



Research Journal of **Cardiology**

ISSN 1819-3404



Academic
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A Tricuspid Valve Endocarditis with a Large Vegetation Encroaching on the Papillary Muscle and Right Ventricular Cavity in Patient with a Ventricular Septal Defect

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ABSTRACT

Ventricular septal defect is the second most common cardiac malformation accounting for almost a fifth of all congenital cardiac anomalies. A large vegetation size in right heart endocarditis is associated with a high mortality rate up to 33%. The mortality rate varies according to the valves involved and the size of vegetation has a prognostic correlation. A 23 years old man was treated successfully who developed tricuspid valve endocarditis in un-repaired peri-membranous ventricular septal defect with vegetation size of 3.5 cm. An emergency surgical plan would be appropriate for a young man with a large right side vegetation complicated with a pericardial effusion and congestive heart failure. The study highlighted the potential of combined medical and surgical approach to handle such situation in hospitals.

Key words: Peri-membranous ventricular septal defect, Infective endocarditis, vegetation, *Staphylococcus hemolyticus*, mortality, cardiac malformation

INTRODUCTION

Ventricular Septal Defect (VSD) is usually identified during childhood. In adults, it is diagnosed less often owing to correction of large ventricular septal defects and the spontaneous closure of smaller ones during the patient's early years. Peri-membranous VSD, an opening in the upper section of the ventricular septum and near the valves, occurs in 75% of all VSD cases. Small ventricular septal defects pose a small but relatively high risk of infective endocarditis (Alabdulgader, 2001).

Although spontaneous closure has been reported in adults (Mehta *et al.*, 2000; Tomita *et al.*, 2001) but a significant number of small VSDs (30-50%) close spontaneously during the first 2 years of life, with the vast majority of defects that close before the patient is 4 years of age.

Most children with small defects remain asymptomatic without evidence of an increase in heart size, pulmonary artery pressure, or resistance. One of the long-term risks for these patients is infective endocarditis. Follow-up studies in adults with small VSDs without surgical treatment showed an increase in the incidence of arrhythmia, sub-aortic stenosis and exercise intolerance (Roso *et al.*, 2001).

Although right sided endocarditis is common in intravenous drug abusers but it also occurs in the cases of congenital heart disease and is known for its aggressiveness as it can lead to serious complications such as pulmonary abscesses and death (Alafify *et al.*, 2006).

This paper reported a case of a patient with peri-membranous VSD having an unusual large vegetation on his tricuspid valve, its diagnosis and subsequent management.

CASE REPORT

A 23 years old Somali (a male) university student suffering from congenital heart disease since early childhood visited our accident and emergency department complaining of fever, cough with blood streaked sputum, shortness of breath, swelling of lower limbs and abdomen for two weeks.

After preliminary investigation, he was admitted to orthopnea and paroxysmal nocturnal dyspnoea for one week. On examination, it was observed that he is in serious conditions looking ill, pale and jaundiced. He was conscious and oriented with vital signs showing a temperature of 38° Celsius, heart rate of 140 beats/minute, respiratory rate of 30 breaths/minute, blood pressure 95/60 mm Hg, with pitting lower limb edema and a petechial rash over both lower limbs.

He had a Janeway lesion in the sole of his right big toe and second degree clubbing of his fingers. No splinter hemorrhages or Roth's spots were identified. Examination of the abdomen revealed tender hepatomegally and ascitis. The apex beat was not displaced and a holo-systolic murmur 3/6 was heard in the mid-left sternal border. There were fine bilateral basal crackles elicited on auscultation of the chest. Urine analysis revealed microscopic hematuria.

Laboratory tests showed white cell count of 17.5×10^9 with 90% neutrophils and 10% lymphocytes. Hemoglobin level was 6.58 g dL^{-1} and the platelet count of 29 000. Liver function test showed an alanine transferase of $29 \mu \text{ dL}^{-1}$, an aspartate transferase $36 \mu \text{ dL}^{-1}$, total bilirubin $40.58 \text{ mmol L}^{-1}$ and direct bilirubin of $30.13 \text{ mmol L}^{-1}$.

