Urgent Coronary Revascularization In A Familial Polycystic Kidney Disease Patient With Unstable Angina Pectoris
C Bolcal, B Sava?, E Ozul, H Tatar

Citation

Abstract
Familial polycystic kidney disease is a rare cause of renal insufficiency and it could be mentioned together with gastrointestinal and cardiovascular diseases such as aneurysms and diverticulosis. A congenital familial polycystic kidney disease patient with unstable angina pectoris underwent urgent coronary revascularisation operation without respecting the high levels of serum urea (125 mg/dL) and creatinine (4.8 mg/dL). We administered ultrafiltration during the cardiopulmonary bypass period and then we continued renal supplement therapy via veno-venous hemofiltration. The patient's postoperative period was uneventful and his blood urea nitrogen and creatinine levels decreased to normal levels after this therapy. We conclude that urgent coronary revascularization operations can be done safely in familial polycystic kidney disease patients with unstable angina pectoris by the using of proper renal supplement therapy methods.

INTRODUCTION
There is a clear relationship between Familial Polycystic Kidney Disease (FPKD) and gastrointestinal and cardiovascular diseases. Coronary and other arteries’ aneurysm, hepatic cysts, small intracranial cysts, aortic aneurysm, annuloaortic ectasy and gastrointestinal diverticulosis can be mentioned among these diseases (1). Therefore, it is possible to add the FPKD to the etiology list of coronary artery disease (CAD). It is also well-known that some degrees of renal dysfunction can be seen after open heart surgery (2). Acute renal failure is one of the most serious complications that can even cause mortality among these renal dysfunctions. We report a patient with familial polycystic kidney disease who had undertaken urgent revascularization operation for unstable angina pectoris.

CASE REPORT
A 64 years old, male patient with FPKD admitted to our Cardiology department with the complaint of chest pain. His coronary angiography revealed a serious lesion in the two years ago stented diagonal artery and additional lesion in the LAD. The patient then transferred to our clinic’s intensive care unit ward. On the first physical examination, the arterial blood pressure (ABP) was 160/90 mm Hg and the pulse rate 90/minute and rhythmic. There was an anterolateral ST elevation on the ECG. The cardiac enzyme profile was within normal limits. Blood urea was 125 mg/dL and creatinine was 4.8 mg/dL. Because of the critical coronary lesions and the clinical status it was decided to perform a CABG operation without respecting the high levels of urea and creatinine. We performed ultrafiltration and got 50 cc/hour during CPB and maintained an optimal level for arterial blood pressure. Renal supplement therapy was continued as veno-venous hemofiltration in intensive care unit after operation. His urine output was 20 cc/hour in first 5 hours and his BUN and creatinine was 72 mg/dl and 2.7 mg/dl. Renal supplement therapy was discontinued at postoperative 20th hour after his urine output increased to 200 ml/hour and BUN and creatinine decreased to 52 mg/dl and 1.7 mg/dl. The patient was discharged at postoperative 7th day with BUN 85mg/dl and creatinine 2.1 mg/dl.

DISCUSSION
The mortality and morbidity of CABG has a great variability according to multiple factors like patient’s age and gender, left ventricle functions, severity of coronary artery lesions, timing of the operation, etc. While operative risk and morbidity of CABG in patients with chronic stable angina pectoris is very low, it is very high in patients with unstable angina pectoris. Goldman et al indicated the operative mortality in stable angina pectoris (SAP) patients as 2.9% and as 5.4% in unstable angina pectoris (UAP) patients. Likewise the risk of perioperative myocardial infarction is 9.1% in SAP patients and 13.7% in UAP patients (3). The
incidence of acute renal failure after open heart surgery is between 2.5-7% and the mortality rate in this group is between 24-70% (2,3,4). In a study, low cardiac output after cardiac surgery was indicated as one of the main or adjunctive cause of postoperative renal failure (5). The patients who progressed to severe renal failure need renal replacement therapy get inotropic support and stay long in the ICU ward. This group of patients may also stay intubated longer and be in need for intraaortic balloon pumping. There is no determined level of urea and creatinine to begin the renal replacement therapy. The mean time for beginning replacement therapy is two to four days. But in acute renal failure hyperpotasemia and volume overload causing acute lung odema may develop rapidly. It is necessary to use an effective and sufficient renal supplement therapy method because of the knowledge of hyperuricemia increases the mortality and morbidity despite the long waiting time for criteria of starting to renal supplement therapy. Demirkilik and co-workers showed that it is important to recognise acute renal failure and begin to veno-venous hemofiltration immediately to decrease mortality and morbidity (6). The type of renal supplement therapy is also important. Hemodialysis is preferable for its some advantages like short time to get more liquid and rapid change of solute concentration of plasm. But it has also some drawbacks like need for a specialised team, heparinisation and hypotension at the beginning of the hemodialysis due to the shifts of liquids between different compartments. In addition, it can also cause dyshytrhymia because of rapid decrease in potassium level (7). Macias developed veno-venous hemofiltration in 1991 and in this technique via a double-lumen canula inserted in a large vein, a pump circulates the blood (8). Because of the rolling pump it can be used safely in patients who are hemodynamically instable (even MAP is 50 mm Hg).

We used intermittent veno-venous hemofiltration in our patient to maintain a small level of heparin and not to effect his limited hemodynamic criteria. We did not see any problem about renal failure especially this prerenal disease and high level of BUN and creatinine patient. We also did not increase the operative time in the patient who had a serious coronary disease and high peroperative mortality. We discharged the patient without any mortality and morbidity.

CONCLUSION

FPKD is a rare cause of renal insufficiency and the combination of FPKD and ischemic heart disease is also very rare. But preoperative or postoperative renal insufficiency can cause high mortality or morbidity rate and a longer hospital stay. We conclude that starting a renal supplement therapy immediately as hemofiltration during the operation and continuing the therapy with veno-venous hemofiltration in postoperative period may decrease the mortality and morbidity without delaying the operation in unstable angina pectoris patients.

CORRESPONDENCE TO

Bilgehan Savaş OZ Gülhane Askeri Tip Akademisi Kalp ve Damar Cerrahisi Etlik / Ankara / TURKEY 06010 Phone: 00 90 312 2860668 bsavasmd@hotmail.com bsavoz@yahoo.com

References

Author Information

Cengiz Bolcal, M.D.
Assistant Professor, Cardiovascular Surgery Department, Gulhane Military Medical Academy

Bilgehan Sava, M.D.
Assistant Professor, Cardiovascular Surgery Department, Gulhane Military Medical Academy

Ertugrul Ozul, M.D.
Associated Professor, Cardiovascular Surgery Department, Gulhane Military Medical Academy

Harun Tatar, M.D.
Professor and Chairman, Cardiovascular Surgery Department, Gulhane Military Medical Academy