Original Article

A Hospital-Based Study on Causes Peculiar to Heart Failure

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Abstract

Background: *We sought to determine the frequency of the risk factors for congestive cardiac failure (CCF) in a tertiary care hospital in Peshawar, Pakistan.*

Methods: *This retrospective, observational study was conducted in the department of cardiology, Postgraduate Medical Institute, Lady Reading Hospital Peshawar, from March 2005 to September 2007. Relevant information regarding the risk factors of CCF was recorded on questionnaires, devised in accordance with the objectives of the study.*

Results: *This study recruited 1019 patients with an established diagnosis of CCF on the basis of clinical findings and pertinent investigations. The study population comprised 583 (57.12%) men and 436 (42.78%) women. The patients' age ranged from 6 years to 82 years with a mean age of 48.5 years and a mode of age of 45 years. The distribution of the causative factors of CCF was as follows: ischemic heart disease in 38.56%; hypertension in 26.30%; dilated cardiomyopathies in 10.10%; obstructive and restrictive cardiomyopathies in 5.39%; valvular heart diseases in 9.32%; congenital heart diseases like ventricular septal defects and atrial septal defects in 4.41% and 0.58%, respectively; constrictive pericarditis in 1.07%; pericardial effusion in 0.68%; chronic obstructive pulmonary disease and pulmonary hypertension in 1.47%; thyrotoxicosis in 0.68%; complete heart block in 0.29%; and Paget's disease in 0.09% of the cases.*

Conclusion: *Ischemic heart disease, hypertension, cardiomyopathy, valvular heart disease, and congenital heart disease were the major contributors to CCF in our patients.*

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Introduction

Congestive heart failure, also referred to as congestive cardiac failure (CCF) or just heart failure, is a condition that can result from any structural or functional cardiac disorder that impairs the ability of the heart to fill with or pump a sufficient amount of blood through the body.[1] It is not to be confused with "cessation of heartbeat", which is known as asystole, or with cardiac arrest, which is the cessation of normal cardiac function with subsequent hemodynamic collapse leading to death. Because not all patients have volume overload at the time of initial or subsequent evaluation, the term "heart failure" is preferred over the older term "congestive heart failure".[2] CCF is often undiagnosed due to a lack of a universally agreed definition and difficulties in diagnosis, particularly when the condition is considered "mild". Even with the best therapy, CCF is associated with an annual mortality of 10%.[3]

The symptoms depend largely on the side of the heart which is failing predominantly. Given that the left side of the heart pumps the blood from the lungs to the organs,

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failure to do so leads to the congestion of the lung veins and symptoms that reflect this, as well as a reduced supply of blood to the tissues. The predominant respiratory symptom is shortness of breath on exertion (or in severe cases at rest) and easy fatigability.[4] Echocardiography is commonly used to support a clinical diagnosis of CCF. This modality uses ultrasound to determine the stroke volume (SV), which is the amount of blood that exits the ventricles with each beat; the end-diastolic volume (EDV), which is the total amount of blood at the end of diastole; and the SV in proportion to the EDV, which is a value known as the ejection fraction (EF). Normally, EF should be between 50% and 70%; however, in systolic heart failure, it drops below 40% (Smith A, Aylward P, Campbell T. Therapeutic Guidelines: Cardiovascular, 4th edition. 2003). No system of diagnostic criteria has hitherto been agreed upon as the gold standard for CCF. Commonly used systems are the Framingham criteria[5] (derived from the Framingham Heart Study), the Boston criteria,[6] and the Duke criteria.[7] Functional classification is generally done in accordance with the New York Heart Association Functional Classification.[8]

The American Heart Association has reported the following causes of CCF:[9]

- Causes of left-sided CCF: hypertension (high blood pressure), aortic and mitral valve disease, and aortic coarctation
- Causes of right-sided CCF: pulmonary hypertension (e.g. due to chronic lung disease), pulmonary valve disease, and tricuspid valve disease
- Causes that may affect both sides: ischemic heart disease (due to insufficient vascular supply, usually as a result of coronary artery disease); this may be chronic or secondary to acute myocardial infarction (a heart attack), chronic arrhythmias (e.g. atrial fibrillation), cardiomyopathy of any cause, cardiac fibrosis, chronic severe anemia, and thyroid diseases (hyperthyroidism and hypothyroidism)

The present study was designed to determine the frequency of the risk factors for CCF in a tertiary care hospital in Peshawar, Pakistan.

