CLINICAL PROFILE AND ONE YEAR FOLLOW UP OF PATIENTS WITH CARDIAC TAMPONADE

Kader Muneer[1], Sajeer Kalathingathodika[2], Divya E. Mukund[3]

[1]Associate Professor, Department of Cardiology, Government Medical College, Kozhikode, Kerala.
[2]Assistant Professor, Department of Cardiology, Government Medical College, Kozhikode, Kerala.
[3]Former Senior Resident, Department of Cardiology, Government Medical College, Kozhikode, Kerala.

ABSTRACT

BACKGROUND

Cardiac tamponade is a medical emergency that requires pericardiocentesis. Most of the cases may be tuberculosis or malignant in aetiology. We wanted to assess the clinical profile and aetiology of pericardial effusion and 1-year outcome of these patients.

METHODS

Patients who presented with moderate to large pericardial effusion were evaluated clinically and with echocardiography for presence of cardiac tamponade. Subsequently analysis of fluid was done in those who underwent pericardiocentesis.

RESULTS

59 patients fulfilled the criterion of moderate to large pericardial effusion and 45 of them underwent pericardiocentesis. Tuberculosis was the aetiology in 16 patients, 13 had malignant effusion and 7 patients had CKD.

CONCLUSIONS

There is a strong clinical correlation between pulses paradoxes and cardiac tamponade. Myocardial injury as evidenced by troponin I elevation was rarely seen. Development of chronic constrictive pericarditis is a rare event in a timely managed patient.

KEYWORDS

Pericardial Effusion, Cardiac Tamponade, Pericardiocentesis

HOW TO CITE THIS ARTICLE: Kader M, Sajeer K, Divya EM. Clinical profile and one year follow up of patients with cardiac tamponade. J. Evid. Based Med. Healthc. 2019; 6(26), 1777-1781. DOI: 10.18410/jebmh/2019/362

BACKGROUND

Cardiac tamponade is a symptom complex characterized by systemic venous congestion and low cardiac output due to restriction of ventricular filling caused by rapid accumulation of pericardial fluid. The clinical feature of tamponade mainly depends on the rapidity in the accumulation of pericardial fluid rather than the absolute volume as evidenced by the lack of tamponade features in slowly accumulating large infusion, on the contrary even fluid as little as 200 mL can cause cardiac tamponade if accumulation is rapid.[1-9]

Several inflammatory conditions can lead to pericarditis and subsequent tamponade. Effusion due to bacterial and fungal infection have higher predilection for progression to tamponade. As idiopathic pericarditis is fairly common, it accounts for most of the cases of cardiac tamponade in clinical practice. Traumatic, often iatrogenic and malignant effusion usually present with cardiac tamponade as the collection is rapid.[10,11,12,13]

Financial or Other, Competing Interest: None.
Submission 01-06-2019, Peer Review 11-06-2019,
Acceptance 17-06-2019, Published 26-06-2019.
Corresponding Author:
Dr. Divya E. Mukund,
Former Senior Resident,
Department of Cardiology,
Government Medical College,
Kozhikode, Kerala.
E-mail: divyaem@gmail.com
DOI: 10.18410/jebmh/2019/362

Haemodynamics

The basic hemodynamic feature of cardiac tamponade (CT) is the impedance to the filling of right sided heart chambers and subsequent under filling of left hart chambers leading to hemodynamic collapse. As fluid accumulates in the pericardial sac the intrapericardial, pressure first equalizes and then exceeds the atrial pressure resulting in reduction of transmural filling pressure (the difference between intracardiac and intrapericardial pressures). This reduction in transmural filling pressure on the right side leads to systemic venous congestion and characterized by elevate JVP. Absent Y descent and prominent X descent is a characteristic hemodynamic finding of CT. Predominant cardiac filling occurring during ventricular systole is manifested as prominent X descent and reduced filling during ventricular diastole is manifested as absent Y descent. The most important hemodynamic feature of CT is reduced LV filling and subsequent fall in cardiac output and systolic BP during inspiration. This is due to marked under-filling of LV during inspiration due to negative intrathoracic pressure. This leads to pulsus paradoxes, an important clinical manifestation of CT characterized by exaggerated fall in systolic BP during inspiration.[1,14,15,16,17]

