POSTPARTUM PULMONARY HYPERTENSION

(A Case Report)

by

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Postpartum pulmonary hypertension is a less known condition but, it is now recognised as a distinct entity. In a number of women the onset of symptoms of pulmonary hypertension can be undoubtedly traced to a recent pregnancy and delivery. (Olley & Whitaker, 1967).

The aetiology of the condition is micro-thromboembolic phenomenon in lungs following pregnancy and delivery, but the mechanism by which the pulmonary hypertension becomes established is not clear. Once initiated, there is a progressive obliteration of the smaller arteries leading to a gradual rise in the pulmonary blood pressure. It is difficult to show that the women who develop postpartum pulmonary hypertension have been cases of primary pulmonary hypertension, but it is suggested that some of them could be. McCaffery and Dunn, 1964, observed that the primary pulmonary hypertension is considerably worsened because of pregnancy. Moreover, absence of clinical evidence of thromboembolism is notable in the majority of women with postpartum pulmonary hypertension.

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The symptoms usually manifest within few months of delivery. Breathlessness on exertion and fatigue are prominent. Angina on effort, syncope and congestive cardiac failure follow. Each subsequent pregnancy seems to worsen the condition. Pregnancy is not known to be influenced by the pulmonary hypertension. The symptoms invariably progress rapidly and may lead to death earlier than expected.

The diagnosis may be in doubt initially. A high rate of suspicion must be kept in women with dyspnoea following delivery. The x-ray of the chest reveals an enlarged heart, dilated pulmonary arteries, and prominent broncho-vascular markings especially in the proximal region. The electrocardiogram too reveals a right-sided hypertrophy and strain. A cardiac catheterization early in the disease clinches the diagnosis, and excludes the other aetiological factors of pulmonary hypertension, like mitral stenosis and inter-atrial or inter-ventricular defects.

The prognosis of postpartum pulmonary hypertension is not good, in spite of early therapeutic regime. Anti-coagulants offer some chance of slowing down the progress of the condition. Pregnancy should preferably be debarred to a patient who has pri-

mary or postpartum pulmonary hypertension, in view of the excessive risk (30 to 50%) to her life subsequent to the delivery. Termination of pregnancy in a known case may be of value to avoid future hazards, provided it is done within 8 weeks of pregnancy. No clear experience in this respect is, however, documented.

A case of postpartum pulmonary hypertension is reported to support the scant literature so far published.

Case Report

Mrs. A. S., had her first full-term normal delivery at the age of 22 years, in August 1965. The pregnancy, labour and the puerperium were uneventful. At a 6 weeks' follow-up examination, there was no complaint.

From October 1965, mild dyspnoea on exertion, cough and low fever were apparent to the patient. For this, she took non-specific treatment in the form of tonics. In February 1966, a physician was consulted for the above complaints. A urine analysis indicated a moderate degree of u. fection, and blood examination leucocytosis of 13,200 cells per c.mm. increased polymorphonuclear leucocy. The E.S.R. was 33 mm. (Wintrobe method) with a packed cell volume of 38. The urinary infection was treated with nitrofurantin for 3 weeks. Electrocardiograms in early February and in April 1966, indicated non-specific T wave and S-T changes in V1, V2, & V3R (Fig. 1). An x-ray examination of the chest in February 1966 in P.A. (Fig. 2), R.A.O., and left lateral views showed a normal cardio-thoracic ratio. There was no enlargement of the cardiac chambers. The lung fields did not show any sign of congestion, and the pulmonary vascular markings were within normal limits. There was no evidence of tuberculosis or pleural effusion. A cardiac catheterization was thought of, but was deferred.

In May 1966, the patient conceived again and during the pregnancy she continued to have mild dyspnoea on exertion. Though there was no detectable cardiac or respiratory lesion, she was graded functionally as grade 2. (American Cardiac Association Classification), and advised extra rest in the afternoons. Besides, she received hae-

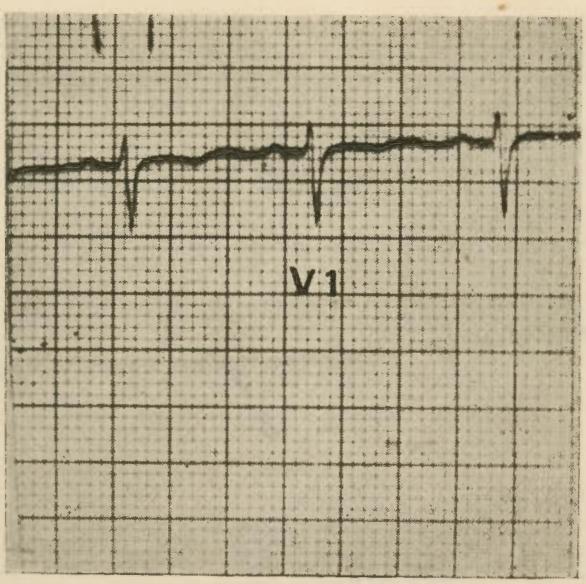

Fig. 1a
E.C.G. slightly depressed S. T. segment.

