The Diagnostic Approach of Massive Ascites in Constrictive Pericarditis

Kamsi Rachmawati*, Astri Handayani*, Hamzah Shatri**, Idrus Alwi***, Hanafi B Trisnohadi***

INTRODUCTION

Constrictive pericarditis is thickening, fibrosis and attachment of visceral and parietal pericardium, so that it blocks ventricle loading of diastole. On most of case report, the etiology is tuberculosis. But nowadays tuberculosis is not the most-found etiology. Current etiology are post radiotherapy, heart operation, viral and bacterial infection, and malignancy such as breast cancer, lung cancer and lymphoma.1,4,5,6,9

Signs and symptoms of constrictive pericarditis resulted from; low cardiac outflow which causes fatigue, hypotension and tachycardia reflex; elevation of systemic pressure that are: elevation of jugularis vein pressure, hepatomegaly which is accompanied by ascites and peripheral edema; pulmonary vein congestion that are: dispnoe d’effort, cough and ortopnoe. The other signs that could be found are: paradoxical pulse i.e. decrease of pulse pressure up to 15 mm Hg at inspiration, because of reduction of heart loading as the consequences of elevated intra pericardial pressure and knock pericardial i.e. first diastolic sound which is frequently heard in the left sternum border that follows the second heart sound.1,4,5,6,9,10

The diagnostic approach was performed by careful history taking and physical examination accompanied by laboratory findings such as chest x-ray, electrocardiography, echocardiography, CT scan or MRI and heart catheterization. The chest x-ray usually reveals normal heart size, which is slightly enlarge in 10 % of cases. The cava vein is distended in upper right mediastinum area, the left atrium is enlarged in 1/3 cases, pericardial thickening seen in 60% of cases, but calcification is not specific for constrictive pericarditis. The electrocardiograph reveals abnormality i.e. low voltage QRS in 90 % cases, T wave inversion or flat T wave. Echocardiography findings reveals pericardial thickening, small ventricle chamber and diastolic load which is abruptly stopped at early diastolic session.

Diagnosis is established by heart catheterization, with three major key points i.e. : elevation and equalization of diastolic pressure in every heart chamber, configuration of dip and plateau at early diastolic in left and right ventricle and the right atrium pressure shows prominent y descent.1,4,6,13

Pericardiectomy is the only treatment to eliminate ventricle load resistance at diastolic phase.1,4,5,9,10

CASE

Mrs, A, 30 years old administered to Dr Cipto Mangunkusumo hospital with main symptoms of swollen foot and distended abdomen since 8 month before admission. First, it was transient, then became more persistent, and the abdomen more distended. Then, she hardly breathe, easily exhausted and felt better if she slept with 2 pillows. The patient’s abdominal fluid has been taken twice, it was about 3 litres by doctor. But in 2 weeks later, the patient felt her abdomen became distended again. There was unstable fever, loss of weight, and decrease urinary flow, with clear-yellow color. There was contact history to Tuberculosis patient (the patient’s mother). No history of tuberculosis, malignancy and radiation.

Physical findings by the time of admission were: general condition moderately ill. Fully alert blood pressure 90 / 70 mmHg, pulse rate 100 x/minute, respiration rate 20x/minute, temperature 36.7 °C. There

---

* Residents of Internal Medicine, Faculty of Medicine, University of Indonesia, Dr. Cipto Mangunkusumo Hospital, Jakarta
** Division of Psychosomatic, Department of Internal Medicine, Faculty of Medicine, University of Indonesia– Dr. Cipto Mangunkusumo Hospital, Jakarta
***Division of Cardiology, Department of Internal Medicine, Faculty of Medicine, University of Indonesia, Dr. Cipto Mangunkusumo Hospital, Jakarta
was vein dilatation on the forehead. JVP 5 + 2 cm H2O, no lymph node enlargement. First heart sound normal, second heart sound was markedly accentuated at tricuspid valve, no murmur and gallop were heard. Pulmonary findings showed vesicular breath sound, no rales and wheezing. Abdomen seen distended, liver and spleen were hardly evaluated because of distended abdomen, there was shifting dullness, and pitting edema on both of lower extremities.

