50 CASES IN CLINICAL CARDIOLOGY

ATUL LUTHRA

JAYPEE

50 Cases in Clinical Cardiology

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A Problem Solving Approach

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Foreword

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JAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD
New Delhi • London • Philadelphia • Panama

Jaypee Brothers Medical Publishers (P) Ltd.

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New Delhi 110 002, India
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SW1H 0HW (UK)
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Email: info@jpmedpub.com

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Website: www.jaypeebrothers.com
Website: www.jaypeedigital.com

50 Cases in Clinical Cardiology: A Problem Solving Approach

First Edition: **2014**

ISBN 978-93-5152-110-5

Printed at

Dedicated to
My Parents
Ms Prem Lata Luthra
and
Mr Prem Prakash Luthra
Who guide and bless me
from heaven

Foreword

With the widespread availability of sophisticated cutting-edge technology, the clinician's approach towards the diagnosis of heart disease has undergone a paradigm shift. These days, it is not uncommon for the patient to be wheeled into the ECHO-room or even the cath-lab, without anyone taking a medical history or even caring to place a stethoscope over the patient's precordium. This is not a good sign since history-taking along with clinical examination should continue to occupy their rightful place in the practice of bedside cardiology. Moreover, a wealth of information is available in simple diagnostic modalities such as the ECG and X-ray chest, which should be interpreted in the light of clinical data.

I must compliment Atul Luthra for this brilliant compilation of a wide variety of real-world clinical situations, encountered during the practice of cardiology. He has elegantly discussed each case and solved the clinical problem in a meticulous way. The section on discussion incorporates a bewildering array of high-quality ECG strips, X-ray films and ECHO images. Students preparing for their examinations, resident doctors working in cardiac units and clinicians involved in heart-care are bound to benefit from this book. I wish Atul and his excellent book, all the best.

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Preface

Present-day cardiology is replete with a bewildering array of sophisticated investigative techniques, that have eclipsed the art of arriving at a diagnosis on the bedside of the patient. Yet, a relevant medical history and a meticulous physical examination are indispensable tools to mentally construct a plausible clinical diagnosis. Further, simple but informative investigations such as electrocardiography (ECG), chest radiography (X-ray) and echocardiography (ECHO), have withstood the test of time in clinical cardiology. Moreover, they are cost-effective in resource-sensitive settings and can be performed at the patient's bedside.

It gives me immense pleasure to proudly present *50 Cases in Clinical Cardiology: A Problem Solving Approach*, a compilation of real-world situations in clinical cardiology. Each case is introduced with a brief history and findings on physical examination. The clinical problem is then discussed analytically and ultimately solved with the aid of one or more simple bedside investigations. The case concludes with pertinent management issues along with some recent advances in diagnostics and therapeutics pertaining to that clinical entity. The text is suitably complemented by impressive illustrations of ECG strips, chest X-rays and ECHO images.

I have tried to incorporate most clinical situations encountered in heart clinics and cardiology ward-rounds, but there might be some omissions. Nevertheless, I sincerely hope that the wealth of clinical material on cardiac symptoms, physical signs and auscultatory findings, will rekindle the romance between the clinician and clinical cardiology. This book should be most useful for cardiology students preparing for examinations, resident doctors working in cardiac units as well as for physicians involved in the care of heart patients.

Atul Luthra

Acknowledgments

I am extremely grateful to:

- My teachers in school, who helped me to acquire good command over spoken and written English language.
- My lecturers and professors in medical college, who taught me the science and art of bedside cardiology.
- My heart patients, whose findings on clinical examination and results of investigations made me wiser.
- Learned authors of textbooks on clinical cardiology to which I referred liberally, while preparing the manuscript.
- My esteemed readers of earlier books, whose generous appreciation and constructive criticism keep me going.
- M/s Jaypee Brothers Medical Publishers (P) Ltd., New Delhi, India, who repose their unflinching faith in me and provide excellent editorial support.

