

## MENDELSON'S SYNDROME

(Report of 2 Cases)

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Anaesthetic complications account for some deaths in almost every maternal mortality survey. Inhalation of gastric contents during anaesthesia produces two types of symptoms which according to Mendelson (1946) are—(1) Obstructive type, due to entry of solid food particles into the bronchial tract and (2) asthmatic type of reaction due to bronchiolar spasm caused by inhalation of acidic stomach fluid.

The second type is commoner danger in obstetrics and the underlying pathology of improper ventilation are (a) irritation of alveolar space by the sucked in gastric content giving rise to considerable accumulation of fluids in the alveolar space. This results in complete occlusion of alveolar spaces for gaseous exchange in the affected alveoli, (b) spasm of the bronchioles by the same irritants. The two elements together are responsible for complete inaction of affected alveoli. Thus the total space for gaseous exchange is considerably diminished, resulting in anoxia. The anoxia when continues for long, affects the vital centres, including the respiratory centre and ultimately puts the respiratory centre out of gear. Death

may occur within 12 hours in worst cases (Donald, 1969).

Two cases of this syndrome encountered in Calcutta National Medical College, within a fortnight are reported.

### Case 1

P.G., aged 26 years, P<sub>2+0</sub> booked, was admitted in labour at 22.30 hours on 20-2-1980. Her date of delivery was 8-3-1980. She had L.U.C.S. twice 6 and 2 years back, for pre-eclamptic toxæmia and foetal distress respectively.

On examination, general condition fair. oedema legs ++, pulse—92/min, B.P.—180/110 mm. of Hg, Heart Lungs—NAD. Per abdomen—Uterus—term size, contraction +, Vertex floating, scar tenderness +, F.H.S. 140/min. Vaginally, os 3 cm, cervix—fully effaced, membranes present, Lower pole at—2 cm. Hb—9.5 gm%, urine—albumin +.

As she took food in the evening gastric suction was done followed by a wash with normal saline and sodi bicarb. At 01-20 hours section was done. Two live male babies born each weighing 2 kg. The anaesthetic consisted of Pentothal 200 mg, flexedil 80 mg, O<sub>2</sub> and N<sub>2</sub>O in 50:50 ratio.

On closure of abdomen, some yellowish brown fluid came out on withdrawal of tube. Moderate cyanosis developed, pulse—140/min. and respiration 40/min. The B.P. was 100/70 mm. of Hg. and rales and ronchi developed in both lungs. Tracheal suction was done, Hydrocortisone 1000 mg, and theophyllin 200 mg. I.V. was given. Intratracheal tube was inserted again and O<sub>2</sub> administered under positive pressure. At 02-35 hours, the pulse was 160/min. resp. 48/min. and B.P. 90/60 mm. of Hg. Her

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condition deteriorated and she expired at 5 hours.

#### Case 2

B.G., aged 32 years, P<sub>1+0</sub> booked, was admitted on 22-2-1980. She was overdue by 8 days and had a full term normal delivery 10 years back.

On Examination, general condition fair, oedema legs ++, B.P.—100/80 mm. of Hg, Heart, lungs—NAD.

Per abdomen, Uterus-term size head floating, FSH 136/min. Vaginally os parous, cervix 25% effaced, pelvis—adequate. Hb. 10.9 gm%, Urine—albumin +, blood sugar (PP) 85 mg%. X-ray abdomen revealed twins—1st vertex, 2nd breech. On 2-3-1980 elective LUCS was done at 01-40 hours, male live babies, 2.3 and 2.1 kg respectively.

During closure of abdomen, she developed sudden bronchial spasm followed by gross cyanosis, tachycardia (150/min) and tachypnoea (40/min). The B.P. was 110/70 mm of Hg. Yellowish brown fluid came out on suction of larynx. Besides the measure taken in case—1, Inj. aminophyllin 250 mg, Digoxin 0.25 mg. and lasix 80 mg. was given I.V. Intratracheal suction was copious. Rales and ronchi appeared in both lungs. I.V. dextrose, molar lactate, sodium bicarb 100 c.c. and mannitol 100 c.c. given. At 20.30 hours pulse—was 160/min. Cyanosis + + +, B.P. 84/60 m.m. of Hg. Mephentine 200 mg. given and later intracardiac adrenaline. She expired at 21.10 hours.

#### Discussion

The emptying of stomach is delayed during labour, so food should be restricted in those expected to need anaesthetic delivery. But even with restriction, one cannot avoid asthmatic type of this syndrome as evident in case—2. Dinnick (1957) suggested antacids before anaesthesia to neutralise or diminish gastric acidity and this should be carried out routinely. Though the cause is not clear but it has been postulated by O'Mullane (1954) that fundal pressure to facilitate delivery may act injuriously by spilling

of gastric content into the larynx and thus acid gastric inhalation.

Both the cases had pre-eclamptic toxæmia and so also the case reported by Dutta Gupta *et al* (1979). Kerr (1961) observed that the bronchopulmonary resistance increases in toxæmia and there is a fall of plasma histamine which may be the basis of this syndrome. Further, both of our cases had twins and as such raised intra-abdominal pressure. Hausmann and Lunt (1955) suggested acute adrenal failure to be the cause and found hydrocortisone beneficial but we lost both the cases inspite of massive dose of steroid, besides other measures. These deaths constituted 3.92% of maternal mortality during the period from June, 1977 to May, 1980 in this institution. Parker (1956) found an incidence of 4% in Birmingham.

#### Conclusion

(1) The unbooked and illiterate patients who usually form the bulk of emergency obstetrical surgery are most difficult from anaesthetic point of view. Paradoxically, they do not get the service of senior and experienced anaesthetist. (2) In all cases of emergency anaesthesia proper steps to diminish gastric acidity prior to induction is a must. (3) Too much pressure should not be exerted while extracting the head. (4) If twin delivery require anaesthesia the risk of Mendelson's syndrome, must be kept in mind. (5) There must be provision of intensive care unit including facility of immediate bronchoscopic suction and artificial respiration.

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