline creatinine level in the literature, while undergoing coronary angiography and PCI without developing CIN and requirement of dialysis. Common coincidence of chronic renal failure and coronary artery disease can lead to this dilemma more frequently than we think. We suggest that patients who present severe baseline renal dysfunction and severe angina pectoris can still be candidates for invasive approach without inducing CIN by using preventive measures and close monitoring.

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