ACCIDENTAL HAEMORRHAGE ASSOCIATED WITH PULMONARY HYPERTENSION *

by

D. PARANJOTHY, M.R.C.O.G., M.B.B.S., D.T.M.,

Professor of Obstetrics and Gynaecology, C. M. C. Hospital, Vellore.

Accidental haemorrhage in heart disease is extremely rare. Many of the standard text-books in Obstetrics do not even make a reference to such an association. Johnstone, in his text book on Midwifery, casually mentions that accidental haemorrhage may sometimes be associated with some general diseases of the mother, such as heart disease or syphilis.

This case is reported because of the association of accidental haemorrhage with an unusual form of heart failure, right heart failure following pulmonary hypertension. The combination of these two conditions resulted in the unexpected death of the patient.

Case Report: D. aged 19 years; gravida I; (Hospital No. 24308) was admitted in C.M.C.H., Vellore on 21-4-'60 with the following complaints.

2. Amenorrhoea — 5 months.

2. Difficulty in breathing from childhood which used to increase on mild exertion such as walking or climbing.

Previous History: She has had an attack of tonsilitis at the age of 9 years. No history of rheumatic fever.

Family History: The patient was the eldest of three children. Two younger brothers are alive and well.

On Examination at the Time of Admission. The patient was a thin woman of 19 years. She was not anaemic, but had slight oedema of feet. The patient appeared extremely ill and breathless. Orthopnoea was present. There was no cyanosis or jaundice. Lungs — a few crepitations were present over the bases. In consultation with the Cardiologist, the following signs were elicited from the cardio-vascular system:—

The patient was very orthopnoeic with distended neck veins and slight oedema of feet. The individual venous waves could not be analysed. There was pre-cordial bulge with grade II left para-sternal lift. The apical impulse was of the right ventricular type and was felt in the 5th left intercostal space within the mid-clavicular line. There were no thrills either at the apex or over the base of the heart. The pulmonary 2nd sound, in other words a diastolic shock, was distinctly palpable over the pulmonary area.

On auscultation, the heart sounds were audible and the pulmonary 2nd sound was loud split and accentuated. No murmurs were heard.

Pulse 90 p.m. poor in volume, tension B.P. 100/80 mm. Hg.

E.C.G.:—Showed severe right ventricular hypertrophy pattern.

On Fluroscopy. There was moderate enlargement of the heart, particularly of the right ventricle, and the main pulmonary artery segment. Hilar clouding was present. The pulmonary arteries were large, and the periphery of the lungs appeared oligaemic, thus giving a coppicing effect.

X-ray. P. A. & R. A. O. confirmed the fluroscopic findings of pulmonary hypertension due probably to congenital defect or acquired heart disease. Cardiac catheterization could not be done because of orthopnoeic condition.

*Paper read at the 11th All-India Obstetric and Gynaecological Congress at Calcutta in January 1961.

The impression was pulmonary hypertension. In view of the history of breathlessness and palpitation since childhood and absence of rheumatic history, it was presumed that the pulmonary hypertension was probably due to: (a) atrial septal defect (commonest form of congenital cardiac lesion), (b) ventricular septal defect, or (c) primary pulmonary hypertension, though rare, not uncommon.

Treatment Given

1. Patient was digitalized and congestive failure was alleviated.
2. Priscolin, a vasodilator drug, was also tried.
3. Patient was advised absolute rest.

Investigations

Haemoglobin, 11.5 G%.

Urine—No albumin or sugar or caste. W.B.C.—8,100 per/c.mm. Differential count poly 63%, eosinophils 6%, monocytes 28%. Nucleated red cells 3%—blood group 'B'. Stool—No ova or cysts. Weight of the patient —85 lbs.

Progress: The patient had a slight rise of temperature at the time of admission which touched normal the next day and remained normal until death. Her pulse varied between 90 and 100. Blood pressure was 120/80. Her general condition improved with rest and the above treatment. She was seen frequently by the cardiologist. Exerything seemed to be going on smoothly, but on 17-7-1960, about 3 months after admission, the patient noticed slight vaginal bleeding at 6 p.m. She was transferred to the labour ward. The height of the uterus was 30 weeks and the foetal heart could be heard.

10 P.M.: The patient started to bleed profusely. Pethidine 100 mgm. was given. Blood transfusion of packed cells was started. Uterine contractions were present occurring every 10 minutes and lasting for half a minute. Foetal heart rate was 160.

Vaginal Examination: Cervix admitted 3 fingers. Presenting part was high. No placental tissue was felt. A diagnosis of accidental haemorrhage was made. Membranes were ruptured artifically and an abdominal binder was applied.

11 P. M.: Bleeding continued. It was decided to give pitocin drip to hasten labour. 2½ units of pitocin in 500 cc. glucose was started. Pulse 120. Bleeding continued and foetal heart was not audible. Patient delivered a still-born foetus, weight 1 lb. 6 oz., soon after mid-night. This was followed immediately by expulsion of the placenta and huge retroplacental clots. The patient had slight post-partum haemorrhage also. This was controlled by ergometrine, 0.25 mgm., intravenously. Blood pressure 100/70—pulse 130. Four hours after delivery, patient was returned to the ward. Her pulse was 120.

