รายงานผู้ป่วย

Post acute myocardial infarction ventricular septal defects : surgical management.

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Acute rupture of the interventricular septum is a rare complication of acute myocardial infarction (MI). It usually occurs 2-3 days after the onset of acute MI. The patient will develop sudden onset of dyspnea and low cardiac output syndrome. Without aggressive medical and surgical treatment. The mortality is very high. We report have a male patient who was transferred to us because of shock after acute anteroseptal myocardial infarction. He developed severe pulmonary congestion and acute renal failure despite maximal support with Dobutamine, nitroglycerine and intraaortic balloon pump. An emergency echocardiography revealed a large anterior Ventricular Septal Defect (VSD) with a shunt flow of 4:1. Coronary angiography showed total occlusion of LAD and severe stenosis of PDA of RCA. An emergency closure of the VSD and a coronary artery bypass grafting to PDA was performed. The patient recovered remarkably well and had progressive improvement in renal functions. Three months after the operation, the patient was in good condition without recurrence of the VSD.

Key word : *Myocardial infarction ventrcalar septal defect.*

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การทะลุของผนังกั้นหัวใจด้านล่างภายหลังภาวะหัวใจตายจากการขาดเลือดเฉียบพลันพบได้น้อย มักเกิดหลังจากการตายของกล้ามเนื้อหัวใจ 2-3 วัน ผู้ป่วยมีอาการเหนื่อยทันที และมีภาวะโลหิตไปเลี้ยงร่างกายไม่เพียงพอ อัตราตายจะสูงมาก ถ้าหากไม่ได้รับการรักษาทางอายุรกรรมควบคู่กับการผ่าตัดให้ทันท่วงที เสนอรายงานผู้ป่วยรายที่ถูกส่งมาที่โรงพยาบาลจุฬาฯ เนื่องจากภาวะช็อค หลังการตายของกล้ามเนื้อหัวใจทางด้านหน้าและผนังกั้นห้องหัวใจ ผู้ป่วยมีอาการเลือดคั่งในปอด และไตวายเฉียบพลันแม้ได้ยาโดปามีน ไนโตรกลีเซอรีน ขนาดสูงสุด และอินตราเออร์ติค บอลลูน ปั๊มป์ เอคโคคาร์ดิโอแกรม ตรวจพบมีรูที่ผนังกั้นห้องหัวใจขนาดใหญ่ มีเลือดลัดในอัตรา 4 ต่อ 1 โคโรนารีแจงจิโอแกรม พบการอุดตันเต็มที่ของ แอลเอดี และการตีบ 90% ของเส้นเลือดพีดีเอ ซึ่งเป็นแขนงของหลอดเลือดแดงหัวใจด้านขวา การผ่าตัดฉุกเฉินเพื่อปิดรูรั่วของผนังหัวใจ และต่อหลอดเลือดพีดีเอ ทำให้ผู้ป่วยมีอาการดีขึ้นอย่างมาก ภาวะหัวใจวาย และไตวายดีขึ้นจนหายไป กลับสู่ปกติ 3 เดือน หลังการผ่าตัดผู้ป่วยไม่มีอาการเหนื่อยหรือเจ็บหน้าอกสามารถทำงานได้เป็นปกติ

Case report

The patient was a 42 year old male who was previously healthy. He was obese and hypertensive but a non-smoker. He denied any history of chest pain or heart problems. Four days before admission, while he was working as a truck driver, he experienced severe substernal discomfort. He was taken to a nearby private hospital and the diagnosis was acute anteroseptal myocardial infarction. Hew was admitted to the ICU and intravenous heparin and nitroglycerin were given. The pain subsided but on the fourth day after admission, he became severely dyspneic. His blood pressure dropped to 90/70 mmHg and the heart rate was 120/min. He was transferred to Chulalongkorn Hospital.

Upon admission to the Coronary Care unit he was found to be dyspneic and confused. His neck vein swere engorged. A loud holo systolic murmur was heard over his lower left sternal border. His blood pressure was 97/70 mmHg. There was fine crepitation over his lower lung areas bilaterally. An EKG showed acute anteroseptal MI. His chest X-ray showed cardiomegaly and bilateral pulmonary congestion. An emergency echocardiogram showed a defect in his interventricular septum with a left to right shunt of about 4 to 1. A Swan Ganz catheter was inserted via his right subculavian vein; his wedge pressure was 22 mmHg. There was definite oxygen stepup in his right ventricular level. Intravenous nitroglycerin, furosemide and dopamine were given. He did not respond well so an intraaortic balloon was inserted via his right femoral artery.

