PULMONARY AND HEPATIC INFARCTS

(A Case Report)

by

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Pulmonary embolism as an aetiological factor of maternal mortality has been established for quite some time. These were the cases, which were passed off as cases of obstetric shock without a post-mortem examination. Sheehan (1958), in his series of 147 cases of obstetric shock, did not find pulmonary embolism in any case. The importance of a post-mortem examination cannot be over-emphasised. Autopsy often reveals hitherto unsuspected and rare conditions not realised as causes of maternal death. Hepatic infarction, in human beings, is quite a rare entity, more so in obstetric practice. One such case accounted for a maternal death at the Nowrosjee Wadia Maternity Hospital. Details of the case are presented.

CASE REPORT

Mr. M. S., aged 22 years, para II, was admitted to a Maternity Hospital on 14th January 1966, for mild oedema of feet. There was no other abnormality. She delivered spontaneously a male baby, weighing 2 kg. 500 gms. at 7-0 a.m. on 16th January 1966. The patient had mild post-partum haemorrhage for which she was given parenteral methergine and oxytocin drip for half an hour. Three hours later, the patient complained of severe substernal pain, vomited and collapsed. She was kept on a nor-adrenaline drip for almost 24 hours. She was then transfused with a pint of blood as she was anaemic.

During the above period of observation, she complained of repeated attacks of chest pain. On 17th morning (24 hours later) a mild icteric tinge was noted, and the liver was just palpable. Investigation at this stage revealed a serum bilirubin level of 4 mg.%

In the next 12 hours, the patient became restless, her jaundice rapidly deepened and her blood pressure dropped. She complained of pain in the right hypochondrium as well. At this time, the patient had haematuria. The possibility of a mismatched transfusion was thought of and excluded. Follow-up serum bilirubin level 12 hours after first detecting icterus had risen to 16 mg.%. In spite of resuscitative treatment the patient expired 48 hours after delivery.

Post-mortem examination revealed pulmonary embolism leading to infarction of the lower lobe of the right lung. The liver was normal in size and shape. The edges were sharp. Its external surface showed a huge area of infarction, involving about 50% of the right lobe. The area was soft, markedly congested and raised above the

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Received for publication on 16-1-67.

surface. Cut section showed a wedge-shaped area, the base of which was 8 inches in size. There was an embolus in one of the vessels which was seen while cutting the slices. Surrounding liver parenchyma was pale yellow in appearance. Portal vein and hepatic artery did not show any embolus. There was endometritis and mild changes of pyelonephritis.

Discussion

Pulmonary embolism is the important cause of death in 26 per cent of persons coming to necropsy in general hospitals. In the majority of cases, the sources of emboli are the veins of extremities, pelvis and abdomen, but sometimes the veins of head, neck and arms or the valves of the heart are the sites of origin.

Venous thrombosis may occur at these places either as a result of trauma or infection. Although frequently thrombosis occurs in veins by intravenous infusion, embolisation from this is rare as the inflammatory reaction binds the associated thrombi to the vascular wall. Pulmonary infarction occurs in only 50 per cent of cases of pulmonary embolism.

A patient with pulmonary infarction may present herself with varying picture like cough, haemoptysis, pain, pleural rub. Involvement of diaphragmatic pleura produces abdominal pain and rigidity which may suggest acute abdomen. Occasionally icterus may be also present. The patients of pulmonary infarction usually die of circulatory collapse. At times, massive embolism causes ventricular fibrillation and acute cardiac arrest. Coronary insufficiency caused by lowered blood pressure is believed to be a major contributory cause of death by some.

When the diagnosis of pulmonary embolism or thrombophlebitis has been made or even when it is strongly suspected active anticoagulant therapy should be started at once. Ligation of veins is not done now as often as formerly. It is now resorted to only if the use of anticoagulants is contraindicated.

This patient had strangely enough hepatic infarct with pulmonary infarct. It is the systematic circulation that is involved in pulmonary infarction and portal circulation in hepatic infarction.

The rarity of hepatic infarction in the human beings is due to the dual hepatic blood supply, via the hepatic artery and the portal vein. This explanation can only be accepted with reserve, as arrest of arterial blood supply does not protect the liver from infarction.

The factor of importance is that the arterial blood supply comes from 3 different routes and it is difficult to obstruct these collaterals simultaneously. Hepatic infarction can be caused by thrombosis or ligation of the main hepatic artery between the origin of the right gastric artery and the hilum of the liver. The common causes are infected emboli or polyarteritis nodosa. These interfere not only with the main arterial supply, but also with some of the collateral arterial supply. According to some authors, occlusion of the portal vein may lead to infarcts if it is associated with some other factor tending to produce anoxaemia. Thus aetiological factors responsible for causation of hepatic infarcts are surgical ligation, bland embolism, local lesion in small vessels in liver and thrombosis.

The extent of hepatic infarction would depend on the extent of the intra-hepatic collateral blood flow. Rarity of embolism in hepatic artery is due to the fact that it arises perpendicularly from the coeliac axis. Wooling *et al* (1951) in their series found it occurring in patients with primary disease of the gastro-intestinal tract and spleen, on whom operations had been performed.

Wooling *et al* (1951) also mention that massive hepatic infarction is one specific lesion, which should be recognised as capable of producing hepato-renal syndrome. They also observed that complicating factors tending to result in anoxia were more frequent among the group with no demonstrable vascular lesion than in the group with definite vascular lesions. Frequency of large infarcts was not appreciably greater among the cases with vascular occlusion than among those with demonstrable occlusion.

Acknowledgement

We take this opportunity to thank Dr. B. N. Purandare, M.D., F.R.C.S., F.I.C.S., F.R.C.O.G., F.A.M.S., Honorary Principal Medical Officer, Nowrosjee Wadia Maternity Hospital, Bombay-12, for permitting us to report the hospital case.

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