The Spectrum of Paediatric Congenital Heart Disease at The Kenyatta National Hospital: Implications for Surgical Care.

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Abstract

Background

Congenital Heart Disease (CHD) is a significant cause of morbidity and mortality amongst infants and children globally. Complex heart lesions are more costly to manage than simple lesions. Geographical differences in the spectrum of CHD have been reported; knowledge of the spectrum of CHD provides a foundation for the rational allocation of health care resources. We undertook a study to determine if the CHD spectrum in Kenya differs from that encountered elsewhere in the globe.

Methods

A five year retrospective study was carried out at the Kenyatta National Hospital, Nairobi between January 1st 2000 and December 31st 2004. The frequency of selected CHD lesions was examined and compared with that reported from other parts of the world.

Results

Two hundred and fourteen patients were studied. The mean age at confirmation of diagnosis by echocardiography was 18.6 months. The frequency of ventricular septal defects, atrial septal defects, patent ductus arteriosus, tetralogy of Fallot and transposition of the great arteries, were 18.7%, 4.7%, 10.7%, 8.4%, 6.5% and 4.2% respectively.

Conclusion

The spectrum of CHD encountered in Kenya may contain more complex lesions than the spectrum found in developed regions of the world. Health resource allocation formulae applied to developed nations may not optimize the care of patients with CHD in developing nations. This may have important implications for health care resource allocation towards CHD in Kenya.

Introduction

Congenital heart disease (CHD) is a significant cause of morbidity and mortality amongst infants and children (1). The optimal care of patients with CHD requires an organized Paediatric Cardiac Care (PCC) program; the development of such programs requires adequate financial and human resources. Knowledge of the local spectrum of CHD provides the foundation for the rational allocation of healthcare resources. It has been reported that CHD spectra differ according to geographical location (2). The surgical management of complex CHD lesions utilizes more resources than the management of simple lesions (3). Current resource allocation towards PCC (in the study locality) is based on data obtained from studies carried out in developed nations; this practice may not facilitate optimal resource allocation (4). CHD may be classified as being cyanotic or acyanotic. Cyanotic lesions tend to be more anatomically complex than acyanotic ones. Typically, the incidence of acyanotic lesions is higher than that of cyanotic ones and so anatomically simpler lesions are more common than complex lesions (5, 6). One study examining the spectrum of CHD has been carried out locally but the sample size was small (7). The current study aims to determine the spectrum of CHD encountered at a referral hospital in Kenya; with a view to providing data that may contribute to the rational allocation of resources to PCC programs locally.

Methods

A five-year retrospective study was carried out at a tertiary level hospital in Nairobi [Kenyatta National Hospital (KNH)] between January 1st

2000 and December 31st 2004. Patients less than 13 years old with CHD confirmed by transthoracic echocardiography (ECHO) were studied (The ECHO may have been performed prior to referral to Kenyatta National Hospital). Relevant data were obtained from patient case notes and was entered onto data sheets. The study commenced after approval by the KNH Research and Ethics Committee.

Results

Two hundred and fourteen patients were studied. The mean age at confirmation of diagnosis by ECHO was 18.6 months (Range; 1 week to 17 years). The incidence of lesions encountered is shown in table 1.

LESION	NUMBER OF PATIENTS (n=214)
VSD	40 (18.7%)
ASD	10(4.7%)
TGA	14(6.5%)
TOF	18 (8.4%)
C-AVCD	14 (6.5%)
PDA	23 (10.7%)
TA	9 (4.2%)
CoA	2 (0.9%)
OTHERS	84 (39.2%)
TOTAL	214

Table 1. Spectrum of lesions in current study
VSD=Ventricular septal defect,
ASD=Atrial septal defect,
TGA= Transposition of great arteries,
TOF= Tetralogy of Fallot,
C-AVCD= Complete Atrio-ventricular canal defect,
PDA= Patent DuctusArteriosus
CoA= Coarctation of the Aorta

Discussion

The current study found that acyanotic lesions were more common than cyanotic lesions. This is in keeping with what has been found in other geographical regions (5,6,8,9). The frequency of relatively more complex CHD lesions (TGA and TOF) were higher in the current study than in studies carried out in Europe (5,8,10). The frequency of TOF in these European studies was consistently approximately 3% compared to a frequency of 8.4% in the current study. The frequencies of less complex lesions (ASD and VSD) were lower in the current study than in these European studies. The frequency of VSD's was between 34.6% and 45% in the European studies compared to 18.7% in our study and the frequency of ASD's was between 12.2% and 17% compared to 4.7% in our study. Similar findings are noted when our results are compared with a study from Australia (11).