18-7-'60 (The next day). Patient vomited once. She complained of epigastric pain. Pulse—120, pethidine, 50 mgm., was given. Penicillin was given prophylactically.

2nd day. Patient awoke after a good night's sleep. There was no breathlessness, nausea or vomiting. Blood pressure—100/75, pulse—112. Lungs were clear. In the evening she complained of precordial and epigastric pain. Pethidine 50 mgm., was given.

3rd day (6 A.M.) Patient vomited and became cyanosed. Oxygen was started, pethidine, 50 mgm., was given. Pulse 110.

3 p.m. Vomited again. She became dyspnoeic. Cyanosis increased, oxygen was continued. The condition gradually deteriorated and the patient died at 9-45 p.m. that is less than 72 hours after delivery. Permission for post-mortem was not given.

Discussion

Through all the ages, physicians have feared, to a greater or lesser extent, the combination of heart disease in pregnancy. The general feeling is one of pessimism. It carries a serious risk to the mother. The commonest types of lesions are rheumatic heart disease and hypertensive heart disease. Very little attention has been paid to congenital heart disease in pregnancy.

Pouliot, in 1905, stated that marriage should be forbidden in cyanotic

patients. Hoch-singer believed that cyanosis was a good reason to terminate pregnancy. Even as late as 1945, Semisch held a view that pregnancy should not be continued in cyanotic congenital cases, while in 1933, Breed and White maintained that 'If a patient with congenital heart disease has reached the age for pregnancy, without intolerance to lead a normal life, she can go through pregnancy and deliver successfully and uneventfully.' Shapio and Simons in 1934 were even more optimistic and said that cyanosis in congenital heart disease is not per se contradiction to pregnancy.

Pardee in 1941 gave an outline about the management of congenital heart disease in pregnancy. He was optimistic. In 1950 Van der Veer and Kuo concluded that the prognosis in these cases is good unless it is associated with cyanosis, polycythemia and heart failure.

With the advent of cardiac surgery the outlook is completely changed. In suitable cases, surgery on the heart can be done even during pregnancy, provided the patient is seen before the 20th week. In our hospital, we have had 7 cases where surgery was done during pregnancy and pregnancy continued up to term.

In cyanotic congenital heart diseases too, surgery can be done and the results are encouraging. We had one case of Fallot's Tetrad, who was relieved by an anastomosis between the left subclavian and pulmonary artery. Eighteen months later, she conceived and had a full-term normal delivery. We feel confident that with open heart surgery, the outlook for congenital heart disease will improve.

Pulmonary hypertension can be either primary or secondary. Primary is extremely rare. According to Paul Wood, among 100 cases of chronic pulmonary heart disease, only 3 were idiopathic. It may occur in either sex.

Pulmonary hypertension is the dreaded complication of lesions like an atrial or ventricular septal defect with a large left to right shunt.

Pulmonary hypertension imposes a mechanical burden on the right ventricle. The ventricle hypertrophies and sooner or later, it fails. Fibrillation may develop later on. Cyanosis appears, and cardiac output becomes less. The arterial oxygen saturation depends on the shunt reversal.

Diagnosis. Pulmonary hypertension is not diagnosed until far advanced. It presents a picture of right ventricular failure. In the later stages, it is well nigh impossible to differentiate between various causes like mitral valvular disease, congenital cardiac defects and congenital primary pulmonary hypertension.

Prognosis is very serious in primary pulmonary hypertension. It is a progressive lesion and has no known cure. There is no surgical treatment. The patient usually lives for not more than 2 years after its recognition.

The prognosis in secondary pulmonary hypertension however varies with its aetiology.

Question of Termination of Pregnancy

The extra burden on the left ventricle due to pregnancy would tend to make the patient's condition worse. In all those lesions, where cardiac surgery is contra-indicated or not

possible, termination of pregnancy depends on the period of gestation and the condition of the patient.

Causes of Death in Heart Diseases in Pregnancy

We are all familiar with the common causes of death such as heart failure, pulmonary embolism, pulmonary oedema, pneumonia, and sub-acute bacterial endo-carditis; but we have not realised sufficiently the importance of accidental haemorrhage. This case is a reminder to us that in heart diseases, an accidental haemorrhage, un-associated with toxaemia, can occur suddenly and unexpectedly as a 'bolt from the blue' and take us unawares and snatch the patient from us.

Summary and conclusion

1. A case of accidental haemorrhage in pulmonary hypertension is reported.

2. Its etiology, diagnosis and prognosis are briefly discussed.

3. Pulmonary hypertension, whether primary or secondary, has a serious prognosis.

4. The great advances made in cardiac surgery in recent years have made the outlook of cardiac disease in pregnancy more optimistic.

References

1. Jorge Espino Velo & Dirson Castro Abreu: Amer. Heart Jour.; 51, 542, 1956.
2. Keith: Heart Disease in children.
3. Nadas: Paediatric Cardiology.
4. Pardee H. E. B.: Amer. Jour. of Obst. & Gyn.; 17, 255, 1929.
5. Paul Wood: Diseases of the Heart and Circulation; 2nd Ed., Eyre and Spottiswoode.
6. Vander Veer Joseph and Kuo P. T.: Amer. Heart J.; 39, 2, 1950.