As we discussed earlier, the clinical manifestation of CT occurs when the intrapericardial pressure exceeds intracardiac pressure. If intracardiac pressure is severely elevated as in LV dysfunction, LV hypertrophy, or due to severe aortic regurgitation large pericardial effusion may not cause any hemodynamic compromise and in some patient with low intracardiac pressure can develop features of CT

even with small effusion, (low-pressure tamponade).Pericardial tamponade is a medical emergency and the primary treatment is pericardiocentesis. This is usually done under fluoroscopic guidance. Echocardiography guided pericardiocentesis can be done in emergency situations by an experienced operator. A study over 100 patients with this approach had a high success rate (97%) with low complications (2%).[18,19,20]

Aetiopathogenesis

In most cases of CT analysis pericardial fluid has a low yield for definitive diagnosis. The diagnosis is often made by ancillary findings and other investigation, in rare cases pericardial biopsy may be useful.[3,4,5,8,9]

METHODS

In a prospective study conducted in Government Medical College, Calicut, Kerala during a period from Jan 2016 to Jan 2017, all consecutive patients presented with pericardial effusions diagnosed with the help of echocardiography and/ or with clinical features of CT were included. And all patients with large effusion or cardiac tamponade underwent pericardial aspiration once consented.

Pericardial effusion was defined as echo free space more than 5 mm around the heart and defined as large if the collection is more than 20 mm at maximum point. Epicardial fat was excluded by its greyish appearance, localization to anterior RV and absent compression during cardiac contraction. Cardiac tamponade by echo is defined as diastolic RV collapse with or without RA collapse, exaggerated inspiratory fall in mitral inflow of more than 20% and/ or more than 20% fall in aortic pulse wave Doppler velocity.[11,21,22,23,24,25] All the patients were also evaluated for clinical symptoms contributing to the aetiology of pericardial effusion and clinical signs of tamponade, like elevated JVP, systemic hypotension and pulsus paradoxus. In addition to routine blood investigations, LFT, RFT, TFT were done in all patients. An ECG and X-ray was taken during hospitalization. ANA, rheumatoid factor, HIV screening and cardiac troponin I were measured.

Pericardial aspiration was done under fluoroscopy guidance in Cath Lab with full aseptic precautions. Patient was hydrated with rapid infusion of 1L saline, if hypotension and paradoxical pulse coexisted with non-elevated JVP. Aspiration was done through subxiphoid approach with a 16G needle and 0.035 wire was introduced into the pericardium after confirmation with contrast injection. A pig tail catheter was introduced over the wire for continuing aspiration for the subsequent days. The pericardial fluid was analysed for its appearance; colour and total amount of aspirate was noted. The fluid was tested for biochemical parameters, cytology, routine culture and AFB culture, ADA was also tested.[26-28]

Pericardial effusion was considered idiopathic when no aetiology was identified at the time of presentation and/ or investigations including analysis of pericardial fluid or pathology of specimen didn't reveal any specific aetiology. Though the criteria of tuberculosis pericarditis were the identification of tubercle bacilli in the pericardial fluid or tissue biopsy by culture or PCR the yield was so low, so an exudative pericardial effusion with lymphocytic predominance and elevated ADA and positive Mantoux was considered tuberculosis and treated with anti-tuberculosis drugs for 6 months. Identification of malignant cells in pericardial fluid characterizes of neoplastic effusion but haemorrhagic effusion in a patient with malignancy was considered neoplastic when no other causes were identified. Diagnosis of chronic pericardial effusion was made the effusion persisted for 3 months and all these patients were further evaluated at 6 months and 1 year for the development of constrictive pericarditis.[29-32]

Follow Up

Follow up echo was done daily during hospitalization and the indwelling catheter was removed once there is no fluid to aspirate. Follow up echo was done in 1 month and 6 months in all surviving patients and evaluated for the recurrence of effusion and for the development of constrictive pericarditis. The latter was diagnosed by persistent venous congestion, thickened pericardium. Ventricular interdependence which is characterized by exaggerated reciprocal changes in the LV and RV diastolic volumes during inspiration and expiration and intracardiac and intrathoracic dissociation of pressures as evidenced by exaggerated (>20%) fall in mitral inflow velocity during inspiration were also assessed.[18,19,27,32]

