PULMONARY COMPLICATIONS ASSOCIATED WITH MOLAR PREGNANCY EVACUATION

(Case Report with Review of Literature)

by

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Introduction

The incidence of symptomatic trophoblastic emboli in patients with Gestational trophoblastic disease ranges from 2% toll (Goldstein 1974, Morrow, 1977) clinically, these patients manifest a wide spectrum of pulmonary findings, including the development of pulmonary hypertension and/or acute pulmonary oedema. Some patients may rapidly deteriorate and die within a few days. (Morrow *et al* 1977; Kohorn *et al* 1978; Lipp *et al* 1962).

43 patients of gestational trophoblastic disease have been treated at the Cama and Albless hospitals from March 1980 to February 1983. Over a period of 3 years 1 patient (2.3%) had symptoms suggestive of trophoblastic emboli and haemoptysis soon after evacuation of the mole.

This paper reports the management of the case and suggests the possible pathophysiology for this clinical syndrome and notes that these patients are at high-risk for developing persistent trophoblastic disease (P.T.D.).

CASE REPORT

A 19 year old Hindu housewife, primigravida was admitted on 19-1-1982 with 12 weeks of amenorrhoea and vaginal bleeding off and on since 20 days. Since menarche she had regular menstrual cycles and the date of her last menstrual period was not known. She had been married since 1 year with no history of abortion or stillbirth.

A fortnight prior to the admission she was admitted and treated in another general hospital as threatened abortion, for one week and discharged.

On examination her general condition was fair, moderate pallor present, temperature was normal, pulse was 90/min regular, Blood pressure 110/70 mm Hg. Uterine size was corresponding to 20-22 weeks. On vaginal examination, cervix was soft, os was closed. No adnexal masses were palpable.

A clinical diagnosis of vesicular mole was made and an appointment for ultrasonographic examination was taken. But prior to this investigation patient complained of vomiting and severe pain in the abdomen. Examination at that time showed the temperature to be normal, pulse was 126/min, respiratory rate 28/min, blood pressure 100/74 mm Hg, chest and C.V.S. were normal. Uterine size had increased to 24-26 weeks, uterus was tense and tender. Vaginal examination revealed the os to be 2 cm dilated, no products were felt and the bleeding from the uterus had increased considerably.

Investigations showed Hb to be 8.4 gm%, WBC = 9,100/w.mm. A decision to evacuate the mole was taken. General anaesthesia was administered. Concurrent I.V. Oxytocin, blood, and haemaccele was given during the procedure. Uterine suction was productive of a large amount of molar tissue. Uterine size reduced to 16

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weeks and a gentle blunt curettage was performed.

The patient's condition under anaesthesia was as follows:—Pulse 120/min, R.R. = 28/min. BP = 120/68 mm tig. Blood loss was approximately 1000 ml. During the procedure and over the next 4 hours, 900 ml. of cross matched blood was given slowly to the patient.

Towards the end of the evacuation the patient suddenly became dyspnoeic and was coughing out blood stained frothy sputum. Examination revealed a pulse of 140/min, PR = 38/min, BP = 110/80 mm Hg.

Crepitations were present on both sides. C.V.S. No murmur/rub was present.

The diagnosis of trophoblastic emboli to the lungs was made and patient treated with Nasal 02, sedation of 15 mg of morphine, broad spectrum antibiotics, and I.V. frusemide 80 mg. Over the next 12 hours she gradually improved.

The following day's chest radiograph was normal, Hb was 11 gm%. Gravindex urine test for H.C.G. was positive 1 : 100 and became negative a week later.

Another radiograph taken 4 days later showed an opacity in the Rt Base of lung, this was consistent with a clinical diagnosis of pneumonitis. Patient was treated with broad spectrum antibiotics, bronchodilators and inhalations. She gradually improved and enabled us to do a D & C., Under L.A. and sedation on the 8th post evacuation day. Curettage report was no Vesicles seen. Retained Decidua.

Following the evacuation patient was given methotrexate 5 mg three times a day for 5 days after making sure that the blood counts were normal. Gravindex test repeated on the 15th post evacuation day was also negative and remained so during the 6 months of follow up.

Patient was discharged on the 15th post operative day with a normal chest X-ray, normal haematological investigations and in a good condition. She was advised to take oral contraceptive for a period of 6 months and to come for regular follow up every 15 days for the first 2 months and every month thereafter.

She did come for a fairly regular follow up and had no complaints during her visits. Clinically, there was nothing abnormal detected and the investigations were also within normal limits. After a period of 6 months the oral contraceptives were withdrawn.

Discussion

Kohorn *et al* (1978) also have reported a single patient with this clinical syndrome in a series of 9 patients. His recommended management included oxygen supplementation, narcotics, high dose corticosteroids, and intermittent diuresis, with usual resolution of clinical findings within 72 hours.

The pathophysiology of this clinical syndrome is the same as that of adult Respiratory Distress Syndrome (Staines 1977). This syndrome results from pulmonary emboli including fat and amniotic fluid (Ingram). The initial injury to the alveolar capillary membranes results in fluid filling of interstitial spaces. Eventually this causes alveolar collapse, interference with surfactant production and progressive ventillation/perfusion imbalance with marked right to left shunting. The lung compliance is usually dramatically decreased. A haematologic picture of disseminated intravascular coagulation is a frequently associated finding.

Although Adult Respiratory Distress Syndrome is associated with a 50% to 60% mortality rate (Staines 1977). There is a suggestion, that non-cardiogenic pulmonary oedema may also present with a lesser degree of severity in patients who do not require ventilatory assistance.

Therapy is directed at decreasing hypoxemia with oxygen supplementation, high dose corticosteroids, intermitent diuresis, and mechanical ventilation with P.E.E.P.

Curry *et al* 1978 and Morrow *et al* 1977 have presented some high-risk criteria for patients likely to develop P.T.D. Their criteria include a large for dates uterus, ovarian enlargement or uterus greater than 20 weeks size. Also patients with

pulmonary complications were five times more likely to require chemotherapy for P.T.D. These high-risk patients should therefore be treated immediately with chemotherapy.

Goldstein (1974) has reported a decrease in P.T.D. by using Actinomycia D. in prophylactic regimen. Even though he reported a low toxicity rate this treatment is not advocated routinely by others, since only 15% to 30% of these patients would normally require chemotherapy, and other patients would be subjected to needless drug toxicity.

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