Laboratory findings were Hb 10.2 gr/dl; Ht 29%; erythrocyte 4.1 million/mm3; leukocyte 6500/ul; ESD 10; thrombocyte 230.500/mm3; reticulocyte 1.4; urea 3.8; creatinin 0.8; sodium 132; potassium 3.6; SGOT 40; SGPT 25; albumin 4; globulin 3.8; indirect bilirubin 0.4; direct bilirubin 0.7; total bilirubin 1.1.

The chest x-ray showed absence of cardiac slope, blunt costo-phrenicus angle on the lung. Electrocardiograph showed low voltage appearance. The Mantoux test was negative.

The problem on this patient are massive ascites, considered because of right atrium congestion with differential diagnosis of primary pulmonary hipertension and pericarditis. Beside that, it is also considered as massive ascites, which is caused by peritoneal tuberculosis or because of malignancy. The patient was given diet of 2100 calorie, low sodium intake, measurement of fluid intake and urinary outflow, and fluid intake restriction 500 cc/day, diuretics furosemide 1 x 40mg and spironolactone 1 x 100 mg were given.

During hospitalization, ascites fluid function was done for diagnostic and therapeutic interest. There was 2 ½ litre of fluid, clear-yellow color with analysis result: cell amount 300 / microlitre; PMN 20%; MN 80%; protein 4.4 (serum 7.8); glucose 104 (serum 78); LDH 120 (serum 218); bacteria -; AFB -; exudate impression. Cytologic findings of ascites fluid: showed reactive mesothelial cell. In the ascites fluid, the AFB (Acid-Fast-Bacilli) was not directly found, and after homogenization, AFB was negative.

USG findings revealed liver congestion with distention of hepatic vein. Peritoneoscopy was done and giving an impression of chronic liver disease with differential diagnosis of malignancy. In the peritoneum there was no defect. Echocardiography findings revealed pericardial thickening, very mild tricuspid regurgitation, left ventricle systolic function was 58%, severe diastolic dysfunction, so that they give an impression of constrictive pericarditis. Confirming diagnosis of constrictive pericarditis, we performed heart catheterization, and the result were equalization of diastolic pressure in every heart chamber, the ventricle pressure showed “dip and plateau” apperance.

During hospitalization, the patient had fever accompanied by cough with productive sputum, clear-white color, dry-throat, and the abdomen felt distended again. We performed sputum examination, 3 x AFB stain, microorganisme resistance test, AFB culture, and repeat chest x-ray. We also repeated ascites fluid function for therapeutic interest, there was 2 ½ litre fluid out, dark-yellow color like tea color, and rather cloudy, and then we performed culture and resistance test of microorganism and repeated the AFB test. The patient was given cefotaxime 3x1 gr to prevent the spontaneous bacterial peritinitis (SBP), anti-tuberculosis drugs were also given: INH 1 x300 mg, B6 3x10 mg, ethambutol 2x500 mg, pirazinamide 2x500 mg. Because there were alergy of rifampicine and streptomicine, so that ofloxacine 1x400 mg was given. Then, pericardiec-
tomy was done and we found 2 cm pericardium thickening. After pericardiectomy, the patient condition was getting better. Histopathology findings showed non-specific chronic lymphadenitis, and bacterialis pericarditis.

**DISCUSSION**

Constrictive pericarditis usually started with early episode i.e. acute pericarditis which is might be clinically undetectable, and then established to sub acute phase - it becomes organization and effusion reabsorption, and then followed by chronic phase i.e. fibrosis and pericardium thickening with obliteration of pericardium chamber, so that it inhibits the heart volume distention at diastolic phase in every heart chamber. This alteration usually accompanied by calcium deposit. 1,4

The diagnostic approach of constrictive pericarditis needs special attention, not only in history taking, but also careful physical examination and good selection of laboratory examination.

The patient diagnosis was established based on signs and symptoms such as elevation of jugularis vein pressure accompanied by collateral on the patient’s forehead, hepatomegaly, ascites and peripheral edema. The most clear symptoms are ascites and hepatomegaly so that there is frequent error to disease diagnosis, such as liver chrisrosis, or intra abdominal tumor. The symptoms of fatigue, hypotension and tachycardia reflex in this patient is because of drops of heart flow. The symptoms of dispnue d’effort and ortopnoe are because of pulmonary vein congestion. In this patient, there is also pericardial knock i.e. early diastolic sound which is frequently heard on left sternum border which follows the second heart sound.

