HEART DISEASE AS A CAUSE OF MATERNAL DEATHS*

by

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Published statistics from Western as well as Asian countries show that cardiac disease constitutes one of the major causes of maternal mortality. In a recent study of 377 consecutive maternal deaths at the L.T. Municipal General Hospital, over a 6 year period (1961-66), heart disease was diagnosed in 26 instances (approximately 7% of the total maternal deaths). (D'Cruz *et al*, 1967). For the purpose of this study, maternal deaths included all deaths occurring during pregnancy or within 3 months of the termination of pregnancy, following the definition of the Committee on Maternal and Child care of the Council on Medical Service of the American Medical Association, (Klein *et al*, 1958).

This paper contains a brief description and analysis of the 26 maternal deaths due to heart disease and also a review of maternal mortality data from Indian as well as western centres, with special reference to cardiac deaths.

Rheumatic heart disease, being the most important form of heart disease encountered in pregnant women, will be analysed and discussed in greater detail than the other cardiac causes of maternal death.

Rheumatic Heart Disease

Rheumatic heart disease accounts for 80-95% of all cardiac disease in pregnancy in some centres in India (Panjabi, 1965; Sood and Padmavati, 1965) as well as most centres abroad, (Burwell, 1958, Sutherland and Bruce, 1962, Gilchrist, 1963, and Mendelson, 1960). It is one of the principal causes of death in women of child-bearing age. Thus rheumatic heart disease caused 19.6% of all deaths in the 11-40 age group in the medical wards of a Women's Hospital in Delhi, (Padmavati and Kumari, 1967). At the L.T.M.G. Hospital, Sion, there were 17 maternal deaths associated with rheumatic heart disease during 1961-66, constituting 4.5% of all maternal deaths over this period.

Age

Of the 17 fatal cases, 3 were in the 16-20 age group, 4 in the 21-25 group, 6 in the 26-30 group, 3 in the 31-35 group, and 1 in the 41-45 group.

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* *Part of this paper is based on work done for M.D. dissertation by one of the authors. (S.M.G.).*

Received for publication on 30-10-67.

Parity

One woman was primiparous, one was para 2, 7 were para 3, 4 were para 4. In 4 instances, the parity was unrecorded.

Antenatal attendance

Ten of the 17 fatal cases were emergency admissions, with no antenatal care whatever. Of the 7 women who had attended our antenatal clinic, 3 had attended it for less than 1 months; only 4 had adequate or near-adequate antenatal care.

Type of valvular lesion

Fifteen patients had mitral stenosis, one had mitral regurgitation, and one had both stenosis and regurgitation of the mitral valve.

Radiological evaluation of heart size

In four fatal cases adequate radiological data were not available. All the other 13 patients had cardiomegaly; in 2 it was classified as mild, in 5 it was moderate, and in 6 severe.

Electrocardiography

E.C.G. data were available in 9 instances of mitral stenosis. Right ventricular hypertrophy was present in all 9 cases; it was moderate in 4 and severe in 5. The single patient with pure mitral regurgitation showed left ventricular hypertrophy.

Atrial fibrillation was observed in 2 patients. One patient with mitral stenosis and atrial fibrillation was first seen at the eighth month of pregnancy, in severe congestive failure. Her cardiac failure was treated intensively with digitalis and diuretics, and 3 days after admission the ventricular rate had slowed to a satisfactory extent. Unfortunately, after that, there was an uncontrollable rise in heart rate and the patient died. The other patient had been seen at the fourth month of her pregnancy in frank cardiac failure. At this time she was in sinus rhythm and the failure responded to routine measures. This improvement was maintained for the next 3 months, during which time she was regularly observed at the antenatal clinic. During her seventh month, however, she suddenly became very breathless and was admitted in pulmonary oedema. The E.C.G. showed that she was now fibrillating, and x-ray chest revealed the typical appearance of pulmonary oedema. There was very little response to therapy and she died 2 days after admission.

Effort tolerance

Three patients were classified as Class II, 5 as Class III and 9 as Class IV, according to the New York Heart Association Classification, (New York Heart Association, 1964).