RESULTS

Total 59 patients with large effusion were evaluated for tamponade of which 49 patients with confirmed tamponade either clinically or with echocardiogram were subjected to pericardiocentesis. Though tamponade was seen in all age groups maximum incidence was seen between 30 to 50 years. The mean age was 44.5+/- 16.6. (Table 1). Pericardial effusion was equally distributed among both sexes. Predominant symptom was dyspnoea and cough followed by chest pain and fever in one third of patients. Edema was present in 16 patient's and pulsus paradoxus in 12 patients. Tachypnea was present in 34 patients. JVP was elevated in 26 and hypotension was noted in 4 patients. Anemia was present in 64% of patients with elevated ESR in 39 patients and high CRP in 38 patients. Pericardiocentesis was done in 45 patients and it was transudative in 12 patients, exudative in 34 and chylous in 1. Culture and sensitivity were positive in 2 patients with pyogenic pericarditis, most of the patients were culture negative. AFB culture was also negative. ADA was negative in 76% of the patients. Chest X-ray revealed cardiomegaly in 42 patients with mild pleural effusion in 15 patients and focal consolidation in 2 patients. Most of the patients were in sinus rhythm, low voltage was seen in 27% of the patients. Pulses alternans was rare as with ST/T changes in ECG. The effusion was moderate to large in most of the patients with features of tamponade seen in 38 patients (Table 3). The most common aetiology being tuberculosis (16 patients) followed by malignancy (13 patients) (Table 2). 15 patients expired and 2 patients lost follow up. Most of the remaining

patient effusion subsided by 6 months with persistent effusion in 5 patient and 1 patient developing constrictive pericarditis.

Univariate analysis predicting the association between various clinical and lab parameters with the development of tamponade showed that patients with tamponade were more symptomatic with higher incidence of fever and dyspnoea (Table 3, 4). Elevated JVP, pulsus paradoxus, muffled heart sounds were strongly associated with tamponade than other signs. Sensitivity, specificity and predictive values of many clinical features were analysed, dyspnoea was more sensitive and least specific and pulsus paradoxus was most specific (Table 4, 5).

Age	Number	Percent
0 - 10	2	3.4
11 - 20	3	5.1
21 - 30	7	11.9
31 - 40	13	22.0
41 - 50	13	22.0
51 - 60	9	15.3
61 - 70	9	15.3
71 - 80	3	5.1
Mean ± SD	44.5 ± 16.6	

Table 1. Percentage Distribution of Sample According to Age

Diagnosis	Number	Percent
TB	16	27.1
Pyopericardium	5	8.5
Idiopathic	2	3.4
Malignancy	13	22.0
Chylous	1	1.7
Others	8	13.6
CKD	7	11.9
CTD	3	5.1
Post STEMI	3	5.1
TB with CTD	1	1.7

Table 2. Aetiology

		Tamponade				$\chi 2$	p
		Present		Absent			
		No.	%	No.	%		
Age	<=30	5	41.7	7	58.3	0.25	0.884
	31 - 60	12	34.3	23	65.7		
	>60	4	33.3	8	66.7		
Sex	Male	12	41.4	17	58.6	0.83	0.361
	Female	9	30.0	21	70.0		
Fever	Present	3	13.6	19	86.4	7.38**	0.007
	Absent	18	48.6	19	51.4		
Cough	Present	10	33.3	20	66.7	0.14	0.712
	Absent	11	37.9	18	62.1		
Chest pain	Present	5	21.7	18	78.3	3.16	0.076
	Absent	16	44.4	20	55.6		
Dyspnoea	Present	20	43.5	26	56.5	5.66*	0.017
	Absent	1	7.7	12	92.3		
Fatigue	Present	2	40.0	3	60.0	0.05	0.830
	Absent	19	35.2	35	64.8		
Pallor	Present	7	38.9	11	61.1	0.12	0.726
	Absent	14	34.1	27	65.9		
LN	Present	7	63.6	4	36.4	4.64*	0.031
	Absent	14	29.2	34	70.8		
Oedema	Present	7	43.8	9	56.3	0.64	0.425
	Absent	14	32.6	29	67.4		

Table 3. Comparison of Selected Variables Based on Tamponade

		Tamponade				χ^2	p
		Present		Absent			
		Count	Percent	Count	Percent		
Pulse rate	Bradycardia	1	50.0	1	50.0	5.52	0.063
	Normal	9	24.3	28	75.7		
	Tachycardia	11	55.0	9	45.0		
Blood Pressure	Hypotension	2	50.0	2	50.0	0.4	0.819
	Normotension	15	34.9	28	65.1		
	Hypertension	4	33.3	8	66.7		
Temperature	Normal	21	38.2	34	61.8	2.37	0.124
	Febrile	0	0.0	4	100.0		
Respiratory Rate	Normal	5	20.0	20	80.0	4.6*	0.032
	Tachypnoea	16	47.1	18	52.9		
JVP	Normal	8	24.2	25	75.8	4.21*	0.040
	Elevated	13	50.0	13	50.0		
Pulsus paradoxus	Present	12	100.0	0	0.0	27.26**	0.000
	Absent	9	19.1	38	80.9		
Auscultation	Normal	8	22.9	27	77.1	7.87*	0.020
	Muffled	12	60.0	8	40.0		
	Rub	1	25.0	3	75.0		