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SECTION

1

Congenital Heart Diseases

CASE

1 Ventricular Septal Defect

Case Presentation

A 31-year old man was referred to the cardiologist by a general physician, for evaluation of a heart murmur. This young man had been denied a life insurance policy because the physician, empanelled by the insurance company, had incidentally noticed the murmur during medical examination. The man was normally very active and denied complaints of chest pain, breathlessness, palpitations or syncope. There was no history of cyanotic spells, joint pains or repeated chest infections during childhood and he regularly played cricket and football in school. However, the patient recollected that the doctor in the school medical room had noticed the murmur and made a note of it in his medical report.

On examination, the man was of average built and height and looked healthy. The pulse was 84 beats/min. and regular with no special character. The BP was 134/76 mm Hg in the right arm while sitting. There was no anemia, cyanosis or clinical sign of congestive heart failure. The apex beat was ill-sustained, heaving in nature and slightly displaced towards the axilla. There was a pansystolic murmur over the middle of the left sternal border with a S_3 sound in early diastole. The murmur did not radiate towards the axilla. There was no parasternal heave and the lower border of the liver was not palpable. The lung fields were clear.

CLINICAL DISCUSSION

From the history and physical examination, this asymptomatic young man had a parasternal pansystolic murmur. Typical causes of a pansystolic murmur are mitral regurgitation, ventricular septal defect and tricuspid regurgitation. Sometimes, tight coarctation of aorta or a patent ductus arteriosus with pulmonary hypertension can also produce a pansystolic murmur but these murmurs are usually located at the upper left sternal edge. The murmur of mitral regurgitation radiates towards the axilla while the murmur of tricuspid regurgitation is usually associated with engorged neck veins and an enlarged pulsatile liver.

ECG of the patient showed biphasic RS complexes in the mid-precordial leads. X-ray chest showed mild cardiomegaly with minimal signs of pulmonary congestion. On ECHO, the left ventricle was normal in size with normal ejection fraction. A signal drop-out was noticed in the mid-portion of the interventricular

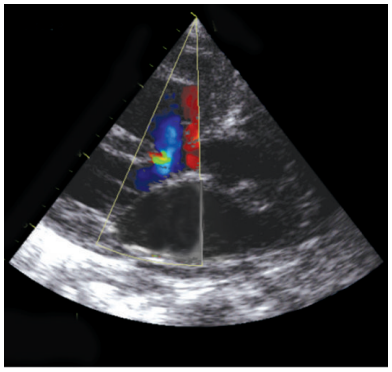

Figure 1.1: Color flow map extending from left ventricle to right ventricle

septum. There was no abnormality of the cardiac valves and the estimated pulmonary artery pressure was normal. On color Doppler, an abnormal flow map was observed extending from the left ventricle to the right ventricle (Fig. 1.1), with a high velocity jet on continuous wave Doppler. Therefore, the definite diagnosis in this case is ventricular septal defect (VSD).

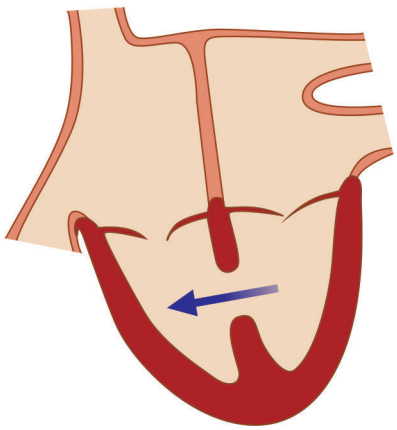

Figure 1.2: Ventricular septal defect

In VSD, a breach in the continuity of the interventricular septum creates a left-to-right shunt between the ventricles (Fig. 1.2). This congenital cardiac defect occurs due to complexity of embryological development of the septum, which has a membranous and a muscular portion. Most (80%) VSDs occur at the junction of these sections and are termed as perimembranous VSD (Fig. 1.3). Some VSDs occur in the muscular section (muscular VSD) and may be multiple (sieve-like). Rare varieties of VSD are endocardial cushion defects (supracristal VSD) and outlet septal defect (subpulmonic VSD) (Table 1.1).