Type of delivery

Spontaneous normal delivery occurred in 7 patients, forceps were used in 2, and cesarean section was performed in one. These procedures were carried out for obstetric, rather than cardiac reasons. Spontaneous abortion occurred in 2 women. Therapeutic abortion was not performed in any. Five women died with the foetus in utero.

Stage of pregnancy or puerperium at time of death

Of the 5 women who died undelivered, two were in the second

trimester and three in the third trimester. No deaths occurred during labour. Of the 12 post-partum deaths, 2 occurred during the first 24 hours, 1 on the fifth post-partum day, and the remaining 9 deaths between the thirtieth and ninetieth day after delivery.

Duration of hospital stay

Two patients died within the first 6 hours, 3 between 6 and 24 hours, 3 between 24 and 72 hours, 4 between 4 and 7 days, and 5 between 7 and 21 days. Thus approximately half the patients died within the first 3 days, and this is an indication of their grave (often moribund) condition on admission.

Bacterial endocarditis was the cause of death in one patient. She had had an abortion terminating a 6 months' gestation 6 days prior to admission, and since then had been suffering from fever and bleeding per vaginam. Typical findings of mitral stenosis were noted on examination. High fever and tachycardia persisted in spite of intensive antibiotic therapy (Chloramphenicol 2 gms. penicillin 8 million units, and streptomycin 1 gm. daily). She rapidly became anaemic, the haemoglobin falling from 11 gms% to 4.5 gms. in a couple of weeks. Blood culture yielded Klebsiella pneumoniae. The cardiac failure became progressively more severe, and she died 3 weeks after admission. At autopsy, the heart showed considerable hypertrophy of the right ventricle and large bacterial vegetations on the stenotic mitral valve. The lungs were oedematous and showed 2 small infarcts. Septic infarcts were seen also in both kidneys and the spleen. Free fluid was present in both pleural cavities and in the peritoneum.

Cerebrovascular accidents

One patient had a spontaneous abortion during the fifth month of her pregnancy, and mitral valvular disease had been diagnosed at that time. Ten days later, she was readmitted with a sudden onset of vomiting and coma. The left upper and lower limbs were found particularly flaccid and immobile, and the left nasolabial fold obliterated. The pulse and blood pressure were unrecordable, and she expired after a few hours. No autopsy was performed. Though the cerebrovascular accident was probably embolic, cerebral venous thrombosis could not be excluded. Another patient was admitted 15 days post-partum with a history of fever, backache and vomiting since the previous day. She had been admitted earlier, during her pregnancy, for congestive heart failure due to mitral stenosis. Seven days after admission she developed right abducent nerve paralysis. She died the next day. Autopsy was not done. The cerebro-spinal fluid had contained 60 cells (lymphocytes 90%) and 200 mg. protein %. The possible diagnoses include cerebral venous thrombosis and tuberculous meningitis.

Pulmonary embolism

This patient had delivered 12 days previous to admission. She had fever and left-sided hemiplegia, and classical signs of mitral stenosis, but no signs of cardiac failure. Three days after admission, she suddenly developed chest pain, dyspnoea, restless-

ness and fall in blood pressure. This death was probably due to pulmonary embolism, the preceeding hemiplegia having been caused by cerebral embolism, with cerebral venous thrombosis as a less likely possibility.

Congestive heart failure

Five patients died in typical congestive failure. Three of them were emergency admissions, and all died within a day of admission. The fourth patient had attended antenatal clinic for 4 months, had been in mild cardiac failure, and was on maintenance doses of digitalis and hydrochlorothiazide. She became post-mature, and cesarean section was performed in the forty-second week. Post-operatively, the patient rapidly passed into cardiac decompensation and died the same day. The fifth patient had aborted 8 days prior to admission. She was in severe congestive failure, and there was a history of similar episodes during the previous two pregnancies. She died 6 days after admission. One additional patient with mitral stenosis had delivered normally and been discharged without any signs of cardiac decompensation. However, 8 days after delivery, she was re-admitted in severe heart failure, and was found dead on arrival in the ward. Autopsy revealed tight mitral stenosis, with generalised anasarca and passive venous congestion.