The correct laboratory examination selection is very important to establish and make precise diagnosis immediately, such as: chest x-ray, electrocardiography, and echocardiography and hear catheterization. On the thorax x-ray, there was absence of heart slope, electrocardiography showed low voltage apperance, liver ultrasonography showed liver congestion and echocardiograph findings showed severe diastolic dysfunction with parietal pericardial thickening. CT scan is the most aacute examination in order to evaluate anatomy and pericardial thickening but it cannot evaluate significant hemodynamic function in constrictive pericarditis.

Echocardiography is an important laboratory examination to establish the diagnosis because it can proof the presence of pericardium thickening and observe smaller ventricle chamber and great contraction and diastolic filling that abruptly stopped in early diastole. Transvalvular and venous flow velocities are effective diagnosis and they have an important role to differ constrictive pericarditis with restrictive cardiomyopathy. In constrictive pericarditis there is respiratory variation of flow profile, while in restrictive cardiomiopathy there is no alteration.

The heart catheterization examination is an important invasive procedure before pericardiectomy. It has been done with the result of elevation and equalization of diastolic pressure in every heart chamber. Hemodynamic appearance could mimics restrictive cardiomyopathy, but we could differ it by the presence of right ventricle systolic hypertension (pressure over 60 mm Hg) and left ventricle diastolic pressure is higher than the right ventricle that is over 5 mm Hg. It is important to differ these condition because constrictive pericarditis can be perfectly healed while most of restrictive cardiomyopathy cases cannot. 1,4,5,6,8,10

The etiology of constrictive pericarditis in this patient is unknown albeit of tuberculosis contact in her
The mantoux test was negative, AFB sputum and AFB culture and AFB of ascites fluid and PCR of tuberculosis were negative, so that pericardiectomy is not only as therapy but also to find its etiology by pathology anatomy examination.

In this patient management, diuretics were given to reduce jugular vein pressure, edema and ascites. Anti tuberculosis drugs also had been given for two weeks before pericardiectomy, and therapy were continued after operation.

Pericardiectomy is the only treatment to eliminate ventricle loading resistance in diastolic phase. Considering that this patient is young and having mild operation risk factor, then she has good prognosis after pericardiectomy. Central vein pressure needs several weeks or month after pericardiectomy back to its normal value. Post pericardiectomy, the left ventricle ejection fraction was low and it can be back to normal in several months.

Pharmacologic therapy in constrictive pericarditis has little role, but it is important. In some patient, pericarditis recovers spontaneously or as a response of drugs combination such as non-steroid anti inflammation drugs, steroid, and antibiotics. In other patients, drugs therapy has supportive importance. Specific antibiotics such as anti tuberculosis drugs should be given before pericardiectomy and it should be continued after the operation. Before pericardiectomy, diuretics should be given to reduce jugular pressure, edema and ascites. After pericardiectomy, diuretics should be given if there is no spontaneous diuresis. Diuretics and digoxin (if there was atrial fibrillation) is also given for the patient who will not have any pericardiectomy because of high operation risk. Drugs which could lower heart beat frequency as beta blocker and calcium antagonist should be avoided because of tachycardia sinus as compensation mechanism.

Prevention of constrictive pericarditis could be done by treating acute pericarditis correctly and adequate pericardial drainage. We could administer fibrinolytic therapy (urokinase 400.000 unit up to 1.600.000 unit, streprocytnase 250.000 up to 1.000.000 unit ). Corticosteroid usually not effective.

Post pericardiectomy, the patient was getting better and she could have normal life as other housewives. The mortality rate of pericardiectomy of 6% up to 19% had been reported, decrease ejection fraction of left ventricle after pericardiectomy happened up to 30%. In successful pericardiectomy, there are 90% symptoms improvement and 50% total recovery. Five year survival rate are ranged from 75 % up to 85 %, depend on right time to perform pericardiectomy.

CONCLUSION

Constrictive pericarditis will have good prognosis if it is managed well. Knowledge about constrictive pericarditis is important in clinical cardiology. History and laboratory findings is very important to establish this diagnosis. Pericardiectomy is the definitive therapy and giving hope to the patient to have his normal activity as before.
REFERENCES