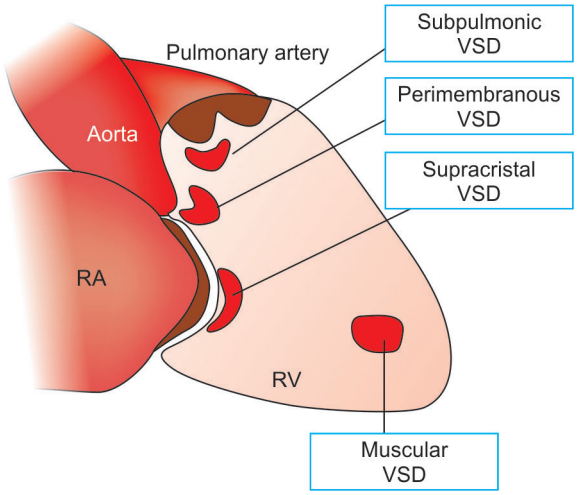

Figure 1.3: Various locations of ventricular septal defect (VSD)
RA: Right atrium; RV: Right ventricle

Table 1.1: Types of ventricular septal defect

- Perimembranous VSD
- Subpulmonic VSD
- Supracristal VSD
- Muscular VSD

A small VSD (Maladie de Roger) generates a loud pansystolic murmur in a localized area on the precordium. The murmur is located in the upper parasternal area in outlet VSD and in the mid-portion in perimembranous VSD. A muscular VSD produces a short systolic murmur since the defect shuts off during muscle contraction in later systole. This murmur is located over the lower parasternal area. A large VSD with elevated right ventricular pressure that equals left ventricular pressure (bidirectional shunt) is also associated with an early systolic murmur. Therefore, there is no correlation between the length or intensity of the murmur and the size of the VSD.

A large shunt may be accompanied by a diastolic flow murmur and a S_3 sound, due to torrential flow across the mitral valve. The S_2 is widely split due to early aortic valve closure. On ECHO, signal drop-out is not observed if the VSD is too small (<3 mm size) or muscular in location. The width of the colour flow map approximates the VSD size. On Doppler, high flow velocity indicates a small VSD. The flow velocity is low if the VSD is large and the shunt is bidirectional.

VSD is the commonest form of congenital acyanotic heart disease and accounts for 25% of all cardiac malformations. VSD may occur in isolation or as part of a complex constellation of congenital cardiac abnormalities. Aortic regurgitation may be associated due to lack of support to the aortic valve in perimembranous

VSD. Complications of VSD in childhood are growth retardation and repeated chest infections. Reversal of shunt can occur later in life when pulmonary pressure exceeds the systemic pressure. Endocarditis can follow any non-cardiac surgical procedure.

MANAGEMENT ISSUES

Large sized VSDs allow large volumes of left-to-right shunt and usually present in childhood with failure to thrive, breathlessness and recurrent respiratory infections. They can lead to pulmonary hypertension, right heart failure and ultimately reversal of shunt (right-to-left). This is designated as the Eisenmenger's syndrome. Such VSDs are usually closed in childhood to avoid complications and before the Eisenmenger's syndrome has developed.

Medium sized VSDs are associated with a moderate sized shunt. The shunt is large enough to cause breathlessness, but not enough to cause pulmonary hypertension and shunt reversal. Such patients do reasonably well during childhood, but may become progressively symptomatic as left ventricular compliance declines with age and pulmonary venous congestion develops. Such VSDs are usually closed in adulthood, to avoid the development of heart failure.