Table 4. Comparison of Selected Variables Based on Tamponade

		Tamponade		
		Present	Absent	Total
Dyspnoea	Present	20	26	46
	Absent	1	12	13
Oedema	Present	7	9	16
	Absent	14	29	43
Pulse rate	Tachy	11	9	20
	Others	10	29	39
Respiratory Rate	Tachypnoea	16	18	34
	Normal	5	20	25
JVP	Elevated	13	13	26
	Normal	8	25	33
Pulsus paradoxus	Present	12	0	12
	Absent	9	38	47
Auscultation	Muffled heart sounds	12	8	20
	Others	9	30	39
ESR	High	16	23	39
	Normal	5	15	20
CRP	High	3	10	13
	Normal	18	28	46

Table 5. Predictive Power of Tamponade by Selected Variables

	Sensitivity	Specificity	False Negative	False positive	Positive Predictive value	Negative Predictive value	Positive Likelihood ratio	Negative Likelihood ratio	Accuracy
Dyspnoea	95.2	31.6	4.8	68.4	43.5	92.3	1.4	0.2	54.2
Oedema	33.3	76.3	66.7	23.7	43.8	67.4	1.4	0.9	61.0
Pulse rate	52.4	76.3	47.6	23.7	55.0	74.4	2.2	0.6	67.8
RESP	76.2	52.6	23.8	47.4	47.1	80.0	1.6	0.5	61.0
JVP	61.9	65.8	38.1	34.2	50.0	75.8	1.8	0.6	64.4

Pulsus paradoxus	57.1	100.0	42.9	0.0	100.0	80.9		0.4	84.7
Auscultation	57.1	78.9	42.9	21.1	60.0	76.9	2.7	0.5	71.2
ESR	76.2	39.5	23.8	60.5	41.0	75.0	1.3	0.6	52.5
CRP	14.3	73.7	85.7	26.3	23.1	60.9	0.5	1.2	52.5

Table 6. Sensitivity Specificity and Predictive Value

DISCUSSION

Pericarditis and secondary pericardial effusion constitute 5% of overall hospital admissions and incidence being more common in male sex and adults. The most common presentation is shortness of breath followed by cough, chest pain and fever. The prevalence of these symptoms in our study correlates with the prevalence in previous studies. Elevated JVP was seen in 26 patients (44%) and pulsus paradoxus in 12 patients (20%) and significant hypotension in 4 patients. The occurrence of pulsus paradoxus is similar to other studies and it the most specific sign of cardiac tamponade.[13,33,34,16] Elevated polymorph nuclear leukocytosis is seen in most of the patients (96%) and elevated ESR in two- third of the patients. 5 patients were positive for ANA and 5 had elevated troponin I. Of the 5 patients with ANA positivity, 4 had systemic lupus and 1 rheumatoid arthritis. ANA is a useful screening test in patients with idiopathic pericardial effusion to rule out connective tissue disorders. Of the 5 patients with elevated troponin I 3 were patients admitted with acute myocardial infarction. The low prevalence of troponin I elevation suggests that myocardial injury is uncommon during pericarditis and CT.[35,15,36] Chest X-ray showed pleural effusion and / or patchy consolidation in 18 patients and Mantoux test was positive in 6 patients.