Pulmonary oedema, rather than classical congestive failure, characterised the terminal illness in 3 patients with mitral stenosis. All 3 presented with severe dyspnoea, orthopnoea, tachycardia and cyanosis. One also had haemoptysis. Two of them had been attending antenatal clinic and had been on maintenance doses of digoxin and hydrochlorothiazide, but one of them had discontinued these drugs on her own.

Discussion

Maternal mortality due to heart disease is shown in Table I (various

TABLE I

Data of Maternal Mortality in India, with special regard to Cardiac deaths

Author	Years studied	Total pregnancies or deliveries	Total cardiac cases	Total maternal deaths	Cardiac maternal deaths	Cardiac deaths	
						% Cardiac cases	% Maternal deaths
Shastrakar & Devi, (1962) Nagpur.	1952-60	14,564	..	563	19	..	3.45%
Devi, (1957), Visakhapatnam.	1950-56	15,000	107	181	13	12%	..
Kirlosker, (1962), Hyderabad.	1958-61	33,553	..	152	16	..	10.5%
Motashaw and Jadhav, (1960), Bombay.	1953-57	47,813	..	218	11	..	5.04%
Bombay Municipal Report	1952-54	..	..	229	12	..	5.25%
Sachdev, Wagh and Dodeya, (1961), Jubblpore.	1958-60	12,132	56	..	5	8.9%	10.00%
Panjabi, (1965), Bombay	1961-63	24,123	60	..	2	3.7%	3.3%
Masani K. M., (1957), Bombay.	1953-56	22,833	47	..	3	6.4%	..
Sood and Padmavati, (1955), Delhi	1958-63	28,068	275	..	11	0.9%	4.0%
Present Series, Bombay	1961-66	37,875	..	377	26	..	4.5%

Indian Series) and Table II (various Western series). In some of these series the cardiac deaths are expressed as a proportion of the total cardiac cases, while in others (maternal mortality studies) they are expressed as a proportion of the total maternal deaths. In some series, the number of deaths due to rheumatic heart disease was not specifically stated.

It will be seen from Table II that the mortality from heart disease in

TABLE II

Data of Maternal Mortality in Western Countries, with special regard to cardiac deaths

Author	Years studied	Total pregnancies or deliveries	Total cardiac cases	Total maternal deaths	Cardiac maternal deaths	Cardiac deaths. % Cardiac cases	Cardiac deaths. % Maternal deaths
Gilchrist, (1963)	1948-60	..	1017	..	..	0.4%	..
Burwell, C.S., (1958)	6 yrs.	298	277	..	..	..	..
O'Driscoll, Barry and Drury (1957).	4 yrs.	289	..	..	1	0.34%	..
O'Driscoll, Coyle and Drury, (1962).	8 yrs.	539	385	..	7	1.3%	..
Gilbert, Sullivan and McLaughlin, (1957).	1945-54	17,128	272	..	2	0.72%	..
Sutherland and Bruce, (1962).	1949-59	500	379	..	7	1.4%	..
Burnim & Appel, (1957)	1934-48	..	205	..	3	1.46%	..
Jewett, (1957)	1941	..	..	247	22	..	8.9%
	1954	..	..	55	4	..	7.3%
Barnes, (1965)	1947-60	69,264 Del.	915	51	4	..	7.8%
	1961-63	17,855 Del.	122	..	1	..	..
Carpenter and Bryans, (1965).	1955-62	..	..	145	9	..	6.2%
Philips, Todd and Davis, (1953).	1936-58	455,553	..	730	56	..	7.7%
Dalziel, (1958)—							
Minnesota	1950-55	..	..	100	..	..	..
Franklin	,,	..	..	97	13	..	13.4%
Philadelphia	,,	..	..	98	10	..	10.2%
Illinois	,,	..	..	117	10	..	8.5%
Michigan	,,	..	..	100	8	..	8.0%
Toronto	,,	..	..	100	5	..	5.0%
Llorees, Griner and Thompson, (1963).	1949-61	80,403	..	97	5	..	5.15%
Barno, Freeman and Baker, (1963).	1950-59	..	..	493	26	..	7.00%
Maternal Deaths in North Ireland, (1965).	1060-63	132,104	..	95	13	..	13.07%
Lane and Andelman, (1966).	1956-60	..	..	232	34	..	14.5%
Maternal Deaths–England and Wales, (1966).	1961-63	..	..	936	81	..	8.65%
Bromwell and Longson, (1938).	..	..	350	..	26	7.4	..
			312 Rheumatic	..	22	7.05	..
Hunt, (1926)	..	..	156	..	17	10.9%	..
Watson, (1933)	..	..	240	..	17	7.0%	..
Mendelson, (1960)	1932-58	..	3690	..	31	0.8%	..
			3353 Rheumatic	..	23	0.69%	..