Small sized VSDs do not cause significant shunting and are often asymptomatic. Some of them may close as the child grows older. Those that do not close spontaneously are closed by intervention for reasons other than the shunt. These reasons are development of endocarditis or associated significant aortic regurgitation (Table 1.2).

Table 1.2: Indications for surgical closure of VSD

• Large-sized VSD with volume overload (pulmonary to systemic flow ratio >2:1)
• Medium-sized VSD with congestive symptoms without pulmonary hypertension
• Small-sized VSD without congestive symptoms with endocarditis or aortic regurgitation

RECENT ADVANCES

The last decade or two have witnessed remarkable progress in the percutaneous techniques for closure of ventricular septal defects, thus avoiding the risks associated with open heart surgery. Although transesophageal echocardiography (TEE) generally suffices to guide the deployment of the closure device, intracardiac ultrasound provides more accurate assessment. Sonography can provide vital information pertaining to the location and size of the defect and the rim around it, so as to facilitate proper device selection and placement.

CASE

2

Atrial Septal Defect

Case Presentation

A 36-year old woman was referred to a physician by a gynecologist, for preoperative assessment prior to elective hysterectomy. The patient had multiple uterine fibroids on ultrasonography and complained of excessive bleeding during menstruation. For the past 6 months, she had been complaining of exertional dyspnea and fatigue, which were attributed to anemia as a result of blood loss. She denied complaints of chest pain, palpitations or dizziness. There was no history of cyanotic spells, joint pains or recurrent respiratory infections during her childhood. The patient was married, had 2 sons aged 11 and 9 years and she had never been hospitalized for any major illness or surgical procedure.

On examination there was mild anemia but no cyanosis, icterus or sign of congestive heart failure. The pulse was 90 beats/min. regular, with a BP of 136/80 mm Hg in the right arm. The apex beat was normal in location with a sustained left parasternal heave on palpation. The S_1 was normal with a loud P_2; no S_3 or S_4 sound was heard. The S_2 components namely A_2 and P_2 were widely spaced and the time gap between them did not increase further during inspiration. A short systolic murmur was heard over the upper left sternal border. The murmur was not preceded by an ejection click or accompanied by a palpable thrill and did not radiate to the neck. The lung fields were clear on auscultation.

CLINICAL DISCUSSION

From the history and physical examination, this young woman had effort intolerance with an ejection murmur in the pulmonary area. Typical causes of such a murmur are innocent hemic murmur (Still's murmur), pulmonary valve stenosis, pulmonary hypertension and atrial septal defect. The murmur of pulmonary stenosis may be preceded by an ejection click and accompanied by a palpable thrill. The P_2 component of S_2 is muffled and the splitting between A_2 and P_2 is wide, but widens further during inspiration. An innocent hemic murmur is not associated with a loud P_2 or wide splitting of S_2. Pulmonary hypertension of any etiology can produce a systolic murmur with loud P_2 but wide fixed splitting of S_2 is only a feature of atrial septal defect.

ECG of the patient showed sinus rhythm with incomplete right bundle branch block and a rightward QRS axis. X-ray chest showed enlarged right-sided

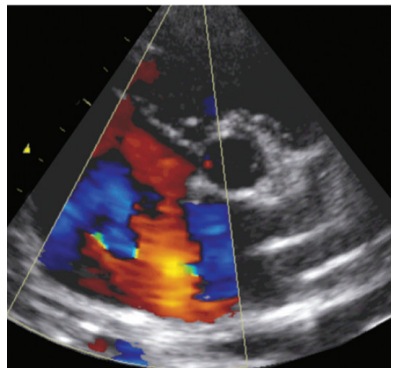

Figure 2.1: Color flow map extending from left atrium to right atrium

chambers with dilated main pulmonary artery, prominent hila and pulmonary plethora. On ECHO, the right atrium and right ventricle were dilated and a signal drop-out was noticed in the interatrial septum. On colour Doppler, an abnormal flow map was observed extending across the area of echo drop-out, from the left atrium to the right atrium (Fig. 2.1). There were no abnormalities of the cardiac valves and the estimated pulmonary artery pressure was normal. Therefore, the definite diagnosis in this case is atrial septal defect (ASD).