Most of our patients had moderate to large effusion and features of cardiac tamponade as evident by diastolic RV collapse with or without RA collapse. Inspiratory fall in mitral or aortic flow velocity was seen in 21 patients. The most common etiological factor identified was tuberculosis (27%), followed by malignancy (22%) and chronic kidney disease (20%) (Table 2). Tuberculosis is a leading cause of pericardial effusion in developing countries with a reported mortality of 25% at 6 months follow up. But all our patients with TB effusion had a good recovery within 6 months of anti-tuberculosis treatment.[36,32,37] 2 patients developed features of constriction in the first follow up echo. One of them improved at 6 months suggestive of transient constriction. The other patient required surgical pericardiectomy for persistent venous congestion with pericardial calcification on CT scan at 1 year.[29,30,31] Overall 15 patients expired during the study period, 10 had malignancy and Ca lung was the most common. 2 patients had pyopericardium and expired within few days after hospital admission. Pericardial fluid culture grown methicillin resistant staphylococci in both of them. All 5 patients with CKD improved and had mild residual effusion on follow up. One patient developed perforation of right ventricle during pericardiocentesis and completely recovered by conservative management. There were no deaths related to pericardiocentesis. 2 patients had recurrent large pericardial effusion, 1 with CKD and another with chylous effusion required surgical pericardiectomy.

Limitations

1. Number of patients enrolled may not be adequate to predict the correlation with many clinical variables.
2. Diagnosis of tuberculosis effusion is made by elevated adenine de-aminase and Mantoux positivity rather than AFB demonstration.
3. Limited follow up time may the reason for the low incidence of constrictive pericarditis.

CONCLUSIONS

This study revealed the clinical characteristics and outcome of patients with large pericardial effusion. Though tuberculosis is still the most common cause of effusion, malignant effusion and CKD are emerging as new leading causes. Pericardiocentesis is required in large effusions showing features of tamponade which can be identified by characteristic clinical features and echocardiographic parameters. It is a safe and lifesaving procedure in these patients and gives a clue to the aetiology of pericardial effusion. Constrictive pericarditis, a sequele of large effusion is rare and only one of our patients developed chronic constriction during follow up.

REFERENCES

[1] Zipes DP, Libby P, Bonow RO. Braunwald's heart disease: a textbook of cardiovascular medicine. 10th edn. Phliadelphia, PA: Elsevier Health Sciences 2015.
[2] Oakley CM. Myocarditis, pericarditis and other pericardial diseases. Heart 2000;84(4):449-454.
[3] Imazio M, Gaita F, LeWinter M. Evaluation and treatment of pericarditis: a systematic review. JAMA 2015;314(14):1498-1506. (Erratum in JAMA 2015;314:1978).
[4] Mayosi BM, Wiysonge CS, Ntsekhe M, et al. Mortality inpatients treated for tuberculous pericarditis in sub-Saharan Africa. S Afr Med J 2008;98(1):36-40.
[5] Gornik HI, Gerhard-Herman M, Beckman JA. Abnormal cytology predicts poor prognosis in cancer patients with pericardial effusion. J Clin Oncol 2005;23(22):5211-5216.
[6] Applefield M, Cole JF, Pollock SH, et al. The late appearance of chronic pericardial disease in patients treated by radiotherapy for Hodgkin's disease. Ann Intern Med 1981;94(3):338-341.
[7] Imazio M, Negro A, Belli R, et al. Frequency and prognostic significance of pericarditis following acute myocardial infarction treated by primary percutaneous coronary intervention. Am J Cardiol 2009;103(11):1525-1529.
[8] Imazio M, Hoit BD. Post-cardiac injury syndromes: An emerging cause of pericardial diseases. Int J Cardiol 2013;168(2):648-652.
[9] Agner RC, Gallis HA. Pericarditis: differential diagnostic considerations. Arch Intern Med 1979;139(4):407-412.