Methods

This retrospective, observational study was conducted in the department of cardiology, Postgraduate Medical Institute, Lady Reading Hospital Peshawar (PGMI/LRH), from March 2005 to September 2007.

This study randomly included 1019 patients, consisting of 583 (57.12%) men and 436 (42.78%) women, with an established diagnosis of CCF based upon clinical findings and relevant investigations. All the data were hospital-based, and only patients admitted with CCF in the cardiology unit of PGMI/LRH were recruited. It may be deserving of note that this teaching hospital, located in a north-west frontier province of Pakistan, is the only cardiac interventional centre in the public health sector that provides cardiac health to afghan refugees as well. Out-patient department patients with CCF were not included in the study.

The modalities utilized for the diagnosis of ischemic heart disease were electrocardiograms (ECG), echocardiography, and coronary angiography. Hypertension was defined as systolic blood pressure more than 140 mmHg and diastolic blood pressure more than 90 mmHg in our study. Cardiomyopathies were diagnosed via echocardiography.

A detailed history of the patients was taken with the help of a predesigned questionnaire, prepared in accordance with the objectives of this study. The questionnaire also contained information regarding the age, sex, address, and occupation of the patients.

All the patients with CCF, irrespective of age and sex, were included. The chief complaints and a detailed history of all the patients were recorded. The past and family history of major risk factors such as hypertension, ischemic heart diseases, valvular heart diseases, congenital heart diseases, and thyroid diseases were also registered. The clinical symptoms recorded were shortness of breath, history of ischemic heart diseases, peripartum history, palpitation, and swelling of the body. Tachycardia, positive leg edema, valvular abnormalities on auscultation, and cardiomyopathies were considered the signs of CCF. The blood pressure of all the patients was recorded. Fasting blood sugar, random blood sugar; serum cholesterol level, and triglyceride levels were also recorded from the ward record of the patients. ECG, echocardiography, and chest X-ray findings suggestive of CCF were also noted.

In a few cases, thyroid function tests and Troponin T cardiac enzymes tests were carried out to further assess the patients with CCF secondary to ischemic heart disease and thyroid dysfunction. Exclusion criteria were all patients with similar clinical features to CCF due to other causes such as asthma and chronic obstructive pulmonary disease that had yet not progressed to CCF.

Finally, the results were analyzed and the association between the risk factors and CCF was studied.

Results

This study included 1019 patients with an established diagnosis of CCF on the basis of clinical findings and pertinent investigations. The study population was comprised of 583 (57.12%) men and 436 (42.78%) women. The patients' age ranged from 6 years to 82 years with a mean age of 48.5 years and a mode of age of 45 years (Table 1). The respective mean age of the males and females was 49.3 and 45.6 years.

The distribution of the causative factors of CCF was:

ischemic heart disease in 38.56%; hypertension in 26.30%; dilated cardiomyopathy in 10.10%; obstructive and restrictive cardiomyopathies in 5.39%; valvular heart diseases in 9.32%; congenital heart diseases such as ventricular septal defects and atrial septal defects in 4.41% and 0.58%, respectively; constrictive pericarditis in 1.07%; pericardial effusion in 0.68%; chronic obstructive pulmonary disease and pulmonary hypertension in 1.47%; thyrotoxicosis in 0.68%; complete heart block in 0.29%; and Paget's disease in 0.09% of the cases (Table 2).

Table 1. Age-wise distribution of congestive cardiac failure patients (n=1019)

Age range	Number of patients	Percentage of total (%)
0-20 (y)	152	14.91
21-40 (y)	225	22.08
41-60 (y)	468	45.92
>60 (y)	174	17.07

Table 2. Distribution of causes of congestive cardiac failure (n=1019)

Cause	Number of patients	Percentage of total (%)
Ischemic heart disease	370	38.56
Hypertension	268	26.30
Dilated cardiomyopathy	103	10.10
Obstructive and restrictive cardiomyopathies	55	5.39
Mitral valve diseases	33	3.23
Mitral and aortic involvement	47	4.61
Combined mitral, aortic and tricuspid diseases	13	1.27
Tricuspid incompetence alone	2	0.19
Ventricular septal defects	45	4.41
Atrial septal defects	6	0.58
Constrictive pericarditis	11	1.07
Pericardial effusion	7	0.68
COPD and pulmonary hypertension	15	1.47
Thyrotoxicosis	7	0.68
Complete heart block	3	0.29
Paget disease	1	0.09