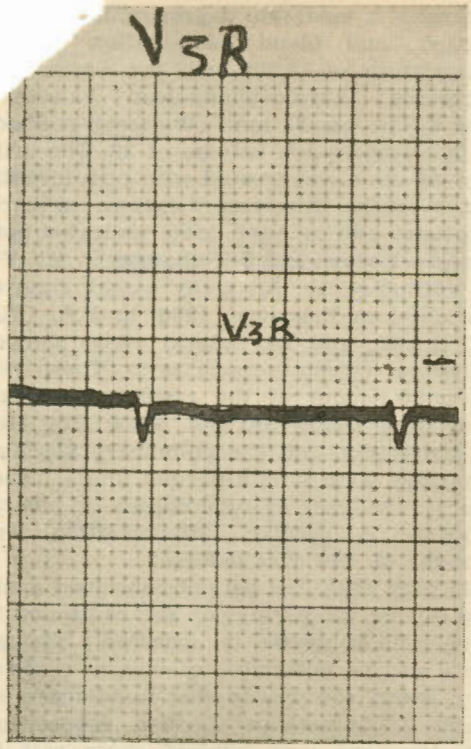

Fig. 1b
E.C.G. normal V_3 R lead

matinics. Throughout the second pregnancy and the delivery she had been cared for by her family doctor and her obstetrician. No need for a physician's consultation had arisen. A full-term normal delivery resulted in January 1967, and a 6 weeks postnatal check-up showed nothing unusual.

A month after the postnatal visit, the patient complained of increasing dyspnoea on exertion and cough. Tonics and haematinics were tried with little success.

In April 1967, she consulted her physician who repeated the E.C.G. to detect, for the first time, right ventricular strain with right axis deviation and inverted T wave and depression in S-T segment in leads V1 to V4 and V3R. (Fig. 3). The x-ray of the chest then revealed an enlarged heart with a prominent pulmonary conus. The proximal pulmonary arteries were markedly enlarged (Fig. 4). A diagnosis of postpartum pulmonary hypertension, due to microembolic phenomenon, with right ventricular failure was made. On auscultation of her heart, pulmonary second sound was marked; a systolic murmur in the pulmonary area and a short presystolic murmur in the paramitral area were heard. The liver was palpable but there was no frank congestive cardiac failure.

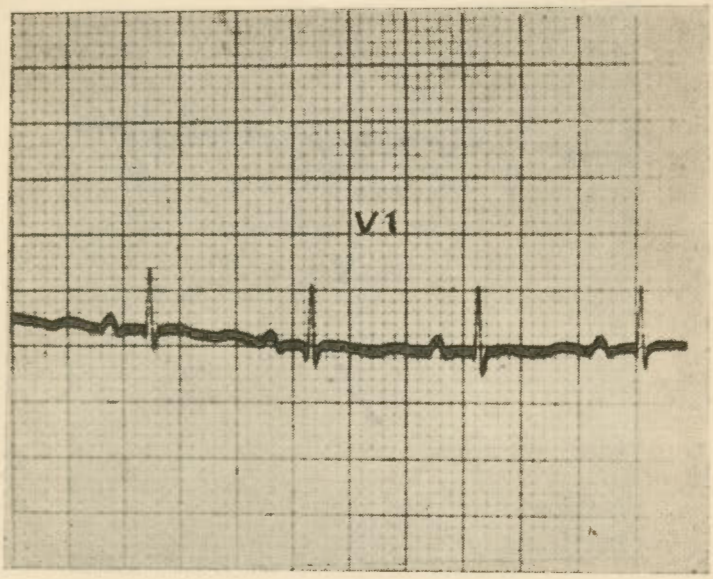

Fig. 3a
E.C.G. altered Q. R. in V_1 indicating right ventricular hypertrophy and strain pattern.

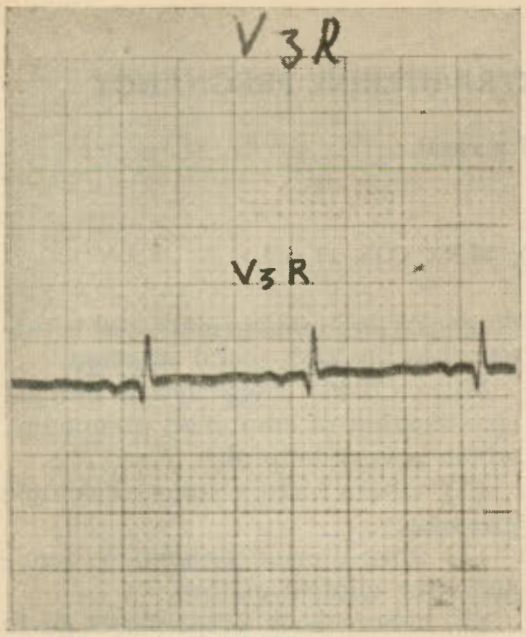

Fig. 3b
E.C.G. altered Q. R. in V_R R indicating right ventricular hypertrophy 6 strain pattern.

The patient was put on the following drugs: Lanoxin, Papaphylon, Uniwarfarin, Furamid, and tonics. The patient, in spite of regular medication, rest and medical supervision, went down rapidly and developed congestive cardiac failure. She expired due to it in November 1967.

Comments

The case reported was not known to be suffering from primary pulmonary hypertension. Her symptoms manifested after the first delivery. They rapidly progressed after the second, to a fast fatal state. It cannot be explained why the condition remained clinically almost status quo throughout the second pregnancy. It is probable, that, had the second pregnancy not occurred, the patient would have lived longer. Anti-coagulant therapy did not alter the fulminating advancement of her disease.

McCaffrey and Dunn have reported a case of primary pulmonary hypertension who conceived five times in presence of the disease, and she improved symptomatically during each of her first four pregnancies. Death, however, occurred in the fifth one.

References

1. McCaffrey, R. M. & Dunn, L.: J. Obst. & Gynec. Surv., 19: 567, 1964.
2. Olley, P. M. & Whitaker, W.: Obst. & Gynec., 29: 369, 1967.

Figs. 2 & 4 on Art Paper XI