His condition improved. The initial laboratory tests revealed BUN/Cr. = 60/4.0 His condition stabilized but on the 4th day after admission he became more dyspneic and diaphoretic. His blood pressure dropped to 80/60 mmHg. An emergency coronary angiography was performed. It showed a total occlusion of LAD with poor runoff and 70% stenosis of PDA from the dominant RCA. An emergency operation was performed. With a median sternotomy incision, a saphenous vein was harvested from right lower leg. The aorta was clamped and cold crystalloid cardioplegia was infused via the aortic root. The infarcted area over the LV apex was opened. There was a VSD over the inferior part of the septum measuring about 5 x 6 cm^2. A synthetic dacron patch was used to closed the defect by interruted pledgetted sutures.

The ventriculotomy was closed with two strips of teflon patches and then a distal anastomosis to PDA was performed. The patient was rewarmed and the aortic clamps were released. The proximal anastomosis was then performed. The heart spontaneously returned beating. There was no bleeding from the ventriculotomy. He was weaned from the heart-lung machine without undue difficulty. His postoperative course was rather uneventful. His hemodynamic status was stable and he had good urine flow. His BUN and Cr. progressively returned to normal values. He was extubated 15 hours postoperatively. Examination revealed normal heart sounds and no murmurs. He was discharged home on the 10th postoperative day. Two years after the operation he is healthy without dyspnea nor angina.

Discussion

For most patients, acute myocardial infarction usually responds well to medical treatment alone. However, in less than 5% of the cases, acute rupture of the interventricular septum occurs.[1] It usually occurs as a complication of a first acute myocardial infarction.[2-4] A well developed collateral coronary circulation is uncommonly found in hearts with a postinfarction VSD.[5] It is also generally associated with complete obstruction (rather than severe stenosis) of a coronary artery, usually the LAD.[6] It is important that stenoses usually coexists in the right coronary artery system. It is most commonly located in the anterior or apical portion of the ventricular septum (60%) in association with a full-thickness anterior myocardial infarction. About 20% to 40% of patients have a VSD in the posterior portion of the ventricular septum in association with an inferior myocardial infarction. Because the posterior type of VSD in near to the crux of the heart. Its repair may injure coronary sinus and right coronary artery. So, the operative mortality is higher than the anterior type of VSD. Because post infarction VSD is usually large and resulting in inractable heart failure and pulmonary congestion. Early operation is usually necessary because of high failure rate with full medical treatment including intraaortic ballon pump. But because of friability of necrotic myocardium, the risk of recurrence of VSD and bleeding of the suture line is high. Delaying the operation for one month, if hemodynamically permissive, will make the operation safer. But this is not the usually case. without early operation, mortality rate is very high. The sudden onset of left heart failure is typically due to high shunt flow and an unadaptive left ventricle. The murmur is usually loud and should be differentiated from acute mitral regurgitation from rupture of papillary muscle. Early death rates are high. Only 75% of patients survive the first 24 hours and only 50% survive the first week.[6] An echocardiogram diagnoses the condition accurately. A Swan Ganz catheter should be inserted to confirm the diagnosis by demonstrating oxygen step-up in the right ventricle and also for hemodynamic monitoring. An intraaortic balloon pump should also be inserted immediately because of the tendency of the cndition of these patients to rapidly deteriorate.[7] Coronary angiography is mandatory because multi-coronary vessel in volvement is common. An immediate operation is required because permanent improvement does not folow use of the support devices only.[8] The VSD is always approached through the left ventricle (infarcted area). If there is significant coronary stenosis, a coronary bypass should be added. The internal mammary artery should be grafted to LAD if possible. In rare patients who have stable hemodynamic, repairs may be delayed for 2-3 weeks to allow the edge of defect to become fibrotic with less tendency to develop recurrent VSD in the postoperative period. In 10-25% of patients recurrent VSD happens usually due to reopening of the closed defect. The hospital mortality rate is usually about 35%.[9] Prompt surgical intrvention after minimal investigation, good myocardial preservation and coronary

grafting, if indicated, are principles to improve the results.

Conclusion

Here we reported a male patient with a ventricular septal defect complicating acute anteroseptal myocardial infarction. Despite miximal inotropic support, including intraaortic balloon counter-pulsation, his hemodynamic condition was severely impaired with pulmonary congestion, oliguria and acute renal failure and cardiogenic shock. Proeoperative coronary angiography revealed total occlusion of LAD and stenosis of PDA from RCA. Emergency patch closure of VSD and CABG to PDA was performed. The patient improved markedly and able to be weaned from a cardiopulmonary bypass with minimal inotropic support. The aortic ballon could be removed shortly after surgery. Follow up of this patient for more than one year this patient is in functional class one. No recurrence of VSD has been detected. We support an aggressive approach to post acute MI VSD with minimal workup and prompt surgery.

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