COPD, Chronic obstructive pulmonary disease

Discussion

CCF is a condition in which the function of the heart as a pump to deliver oxygen-rich blood to the body is inadequate to meet the requirements of the body. CCF can be caused by diseases that weaken the heart muscle, diseases that lead to the stiffening of the heart muscles, or diseases that increase oxygen demand by the body tissues beyond the capability of the heart to deliver.[10] In the present study, we assessed patients within an age range of 6 years to 82 years (mean age, 48.5 years; mode of age, 45 years). The prevalence of CCF is known to rise with increasing age and affects about 10% of the population older than 75 years of age.[11] We observed that ischemic heart disease was the most common cause of CCF as it was recorded in 36.31% of our cases. The findings of many studies reported from various countries of the world chime in with our findings.[12-14] Hypertension, recorded in 26.30% of the cases, was the second most common risk factor for CCF in our patients. Hypertension is a major risk factor for developing cardiac hypertrophy and heart failure. It has been reported that hypertrophied and failing hearts display alterations in excitation-contraction (E-C) coupling. However, it is unclear whether the remodeling of the E-C coupling system occurs before or after the development of heart disease. Chen-Izu Y et al.[15] reported that hypertension caused changes in the E-C coupling system, which in turn induced hypertrophy.

Cardiomyopathies were the third most important cause of CCF in our patients (15.49%). Cardiomyopathy is regarded as primary when the heart is considered to be the only organ involved. In secondary cardiomyopathy, heart lesion is part of a systemic disease.

Dilated cardiomyopathy was recorded alone in 10.10% of our cases and obstructive and restrictive cardiomyopathies in 5.39% of the cases. Our finding is concordant with that of Khan MA et al.[16] from Pakistan. In the present study, 9.32% cases of CCF were attributed to valvular heart diseases. Mitral valve disease was encountered in 3.23% of the cases, which is line with the Aizawa K et al. study.[17] Aortic, tricuspid, and pulmonary involvement was also significant in our study, and this finding matches results observed in various international studies correlating valvular diseases with CCF. [18,19] Congenital heart disease is believed to be the leading factor for the development of CCF. Ventricular septal defect was observed in 45 patients in our series. The prevalence of congenital heart disease is likely to be underestimated because of the trend towards home deliveries and the brief stay of neonates in the hospital in case of hospital deliveries. Most cases are detected upon referral for cyanosis, clubbing, or cardiac murmur. The number of patients with congenital heart disease is on the increase because of a steady addition and increased longevity.[20] The prevalence of ventricular septal defect and atrial septal defect in a study reported from Pakistan was 21% and 16% of the patients, respectively.[21] There were 7 cases of pericardial effusion in our series. These patients had developed pericarditis of myocarditis due to underlying pericardial effusion and had then developed left ventricular dysfunction.

CCF is a serious condition, for which no definite cure has thus far been discovered. Be that as it may, CCF patients can live a full and enjoyable life with the right treatment and constant attention to their lifestyle. Fortunately, the major risk factors for CCF are modifiable and can easily be prevented.

Conclusion

In our patients, ischemic heart disease, hypertension, cardiomyopathy, valvular heart disease, and congenital heart disease were the major contributors to CCF, whereas constrictive pericarditis, pericardial effusion, chronic obstructive pulmonary disease and pulmonary hypertension, thyroid disease, complete heart failure, and Paget's disease were the minor contributors to CCF.