western countries has fallen considerably over the last 30 years, and recently some large series of cardiac patients consisting of several hundred pregnant women included only one or two deaths. (Sutherland and Bruce, 1962; Gilchrist, 1963; Burwell, 1958; O'Driscoll, 1962; O'Driscoll *et al*, 1957; Burnim and Appel, 1957).

In India the mortality is higher, varying from 1 to 15%. Padmavati and Sood's series should not be considered as comparable with the others, because they did not follow most of their pregnant cardiacs until delivery and puerperium. The factors that may influence the prognosis are:

1. *Age:* Older women between 35 and 45 years are supposed to have a worse prognosis than younger women, because the natural history of rheumatic heart disease is such that older patients are more likely to have advanced cardiac damage, with greater incidence of atrial fibrillation. There was no striking relation between age and mortality in our series. We had only one fatal case over 35 years of age.

2. *Parity* does not have an important bearing on prognosis. It has been claimed that women with very severe heart disease will not survive the first pregnancy, thus producing a high mortality in primiparae; those women who are able to safely cross the hurdle of the first pregnancy will probably be in a fit condition to survive one or more subsequent pregnancies. We, however, had only one primipara death, with 7 and 4 deaths respectively in third and fourth paras.

3. *Antenatal attendance:* This seems a very important cause of mortality in our Indian hospital class patients. Public hospitals in our country have a very large proportion of emergency obstetric admissions; such women never attend ante-natal clinics and usually seek admission only when delivery is imminent or when they are suffering from a serious complication such as cardiac failure or pulmonary oedema. Not unexpectedly, the mortality rate tends to be higher among such emergency admissions than in "booked" cases. Even among those Indian women who attend antenatal clinic, many do not attend regularly, do not take drugs such as digitalis regularly, or do not attend hospital at an early stage of deterioration. This partly accounts for a relatively high mortality in patients who are supposed to have received antenatal care. The importance of adequate antenatal care in detecting and treating heart disease cannot be overemphasised, and lack of antenatal attendance is a completely preventable factor in prognosis, unlike most other factors.

4. *Type of valvular lesion*—Mitral stenosis, being more common in females, accounts for 80% or more of rheumatic heart disease in most series. The type of valvular lesion is not as important as the severity of the lesion, in any particular patient.

5. *Effort tolerance:* This is considered the best clinical guide to the severity of the cardiac lesion and to the outcome of the pregnancy as far as survival of the mother (and also foetus) is concerned. Thus our fatal

cases contained a preponderance of Class III and IV patients, while in any clinical series of pregnant cardiacs, the vast majority are Class I and II.

Hamilton and Thomson (Hamilton & Thomson, 1941) classified the patients at the Boston Lying-in-Hospital as 'unfavourable' if there was a history or signs of cardiac failure, atrial fibrillation or serious complicating disease, and 'favourable' including all other cases. The maternal mortality in their unfavourable group was 17% and in their favourable group 3%. It frequently happens that a favourable case corresponding to Class I or II (of the New York Heart Association) early in pregnancy becomes an unfavourable case (Class III) later in pregnancy. Such deterioration occurred in 20% of those originally classified as favourable by Hamilton and Thomson.

6. *The size of heart:* The heart size as estimated *radiologically* and the degree of right ventricular hypertrophy as estimated *electrocardiographically* are both good prognostic criteria. In a patient with mitral stenosis, the heart size depends mainly on right ventricular and right atrial enlargement, and to a lesser extent on left atrial enlargement. Right ventricular hypertrophy and dilatation in turn are directly proportional to the severity of pulmonary hypertension. Severe pulmonary hypertension is always seen in association with a "critical" mitral stenosis and denotes raised left atrial pressure. Thus an enlarged heart and electrocardiographic findings of severe right ventricular hypertrophy are strongly suggestive of marked pulmonary hypertension and signify a worse prognosis.