Coagulation profile showed a pro-thrombin time of 15.4 sec, an activated pro-thrombin time of 44.5 sec and an international normalized ratio of 1.3. A chest radiograph indicated cardiomegaly with upper lobe diversion. Trans-thoracic echocardiography demonstrated a dilated right atrium and right ventricle. Peri-membranous VSD was identified with a maximum pressure gradient of 68 mm Hg and moderate tricuspid regurgitation. There was a large mobile vegetation measuring $20 \times 35 \text{ mm}$ attached to the tricuspid valve and also encroaching on the papillary muscle and right ventricular cavity, ventricular defect and septal margins. Moderate pericardial effusion was also detected. However there were no signs of cardiac tamponade (Fig. 1-2).

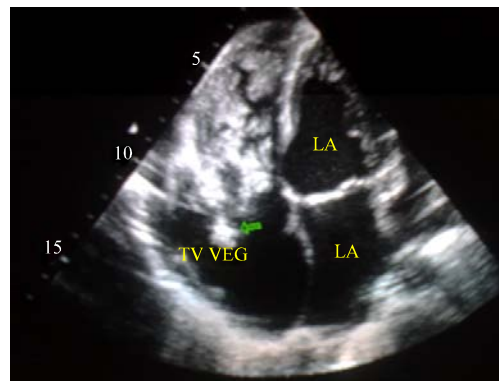


Fig. 1: Apical 4 chamber view showing large vegetation in the tricuspid valve, papillary muscle and pericardial effusion

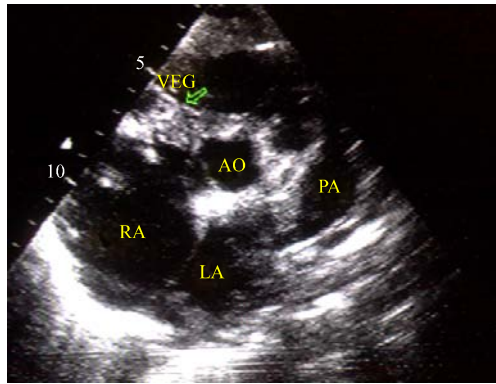


Fig. 2: Parasternal short axis view showing a peri-membranous VSD with vegetation of the tricuspid valve involving the defect margins

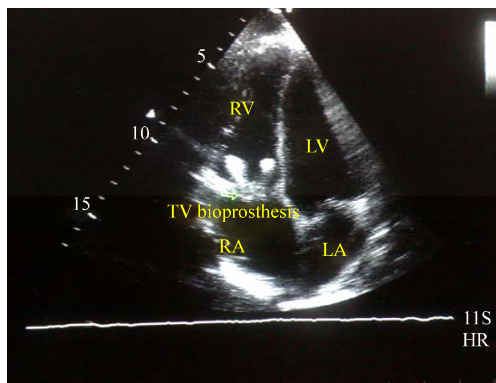


Fig. 3: Post-operative echo showing the tricuspid valve bioprosthesis

The patient was subsequently admitted to intensive coronary care unit and hypotension ensued requiring inotropic support. One unit of packed red blood cells and two units of platelets were administered. Treatment with cloxacillin 2 g intravenously every 4 h, gentamicin 60 mg intravenously every 8 h and ceftriaxone 2 g every 24 h was initiated. The patient was immediately transferred to a tertiary care center where an emergency open-heart surgery was performed on day 4. He underwent excision of the tricuspid valve along with vegetation. Vegetation was removed from the right ventricular cavity as well. Replacement of tricuspid valve with 31 mm bioprosthetic valve was performed and VSD repaired by a Gore Tex™ patch.

Blood cultures indicated *Staphylococcus hemolyticus* sensitive to oxacillin, co-trimoxazole, vancomycin and penicillin G and the patient was post-operatively commenced on vancomycin 300 mg twice daily and rifampicin 600 mg orally once daily for a total duration of 6 weeks.

Post-operative trans-thoracic echocardiography revealed moderate pericardial effusion, mild to moderate depression of right ventricular function, flattening of the septum with mildly depressed left ventricular function and no VSD.