It has been proposed that the aetiology of CHD involves environmental and genetic factors (12). Environmental factors may impact nutrition; sections of some developing countries frequently suffer from food insecurity. Under such circumstances, expectant mothers may not have access to an adequate diet at critical points in gestation. It has been shown that the risk of the occurrence of neural tube defects was reduced by administering folic acid around the time of conception and that the prevalence of severe congenital heart disease was significantly reduced following folic acid fortification of grain products in Canada (13,14). Dietary deficiencies during the organogenesis phase (1st trimester) of gestation may predispose to congenital cardiac lesions. Indeed the embryological development of the heart involves complex interactions between components of the developing heart-tube and the lungs (15). The complexity of these interactions could provide the substrate for an increased likelihood of malformations in settings of nutritional inadequacy.

The mean age at confirmation of a diagnosis of CHD by ECHO in our study was 18.6 months. The majority of VSDs that close spontaneously will do so by about 19 months of age (16). It is possible that the delay in diagnosis of VSDs in our study allowed more VSDs to close spontaneously thereby resulting in a lower incidence of VSDs being found at ECHO. Ventricular septal defects result in a left to right shunt; this causes an increase in pulmonary blood flow, which predisposes to frequent chest infections. An alternative explanation for the lower frequency of VSD may be that death may have occurred in some patients due to a combination of poor access to antibiotics and a delay in CHD diagnosis.

The fact that most ASDs are asymptomatic in childhood and considering the financial implications of seeking medical assistance in some developing nations, it is possible that few ASD patients would present themselves to medical personnel in our study locality. This could be an alternative explanation for the lower ASD incidence in our study (17).

Patients with complex lesions often require more extensive pre-operative investigations and may require more elaborate pre-operative preparation for surgery. The care of patients with complex lesions requires more resources than the care of patients with simple lesions (3). Our finding of an increased incidence of the more complex CHD lesions may have important implications for national health care resource allocation.

In conclusion, this study provides some evidence

to suggest that there may be a significantly greater incidence of complex CHD lesions in developing countries than in developed ones. Developing nations should examine their local CHD spectra as the knowledge yielded may facilitate the rational allocation of health care resources for the management of CHD.

References

1. Boneva RS, Botto LD, Yang Q, Correa A and Erichson D. Mortality associated with congenital heart defects in the United States-Trends and Racial Disparities, 1979-1997 Circulation 2001; 103: 2376-2381.
2. Grech V. Spectrum of congenital heart disease in Malta-An excess of lesions causing right ventricular outflow tract obstruction in a population based study. European Heart Journal. 1998; 9: 522-525.
3. Connor JA, Gauvreau K and Jenkins KJ. Factors associated with increased resource utilization for congenital heart disease. Pediatrics. 2005;116: 689-695.
4. Miltton C and Donaldson C. Health care priority setting: principles, practice and challenges. Cost Effec Reso Allo 2004; 2:3-11.
5. Schwedler G, Lindinger A, Lange PE et al.Frequency and spectrum of congenital heart defects among live births in Germany : a study of the Competence Network for Congenital Heart Defects.Clin Res Cardiol. 2011; 100(12):1111-1117.
6. Kapoor R, Gupta S. Prevalence of congenital heart disease, Kanpur, India.Indian Pediatr. 2008;45(4):309-311
7. Anabwani GM, Book W and Bonhoeffer P. Echo-cardiographic findings in Eldoret: Retropsective study. EAMJ. 1996; 72(11): 714-716.
8. Stephensen SS, Sigfusson G, Eiriksson H, et al. Congenital cardiac malformations in Iceland from 1990 through 1999.Cardiol Young. 2004;14(4):396-401.
9. Subramanyan R, Joy J, Venugopalan P, Sapru A, al Khusaiby SM. Incidence and spectrum of congenital heart disease in Oman. Ann Trop Paediatr. 2000; 20(4): 337-341.
10. Dilber D, MalcićI. Spectrum of congenital heart defects in Croatia.Eur J Pediatr.2010 ;169(5):543-550.
11. Bolisetty S, Daftary A, Ewald D, Knight B and Wheaton G. Congenital heart defects in Central Australia. MJA.2004; 180(12): 614-617.
12. Patterson DF. Epidemiology and Genetic Studies of Congenital Heart Disease in the dog. Circulation Research. 1968; 23:171-202.
13. Werler MM, Shapiro S and Mitchell AA. Periconceptional folic acid exposure and risk of occurrent neural tube defects. JAMA 1993;269(10): 1257-1261.
14. Ionescu-Ittu R, Marelli AJ, Mackie AS and Pilote L. Prevalence of severe congenital heart disease after folic acid fortification of grain products: time trends analysis in Quebec, Canada. BMJ 2009; 338:b1673.
15. Foley A and Mercola M. Heart induction: Embryology to Cardiomyocyte regeneration. Trends in Cardiovascular Medicine. 2004; 14(3): 121-125.
16. Miyake T, Shinohara T, Nakamura Y et al. Spontaneous closure of ventricular septal defects followed up from <3 months of age. Pediatri Int. 2004; 46(2): 135-140.
17. Parvathy U, Balakrishnan KR, Ranjith MS et al. Surgical closure of atrial septal defects in children under two years of age. Asian Cardiovasc Thorasc Ann. 2004; 12: 296-299.