Atrial fibrillation is undoubtedly an adverse factor in a woman with rheumatic heart disease. A few decades ago, it used to be stated that the mortality was so high in the presence of atrial fibrillation, that pregnancy should be terminated. Since then the mortality has fallen, though still high. Szekely (Szekely and Smith, 1961) has reported 50 pregnant cardiacs with atrial fibrillation, of which 3 expired. The onset of atrial fibrillation in a woman who was in sinus rhythm earlier in pregnancy carries a graver prognosis than long-standing atrial fibrillation which ante-dated the pregnancy.

Type of delivery: Normal spontaneous delivery carries the best prognosis. The present trend is to perform obstetrical procedures such as forceps and caesarean section if there are obstetrical grounds for such intervention but not for heart disease per se. The mortality rate is higher in patients who have been subjected to such procedures; we had 3 such cases in our series.

Stage of pregnancy: It is generally agreed that the burden imposed by pregnancy on the heart is maximum in the third trimester, especially about the thirty-second week, and most deaths might be expected at this period. However, equally dangerous is the post-partum period, during which 12 of our 17 patients died. This was realised 80 years ago by the British physician, Angus MacDonald (MacDonald, 1888).

Mode of death: Congestive cardiac failure is the commonest mode of death in all series. Bramwell (Bram-

well, and Longson, 1938) and Morgan Jones (Jones, 1951) called attention to the fact that many pregnant women with mitral stenosis die in acute pulmonary oedema rather than in classical right-sided congestive failure. This happened in 3 of our cases. Such patients may deteriorate rapidly and die within a few hours, sometimes even before they can be attended to in hospital. They often show normal or slightly enlarged hearts on x-ray and do not have severe right ventricular hypertrophy on E.C.G. so that the attending physician may be lulled into a false sense of complacency.

Acute bacterial endocarditis occurred once in our series of fatal cases; presumably the route of infection was the genital tract.

Cerebral embolism occurred in probably 2 and possibly 3 of the patients. Pulmonary embolism probably was the cause of death in one case. These embolic phenomena were not verified since these cases did not come to autopsy.

Hypertensive Heart Disease

Excluding eclampsia and pre-eclampsia, 3 maternal deaths in our series were attributed to systemic hypertension. One patient was a primipara, aged 22, who was 8 months pregnant on admission. Blood pressure was 145/110 mm. Hg. Eight hours after admission she delivered a premature infant, and then collapsed into a shock-like state. She died 11 hours after delivery in spite of pressor drugs and other resuscitative measures. Autopsy revealed extreme hypertrophy and dilatation of the left ventricle. The lungs were oedematous, and the liver showed changes of chronic passive congestion. The marked left ventricular hypertrophy was thought to signify pre-existing hypertension of long duration, which might have been perhaps aggravated during pregnancy.

The second patient was a primiparous woman, aged 22, who had delivered 6 weeks previously. She had experienced symptoms of heart failure since delivery, and was found to have generalised anasarca, hepatomegaly, cardiomegaly and gallop rhythm. Blood pressure was 180/130 admission, and autopsy showed hydropericardium, bilateral pleural effusions, oedematous lungs, chronic passive congestion of the liver, and a dilated heart.

The third patient was a 28 year old woman, para 2, who had complained of cough, dyspnoea and chest pain for 3 days previous to delivery. She delivered a premature infant at home, and was admitted 6 hours later in cardiac failure. Blood pressure was 210/100 mm. Hg. in the upper limbs. Arterial pulsations were absent in both lower limbs, and a loud systolic bruit was heard in the epigastrium. Her heart failure responded to routine measures. Two weeks after admission, she died suddenly, a few hours after an unsuccessful attempt at aortography. Permission for autopsy was not obtained. The probable diagnosis was stenosing aortitis ("middle aortic syndrome").