Tricuspid valve bioprosthesis was well seated with a peak gradient of 11 mm Hg (Fig. 3). During hospitalization, the patient required multiple platelet transfusions. Trans-thoracic echocardiogram 12 days post-operation showed no pericardial effusion and a well functioning valve and hence he was discharged in good general conditions to pursue his studies.

DISCUSSION

This report is of a young man with unusual large vegetation attached to VSD with extension to tricuspid valve and papillary muscle. Some patients with right heart endocarditis have been treated by surgical intervention when medical treatment has failed (Edmond *et al.*, 2001; Domnisse, 1988). However, more urgent treatment may be required. According to clinical situation, an emergency open-heart surgery was done to this patient.

Another study reported a pregnant woman with large vegetation adjacent to a VSD with extension to pulmonary valve. An emergency open cardiac surgery was performed immediately after Cesarean section (Ou *et al.*, 2009).

Echocardiogram was performed to evaluate infective endocarditis. The pericardial effusion receives less attention than other findings such as location, size, or mobility of vegetation, leaflet perforation and severity of valve regurgitation (Miyake *et al.*, 2010). However, pericardial effusion is also clinically important. Among 17 aortic valve endocarditis patients with pericarditis, only 14 (82%) of the patients had perivalvular abscess (Behzadnia *et al.*, 2005). It was reported that cardiac tamponade is an extremely rare complication of infective endocarditis (Miyake *et al.*, 2010). Our patient had right heart endocarditis with moderate pericardial effusion and not complicated with tamponade as surgical approach was appropriate management for his clinical situation. Right heart endocarditis is also associated with pericardial effusion in one study where it was reported that pericardial effusion was present in 30% of patients mainly in those intravenous drug abuser with right sided endocarditis (Arnett and Roberts, 1976).

In our case, the organism involved was *Staphylococcus hemolyticus* (SH) a coagulase-negative member of the genus *Staphylococcus*. The bacterium can be found on normal human skin flora and can be isolated from axillae, perineum and inguinal areas of humans. SH is also the second most common coagulase-negative staphylococci present in human blood (Takeuchi *et al.*, 2005). Only rarely SH has been described as a causative agent for infective endocarditis and our patient is such a rare case (Caputo *et al.*, 1987; Senining *et al.*, 2001).

Coagulase-negative staphylococci are usually considered low-virulent pathogens compared to the well known pathogenic coagulase-positive *Staphylococcus aureus*. However recent studies indicated that coagulase-negative staphylococci have emerged as a major cause of opportunistic infection (Falcone *et al.*, 2007).

Retrospective analysis of one series of infective endocarditis of native and repaired VSDs showed that the commonest localization was the tricuspid valve and always in isolated VSDs, as in our case and the most common source of infection (38%) was dental, followed by Ear, Nose and Throat surgery, skin, gastrointestinal, pulmonary and unknown causes (Di-Filippo *et al.*, 2004). The survival in this series was 97.1, 94.3, 91.4 and 86.6% after 1- month, 6-months, 1-year and 5-10 years, respectively following infective endocarditis.

Studies of right sided endocarditis for size of vegetation versus outcome has been reported (Hecht and Berger, 1992; Di-Salvo *et al.*, 2001). Vegetation >2.0 cm is associated with a significantly higher mortality rate than vegetation ≤2.0 (33.0% compared to 1.3%) according to Hecht and Berger (1992).

CONCLUSIONS

In conclusion VSD is a benign cardiac lesion the prognosis of which can be severely compromised by the occurrence of infective endocarditis. Surgical repair reduces the risk but does

not entirely exclude it because of minor associated abnormalities. Patients whose VSD has been repaired early in life are unlikely to have any significant long-term problems. Prophylactic antibiotic therapy and the diagnosis of latent infectious problems, particularly dental, remain essential before and after cardiac surgery.