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References

1. Neubauer S. The failing heart: an engine out of fuel. N Engl J Med 2007;356:1140-1151.
2. Krumholz HM, Chen YT, Wang Y, Vaccarino V, Radford MJ, Horwitz RI. Predictors of readmission among elderly survivors of admission with heart failure. Am Heart J 2000;139:72-77.
3. Raphael C, Briscoe C, Davies J. Limitations of the New York heart association functional classification system and self-reported walking distances in chronic heart failure. Heart 2007;93:476-482.
4. Auble TE, Hsieh M, McCausland JB, Yealy DM. Comparison of four clinical prediction rules for estimating risk in heart failure. Ann Emerg Med 2007;50:127-135.
5. McKee PA, Castelli WP, McNamara PM, Kannel WB. The natural history of congestive heart failure: the Framingham study. N Engl J Med 1971;285:1441-1446.
6. Carlson KJ, Lee DC, Goroll AH, Leahy M, Johnson RA. An analysis of physicians' reasons for prescribing long-term digitalis therapy in outpatients. J Chronic Dis 1985;38:733-739.
7. Harlan WR, Oberman A, Grimm R, Rosati RA. Chronic congestive heart failure in coronary artery disease: clinical criteria. Ann Intern Med 1977;86:133-138.
8. Raphael C, Briscoe C, Davies J. Limitations of the New York heart association functional classification system and self-reported walking distances in chronic heart failure. Heart 2007;93:476-482.
9. Hunt SA, Abraham WT, Chin MH, Feldman AM, Francis GS, Ganiats TG, Jessup M, Konstam MA, Mancini DM, Michl K, Oates JA, Rahko PS, Silver MA, Stevenson LW, Yancy CW, Antman EM, Smith SC Jr, Adams CD, Anderson JL, Faxon DP, Fuster V, Halperin JL, Hiratzka LF, Jacobs AK, Nishimura R, Ornato JP, Page RL, Riegel B; American College of Cardiology; American Heart Association Task Force on Practice Guidelines; American College of Chest Physicians; International Society for Heart and Lung Transplantation; Heart Rhythm Society. ACC/AHA 2005 Guideline Update for the Diagnosis and Management of Chronic Heart Failure in the Adult: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure): developed in collaboration with the American College of Chest Physicians and the International Society for Heart and Lung Transplantation: endorsed by the Heart Rhythm Society. Circulation 2005;112:e154-235.
10. Balaguru D, Artman M, Auslender M. Management of heart failure in children. Curr Probl Pediatr 2000;30:1-35.
11. Collins SP, Hinckley WR, Storrow AB. Critical review and recommendations for nesiritide use in the emergency department. J Emerg Med 2005;29:317-329.
12. Bradshaw D, Norman R, Pieterse D, Levitt NS; South African Comparative Risk Assessment Collaborating Group. Estimating the burden of disease attributable to diabetes in South Africa in 2000. S Afr Med J 2007;97:700-706.
13. Biccard BM, Bandu R. Clinical risk predictors associated with cardiac mortality following vascular surgery in South African patients. Cardiovasc J Afr 2007;18:216-220.
14. Finsterer J, Stollberger C. Noncompaction and neuromuscular disease with positive troponin-T in a nonagenerian. Clin Cardiol 2007;30:527-528.
15. Chen-Izu Y, Chen L, Banyasz T, McCulle SL, Norton B. Hypertension-induced remodeling of cardiac excitation-contraction coupling in ventricular myocytes occurs prior to hypertrophy development. Am J Physiol Heart Circ Physiol 2007;293:3301-3310.
16. Khan MA, Mohammad J, Hussain M. Frequency and echocardiographic study of dilated cardiomyopathy in children presenting with cardiac failure. Pak J Med Sci 2004;20:113-116.
17. Aizawa K, Tateishi A, Sakano Y, Kaminishi Y, Ohki S, Saito T, Konishi H, Kawada M, Misawa Y. Repair of paravalvular leak after a third mitral valve replacement. Kyobu Geka 2007;60:903-905.
18. Aqel RA, Hage FG, Zoghbi GJ. Percutaneous aortic valvuloplasty as a bridge to a high-risk percutaneous coronary intervention. J Invasive Cardiol 2007;19:238-241.
19. Yang X, Wu Q, Xu J, Shen X, Gao S, Liu F. Repair of flail leaflet of the tricuspid valve by a simple cusp remodeling technique. J Card Surg 2007;22:333-335.
20. Ferencz C, Rubin JD, McCarter RJ, Brenner JI, Neill CA, Perry LW, Hepner SI, Downing JW. Congenital heart disease: prevalence at live birth. The Baltimore Washington infant study. Am J Epidemiol 1985;121:31-36.
21. Ahmad R, Awan ZA, Bukshi F. A prevalence study of congenital heart disease in NWFP, Pakistan. Pak J Med Sci 2002;18:95-98.