Koroner Arter By-Pass Cerrahisi Sonrası Gelişen Şiddetli Hiperbilirubinemi Vakası

Severe Hyperbilirubinemia After Coronary Artery By-Pass Surgery: A Case Report

Yunus Keser YILMAZ¹, Meral EKİM², Savaş SARIKAYA³, Burak AÇIKGÖZ⁴, Mehmet ÇELİKBILEK⁴, Çiğdem Ünal KANTEKIN⁵, Hasan EKİM⁶, Ferit ÇİÇEKÇİOĞLU⁷

¹ Bozok University Faculty of Medicine Department of cardiovascular surgery, Yozgat/Turkey
² Bozok University School of Health, Department of Biochemistry, Yozgat/Turkey
³ Bozok University Faculty of Medicine Department of Cardiology, Yozgat/Turkey
⁴ Bozok University Faculty of Medicine Dept. of Internal Medicine Div. of Gastroenterology and Hepatology, Yozgat/Turkey
⁵ Bozok University Faculty of Medicine Department of Anesthesiology, Yozgat/Turkey

ÖZET


ABSTRACT

The pattern and frequency of hyperbilirubinemia after cardiac surgery and its effect on postoperative complications are not well described. Postoperative hyperbilirubinemia due to liver dysfunction may be associated with increased morbidity and mortality. We present a 60-year-old male patient that we detected pericardial hematoma and collapsed right ventricle after the coronary artery bypass surgery. Patient’s clinical status was improved after the operation for evacuating hematoma. Total bilirubin and direct bilirubin levels of patient were increased up to 8,3 mg/dl and 2,3 mg/dl respectively in the first day of re-operation. In follow up, bilirubin level reached to a maximum level of 23.1 mg/dl on sixth day. In ultrasonography hepatic tissue and bilirubin tract were normal. Hepatic function tests except bilirubin such as alanin aminotransferaz (ALT), aspartat aminotransferaz (AST) and gama glutamil transferaz (GGT) were in normal level. Hyperbilirubinemia resolved spontaneously without any specific treatment.

Key words: Coronary artery bypass, hyperbilirubinemia, Liver function tests.
Introduction

Pattern of hyperbilirubinemia, its incidence and severity after cardiac surgery are not well expressed. Incidence of hyperbilirubinemia after cardiac surgery has reported as 20% to 35.1% in last two decade studies (1). Postoperative hyperbilirubinemia due to liver dysfunction is associated with morbidity and mortality (2). Kraev et al. showed that postoperative hyperbilirubinemia is an independent predictor of long-term outcomes after cardiopulmonary bypass (3, 4).

We present the patient with severe hyperbilirubinemia after coronary artery by-pass surgery complicated with pericardial tamponade.

Case-Report

60-year-old male patient underwent coronary artery bypass surgery due to atherosclerotic coronary artery disease proven with coronary angiography. Left anterior descending artery (LAD) was bypassed with left internal mammary artery (LIMA) and circumflex artery (Cx), and right coronary artery (RCA) were bypassed with saphenous vein graft. Operation duration was 3 hours, and there were no any hemodynamic changes which may affect the clinical status of patient during operation. Operation was completed with success and patient was taken to intensive care unit.

Patient's hemodynamic status was deteriorated on postoperative day one, and there was a refractory hypotension that is unresponsive to medical therapy. Echocardiography was planned immediately for suspicion of pericardial tamponade. Bedside Echocardiography revealed organized hematoma that collapsed right ventricle. Patient underwent re-operation for evacuating hematoma. After evacuating the hematoma, his clinical status was improved, and blood pressure returned to the normal level. After operation, total bilirubin and direct bilirubin levels were increased to 8.3 mg/dl and of 2.3 mg/dl respectively in the first day of re-operation. In follow up, bilirubin level reached to a maximum level of 23.1 mg/dl on sixth day and then returned to basal level on postoperative twentieth day.

Patient was consulted with gastroenterology department and underwent ultrasonography due to suspicion for involvement of hepatic tissue and bilirubin tract. But no pathological findings were detected by ultrasound. And other hepatic tests indicating hepatic function including ALT, AST and GGT were in normal level. Bilirubin levels of patient were spontaneously and gradually decreased without any special medication and patient was discharged with healing.

Discussion

In this case report, we presented a patient with severe hyperbilirubinemia after coronary artery bypass surgery which was complicated with pericardial tamponade that immediately need to be evacuated. Patient was improved without taking any special medication and discharged without any complication.

Bilirubin is a degradation product of heme and therefore partially reflects the activity of heme oxygenase, which has established cardioprotective properties through its products, carbon monoxide and biliverdin (5). Incidence of hyperbilirubinemia after cardiac surgery ranging 20% to 35.1% in last two decade studies. Postoperative hyperbilirubinemia due to liver dysfunction is associated with morbidity and mortality (1, 2). Recently, Horsfall et al. reported a negative association of bilirubin with cardiovascular events and death in both men and women (6). After cardiac surgery, although the exact cause of this liver dysfunction is unknown, many factors are considered to contribute the development of jaundice. Possible factors lead to jaundice after cardiac surgery are liver hypoperfusion (7), hemolysis, a systemic inflammatory response to cardiopulmonary bypass (CPB), the type of surgical procedure, the age of the patient, the bypass time, the aortic cross-clamping time, the number of blood and plasma transfusions. Also, in patients with heart failure and increased right atrial pressure, postoperative jaundice was significantly higher (1,8). In our patient, after a successful cardiac surgery, cardiac tamponade developed. Resorption of hematoma may contribute to development of hyperbilirubinemia,
but in our case level of direct bilirubin (15 mg/dL) was higher contrast to indirect bilirubin (8.1 mg/dL). Also, there was a small residual hematoma after evacuation of hematoma.

In our patient, refractory hypotension developed due to cardiac tamponade so liver may be exposed to ischemia due to hypotension. Hepatic ischemia may be contributed to an increase of bilirubin level but in our patient, interestingly, liver function test except bilirubin, including ALT, AST, GGT were in normal range and did not increase in follow-up. Another possible mechanism to cause jaundice is blood transfusion given during and after first operation may lead to increase in bilirubin level. However, jaundice due to blood transfusion, usually, linked with increase in indirect bilirubin level in contrast to our case in whom direct bilirubin (15 mg/dL) and total bilirubin (23.1 mg/dL) levels were also increased. Also in our patient, preoperative and post-op echocardiography showed no heart failure and increased right atrial pressure which may cause post-op jaundice (1,8).

Cause of jaundice after cardiac surgery may be multifactorial. Exact cause of jaundice is not well known. Our case was different from previous cases presented with jaundice after cardiac surgery. Our case was complicated with cardiac tamponade which needed to be evacuated urgently, bilirubin level was very high and patient developed severe hyperbilirubinemia without any increase of other hepatic markers such as ALT, AST, GGT which are strong predictors of hepatic dysfunction.

In conclusion, cardiac surgery which is complicated with cardiac tamponade can cause severe elevation of bilirubin levels, despite immediate intervention for cardiac tamponade. Hyperbilirubinemia resolved spontaneously without any specific treatment.

**REFERENCES**