REFERENCES

- Alabdulgader, A.A., 2001. Congenital heart disease in 740 subjects: Epidemiological aspects. *Ann. Trop. Paediatr*, 21: 111-118.
- Alafify, A.A., T.S. Al-Khuwaitir, B.A. Wani and S.M. Taifur, 2006. *Staphylococcus aureus* endocarditis complicated by bilateral pneumothorax. *Saudi Med. J.*, 27: 707-710.
- Arnett, E.N. and W.C. Roberts, 1976. Valve ring abscess in active endocarditis. Frequency, location and clues to clinical diagnosis from the study of 95 necropsy patients. *Circulation*, 54: 140-145.
- Behzadnia, N., A. Tabarsi, B.S. Kashani, S.M. Mirsaeidi, M.V. Ollahpour *et al.*, 2005. Evaluation of patients with infective endocarditis in a pulmonary referral center. *Tanaffos*, 4: 41-45.
- Caputo, G.M., G.L. Archer, S.B. Calderwood, M.J. DiNubile and A.W. Karchmer, 1987. Native valve endocarditis due to coagulase-negative staphylococci: Clinical and microbiologic features. *Am. J. Med.*, 83: 619-625.
- Di-Filippo, S., B. Semiond, M. Celard, F. Sassolas and F. Vandenesch *et al.*, 2004. Characteristics of infectious endocarditis in ventricular septal defects in children and adults. *Arch. Mal. Coeur. Vaiss.*, 97: 507-514.
- Di-Salvo, G., G. Habib, V. Pergola, J.F. Avierinos and E. Philip *et al.*, 2001. Echocardiography predicts embolic events in infective endocarditis. *J. Am. Clin. Cardiol.*, 37: 1069-1076.
- Dommissie, J., 1988. Infective endocarditis in pregnancy. A report of 3 cases. *Afr. Med. J.*, 73: 186-187.
- Edmond, J.J., S.J. Eykyn and L.D. Smith, 2001. Community acquired staphylococcal pulmonary valve endocarditis in non-drug abuser: Case report and review of the literature. *Heart*, 86: 17-17.
- Falcone, M., F. Campaanelle, M. Giannella, S. Borbone, S. Stefani and M. Venditti, 2007. M. *Staphylococcus hemolyticus* endocarditis: clinical and microbiologic analysis of 4 cases. *Diagnosis Microbiol. Infect. Dis.*, 57: 325-331.
- Hecht, S.R. and M. Berger, 1992. Right-sided endocarditis in intravenous drug abusers. Prognostic features in 102 episodes. *Ann. Internal Med.*, 117: 560-566.
- Mehta, A.V., S. Goenka, B. Chidambaram and F. Hamati, 2000. Natural history of isolated ventricular septal defect in the first five years of life. *Tenn. Med.*, 93: 136-138.
- Miyake, M., C. Izumi, K. Kuwano, G. Honjo and H. Matsutani, 2010. Cardiac tamponade during transesophageal echocardiography in a patient with infective endocarditis. *J. Echocardiography*, 8: 25-27.
- Ou, T.Y., R.F. Chen, C.S. Hsu, P.F. Kao, F.L. Yu, S.O. Teng and W.S. Lee, 2009. Pulmonary valve endocarditis in a pregnant woman with a ventricular septal defect. *J. Microbiol. Immunol. Infect.*, 42: 92-95.
- Roso, A.P., A.O. Saez and M.P. Garcia, 2001. Bacterial endocarditis in an adult with ventricular septal defect. *Med. Internal*, 18: 396-397.

- Senining, R.C., H. Ahmad, R. Shahzad, A. Stahl-Avicolli, T.J. Lamoste and N.E. Soto, 2001. Prosthetic valve endocarditis caused by *Staphylococcus hemolyticus*. *Clin. Infect. Dis.*, 32: 100-100.
- Takeuchi, F., S. Watanabe, T. Baba, H. Yuzawa and T. Ito *et al.*, 2005. Whole genome sequencing of *Staphylococcus hemolyticus* uncovers the extreme plasticity of its genome and the evolution of human colonizing staphylococcal species. *J. Bacteriol.*, 187: 7292-7308.
- Tomita, H., Y. Arakaki, T. Yagihara and S. Echigo, 2001. Incidence of spontaneous closure of outlet ventricular septal defect. *Jap. Circ. J.*, 65: 364-366.