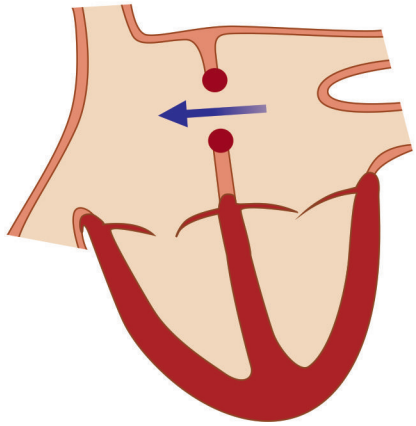

Figure 2.2: Atrial septal defect

In ASD, breach in the continuity of the interatrial septum creates a left-to-right shunt between the atria (Fig. 2.2). The septal defect occurs due to complexity of its embryological development. Most (75%) ASDs occur in the mid-portion of the septum, in the region of the foramen ovale and are termed as ostium secundum ASD. Some ASDs occur lower down the inter-atrial septum and are termed as ostium primum ASD (Fig. 2.3). Ostium primum ASDs are associated with cleft leaflets, regurgitation of the atrioventricular valves and are also known as endocardial cushion defect. An uncommon variety of ASD in the upper portion

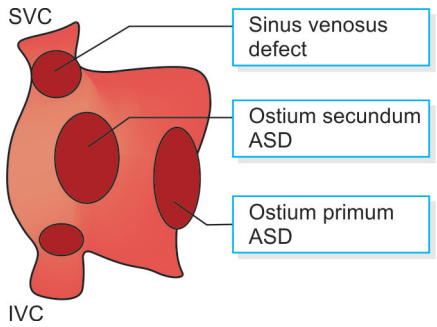

Figure 2.3: Various locations of atrial septal defect (ASD)
SVC: Superior vena cava; IVC: Inferior vena cava

Table 2.1: Types of atrial septal defect

- Ostium secundum ASD
- Ostium primum ASD
- Sinus venosus defect
- Vena caval defect

is sinus venosus defect, which is accompanied by anomalous pulmonary venous connections (Table 2.1). Inferior vena caval defects are very rare. An ASD may be associated with trisomy 21 (Down's syndrome) or abnormalities of the hand (Holt Oram syndrome).

The systolic murmur of ASD is due to increased flow across the pulmonary valve and not due to the shunt. The intensity of murmur does not correlate with the size of the ASD. However, a large ASD is associated with a diastolic flow murmur and a right-sided S_3, due to torrential flow across the tricuspid valve. An accompanying pansystolic murmur due to mitral and/or tricuspid regurgitation is a feature of ostium primum ASD. In ASD, the splitting of S_2 is wide and fixed. It is wide because of increased pulmonary ejection time, which delays the P_2.

Other reasons for wide splitting of S_2 are right bundle branch block or pulmonary stenosis (delayed P_2) and mitral regurgitation or ventricular septal defect (premature A_2). The splitting of S_2 is also wide in WPW syndrome Type A, in which there is pre-excitation of the left ventricle. The splitting of S_2 is fixed in ASD because the shunt equalizes atrial pressures throughout the respiratory cycle and there is no inspiratory augmentation of right ventricular filling.

On ECHO, since the signal from the interatrial septum is weak, false echo drop-out may be seen even in normal persons. The subcostal window may be a better option to diagnose an ASD but transesophageal echocardiography (TEE) provides excellent visualization particularly in endocardial cushion defects and sinus venosus ASD. Sometimes, contrast echo is needed to visualize the shunt using agitated saline, which contains air bubbles that cross over the septal defect.

ASD is the commonest congenital heart disease diagnosed in adulthood, with either absent or mild symptoms. It is 7 times more common in females than