[10] Ditchey R, Engler R, LeWinter M, et al. The role of the right heart in acute cardiac tamponade in dogs. Circ Res 1981;48(5):701-710.
[11] Klein AL, Abbara S, Agler DA, et al. American Society of Echocardiography clinical recommendations formulti modality cardiovascular imaging of patients with pericardial disease: endorsed by the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. J Am Soc Echocardiogr 2013;26(9):965-1012. (e1015).
[12] Imazio M, Adler Y. Management of pericardial effusion. Eur Heart J 2013;34(16):1186-1197.
[13] Sagristà-Sauleda J, Mercé J, Permanyer-Miralda G, et al. Clinical clues to the causes of large pericardial effusions. Am J Med 2000;109(2):95-101.
[14] Kytö V, Sipilä J, Rautava P. Clinical profile and influences on outcomes in patients hospitalized for acute pericarditis. Circulation 2014;130(18):1601-1606.
[15] Setty NHS, Yeriswamy MC, Jadav S, et al. Clinico-etiological profile of cardiac tamponade in a tertiary care centre. J Cardiovasc Med Cardiol 2016;3(1):041-044.
[16] Reddy PS, Curtiss EI, O'Toole JD, et al. Cardiac tamponade: hemodynamic observations in man. Circulation 1978;58(2):265-272.
[17] Guberman BA, Fowler NO, Engel PJ, et al. Cardiac tamponade in medical patients. Circulation 1981;64(3):633-640.
[18] Tsang TS, Enriquez-Sarano M, Freeman WK, et al. Consecutive 1127 therapeutic echocardiographically guided pericardiocenteses: clinical profile, practice patterns and outcomes spanning 21 years. Mayo Clin Proc 2002;77(5):429-436.
[19] Rafique AM, Patel N, Biner S, et al. Frequency of recurrence of pericardial tamponade in patients with extended versus non extended pericardial catheter drainage. Am J Cardiol 2011;108(12):1820-1825.
[20] Gumrukcuoglu HA, Odabasi D, Akdag S, et al. Management of cardiac tamponade: a comperative study between echo-guided pericardiocentesis and surgery - a report of 100 patients. Cardiol Res Pract 2011;2011:197838.
[21] Feigenbaum H, Waldhausan JA, Hyde LP. Ultrasound diagnosis of pericardial effusion. JAMA 1965;191:711-714.
[22] Haaz WS, Mintz GS, Kotler MN, et al. Two dimensional echocardiographic recognition of the descending thoracic aorta: value in differentiating pericardial from pleural effusions. Am J Cardiol 1980;46(5):739-743.
[23] Singh S, Wann LS, Schuchard GH, et al. Right ventricular and right atrial collapse in patients with cardiac tamponade – a combined echocardiographic and hemodynamic study. Circulation 1984;70(6):966-971.
[24] Himelman RB, Kircher B, Rockey DC, et al. Inferior vena cava plethora with blunted respiratory response: a sensitive echocardiographic sign of cardiac tamponade. J Am Coll Cardiol 1988;12(6):1470-1477.
[25] Settle HP, Adolph RJ, Fowler NO, et al. Echocardiographic study of cardiac tamponade. Circulation 1977;56(6):951-959.
[26] Mercé J, Sagristà-Sauleda J, Permanyer-Miralda G, et al. Should pericardial drainage be performed routinely inpatients who have a large pericardial effusion without tamponade? Am J Med 1998;105(2):106-109.
[27] Sagristà-Sauleda J, Angel J, Permanyer-Miralda G, et al. Long-term follow-up of idiopathic chronic pericardial effusion. N Engl J Med. 1999; 341: 2054-2059.
[28] Imazio M, Spodick DH, Brucato A, et al. Controversial issues in the management of pericardial diseases. Circulation 2010;121(7):916-928.
[29] Singh Y, Ahmad S, Srivastava S, et al. Trends in aetiology and outcome of patients with pericardial effusion: a five-year experience. JIACM 2008;9(2):96-99.
[30] Mutyaba AK, Balkaran S, Cloete R, et al. Constrictive pericarditis requiring pericardiectomy at Groote Schuur Hospital, Cape Town, South Africa: causes and perioperative outcomes in HIV era (1990-2012). J Thorac Cardiovasc Surg 2014;148(6):3058-3065.e1.
[31] Haley JH, Tajik AJ, Danielson GK, et al. Transient constrictive pericarditis: causes and natural history. J Am Coll Cardiol 2004;43(2):271-275.
[32] Mayosi BM, Burgess LJ, Doubell AF. Tuberculous pericarditis. Circulation 2005;112:3608-3616.
[33] Curtiss EI, Reddy PS, Uretsky BF, et al. Pulsus paradoxus: definition and relation to the severity of cardiac tamponade. Am Heart J 1988;115(2):391-398.
[34] Roy CL, Minor MA, Brookhart MA, et al. Does this patient with a pericardial effusion have cardiac tamponade? JAMA 2007;297(16):1810-1818.
[35] Reuter H, Burgess LJ, Doubell AF. Epidemiology of pericardial effusions at a large academic hospital in South Africa. Epidemiol Infect 2005;133(3):393-399.
[36] Yaqoob I, Khan KA, Beig JR, et al. Etiological profile of pericardial effusion in Kashmir: a study from northern India. Int Inv J Med & Med Sci 2016;3(1):1-5.
[37] Levine MJ, Lorell BH, Diver DJ, et al. Implications of echocardiographically assisted diagnosis of pericardial tamponade in contemporary medical patients: detection before hemodynamic embarrassment. J Am Coll Cardiol 1991;17(1):59-65.