Pulmonary Hypertension

Two women presented with the clinical signs of marked pulmonary hypertension. One was 30 year old, para 8, who was admitted with severe

right heart failure at the seventh month of pregnancy, and died undelivered. The other woman presented in right-sided congestive failure 8 days after delivery. Right ventricular hypertrophy was evident radiologically and electrocardiographically in both patients.

The main possible causes of such pulmonary hypertension in young women are (1) "Silent" tight mitral stenosis, (2) certain types of congenital heart disease, particularly atrial septal defect and (3) primary pulmonary hypertension. These three conditions can be differentiated only by cardiac catheterisation or autopsy, neither of which were performed in these 2 cases.

One 22 year old primiparous woman, admitted in severe right failure, apparently suffered from chronic cor pulmonale, and had a long history of cough and dyspnoea.

Supraventricular tachycardia

This arrhythmia was present in 2 women aged 26 and 30 respectively. These 2 patients were remarkably similar in that both delivered twins, both developed a regular tachycardia of about 210 per minute which began during labour and continued after delivery, and which did not respond to either digitalis or quinidine. These patients were seen before electrical conversion of arrhythmias had been introduced in Bombay; this technique is now the treatment of choice for drug-resistant ectopic tachycardias. Mendelson, in 1956, collected a total of 82 cases of paroxysmal supraventricular tachycardia from the literature, of which 4 were fatal (Mendelson, 1956).

Haemopericardium

This was the most unusual of our cardiac deaths. The patient was a 32 year old woman, para 5, who was 5 months pregnant when admitted. She complained of pain in the chest and back for 3 days, and had vomited just before admission. She was in a collapsed state, with pulse and B.P. not recordable. She was very breathless, restless and cyanosed. She died one hour after admission. At autopsy, the pericardial sac was distended with about 300 ml. of blood. There was no obvious pathology in the heart or in any other organs, to account for the haemopericardium.

Pperperal Cardiomyopathy

We had one patient who was diagnosed during life as rheumatic mitral valve disease but was found to have normal valves at autopsy. She was a 20 year old woman who had aborted 1 month before admission, terminating a 6 month gestation. She complained of progressively increasing dyspnoea for 15 days before admission. On examination, she was orthopnoeic and dyspnoeic at rest. The heart rate was 140 per minute, regular, and blood pressure 94/60 mm. Hg. An apical pansystolie and mid-diastolic murmur were audible. The liver was slightly enlarged and tender. Bilateral basal crepitations were present. She appeared to improve with routine treatment of cardiac failure, but died suddenly 14 days after admission. At autopsy, the heart weighed 300 gms. The endocardium and all the valves appeared normal. There was generalised dilatation of all chambers. No evidence

of rheumatic inflammation was seen histologically.

The aetiology of puerperal heart disease is obscure, although it is being increasingly recognised as a clinical entity (Walsh J. J. *et al*, 1965; Johnson *et al*, 1966; Meadows, 1960). It differs from other causes of heart failure in puerperal women in the fact that the myocardium rather than the heart valves or other anatomical structures is at fault. A controversy exists as to whether pregnancy and parturition have somehow a direct aetiological role to play in the pathogenesis of puerperal cardiomyopathy, or whether the cardiovascular burden imposed by pregnancy merely 'unmasks' a pre-existing asymptomatic myocardial disease.

Summary

Over a six-year period, there were 26 maternal deaths associated with heart disease at the L.T.M.G. Hospital, Bombay, comprising 4.5% of total maternal deaths.

Of these 26 patients, the 17 who had rheumatic heart disease are considered in detail. Congestive heart failure and pulmonary oedema were the most important causes of death in this group, but in a few cases some other complication such as cerebral embolism, pulmonary embolism and bacterial endocarditis was responsible.

Each of the nine other maternal cardiac deaths are very briefly described. These deaths were due to the following miscellaneous causes:

Systemic hypertension, pulmonary hypertension of undetermined etiology, cor pulmonale, paroxysmal tachycardia, haemopericardium and puerperal cardiomyopathy.

Acknowledgement

We are indebted to Dr. V. N. Panse, Dean Municipal General Hospital and Medical College, for permission to use the hospital records.

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