MENDELSON'S SYNDROME

by

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Aspiration of gastric contents into the tracheo-bronchial tree is known for more than a century, but its importance on obstetric mortality and morbidity was first stressed by Hall (1940) and Apfelbach (1940). Mendelson (1946) published an analysis of 66 cases and described the results of clinical and experimental investigations of the patho-physiology of aspiration of gastric contents. He described this clinical condition as a distinct entity characterised by a syndrome of aspiration of acid contents of the stomach followed by asthmatic attacks due to spasm of the tracheo-bronchial tree as a result of irritation by gastric hydrochloric acid. Since then this anaesthetic complication goes by the name of "Mendelson's Syndrome". Teabeaut (1952) and Vandam (1965) showed by further experimental work that the pH of the aspirated material must be below 2.5 to produce this type of complication. During the past two decades, many papers on Mendelson's Syndrome have been published which reveal that aspiration of gastric contents has been responsible for more than one third of obstetric fatalities associated with anaesthesia and a greater number of serious morbidity. Bannister and Satillaro (1962) have published a comprehensive review on the subject including the patho-physiology of this condition.

In this paper, two cases are presented where the patient exhibited typical features of Mendelson's syndrome. In the first case, along with the other symptoms and signs, the patient developed coagulation failure which has not been described in such a condition so far and the patient ultimately succumbed. In the second case, the clinical features were of relatively mild severity and was successfully treated with supportive measures.

CASE 1:

Mrs. Dayawanti, 33 years, Para + 0 +1+0, married for 13 years was admitted on 29.10.1968 at 4.45 A.M. with the complaints of amenorrhoea 42 weeks, swelling of feet for one month, labour pains and ruptured membranes for 13 hours prior to admission. Her periods were regular and normal; last menstrual period was on 9th October, 1968. She had no antenatal check up. Excepting oedema of feet since last one month, her antenatal period was uneventful.

On general physical examination, build and nutrition was average, pulse was regular, 96 per minute, of good volume; blood pressure was 150/90 mm Hg. She was moderately anaemic (Hb. 9 gms%), oedema was present. She was using artificial dentures. Examination of heart and lungs did not reveal any abnormality.

Per abdomen, uterus was term size, vertex presentation, L.O.T. position, head engaged and foetal heart sounds were regular and 146 per minute.

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Vaginal examination showed os to be fully dilated, lower pole of head at 'O' station, large caput with excessive moulding. There was pelvic contraction at the mid-cavity and outlet. Caesarean section was decided upon in view of contracted pelvis and obstructed labour.

OPERATION:

Lower segment caesarean section was done in the usual way. Placenta and membranes expelled spontaneously. Total blood loss was approximately 350 ml. Uterus and the abdomen were sutured in layers.

ANAESTHESIA:

The last feed of the patient was noted to be 10 hours before the start of anaesthesia. The artificial dentures were removed before induction. Pre-anaesthetic general condition of the patient was good. Respirations were regular, 15 per minute; pulse was regular, 92 per minute; B.P. was 150/90 mm. Hg. Premedication given was 0.6 mg. of atropine sulphate intramuscularly half an hour before induction.

Total duration of anaesthesia was one hour and twenty five minutes. Induction was done by 250 mg. of Thiopentone sodium and 100 mg. of Suxamethonium given intravenously. Intratracheal intubation was done by No. 8 cuffed intratracheal tube. Nitrous oxide (4 litres) and oxygen (2 litres) were used for maintenance of anaesthesia in a closed circuit. 20 mgms. of d-tubocurarine were given after spontaneous respiration was established. 50 mgms. of pethidine five minutes after the delivery of the baby and 5 mgms. of d-tubocurarine during closure of parietal peritoneum were administered intravenously. The patient behaved well throughout the operation. After the anaesthetic was discontinued, pure oxygen was administered for 5 minutes and injection prostigmine (2.5 mgm) and atropine (1.2 mgm) given intravenously. Patient quickly recovered from anaesthesia and endotracheal tube was removed after suction of oropharyngeal secretions.

Forty-five minutes later, during transfer of the patient from the operation theatre, the patient suddenly vomited a large amount of solid and liquid gastric contents. Head end of the patient was immediately lowered by 45° and mouth and oropharynx were cleared by suction. This was followed by intratracheal intubation and tracheal suction. Lavage of the tracheo-bronchial tree was done with 10 ml. of normal saline. At this stage, the patient developed irregular respiration, cyanosis, wheezing and expiratory dyspnoea. On auscultation, both the lungs were full of coarse crepitations and chest x-ray showed irregular mottling of both lung fields. She was given pure oxygen by artificial respiration along with injection Aminophylline (0.5 gms). Dexamethasone (8 mgm) and atropine (1/200 gr.) intravenously. Intravenous Mannitol drip was also started. Four hours after the aspiration while the supportive therapy was being continued, the patient developed oozing from the needle pricks and vaginal bleeding. Bleeding and clotting time done at this stage were more than 25 minutes. Fresh blood transfusion was started. At 3-45 P.M. pulse and B.P. were unrecordable and the patient died at 4 P.M. (5½ hours after the aspiration).

Case 2

Mrs. Surjeeto, 35 years, Para 7+0+0+6, married for 26 years was admitted on 25-12-69 at 4-15 P.M. with the complaints of amenorrhoea 9 months, mild labour pains for 5 days and ruptured membranes for 24 hours prior to admission. Her last childbirth was 2½ years back and this time she conceived in lactational amenorrhoea. She had no antenatal check up.

On general physical examination, she was moderately dehydrated, pulse was 124/minute, of good volume and regular, she was mildly anaemic (Hb. 10.5 gms%), there was no oedema feet, B.P. was 110/70 mm. Hg. and respirations were regular, 14/minute. Examination of heart and lungs did not reveal any abnormality.

Per abdomen, uterus was term size, vertex presentation, L.O.T. position, head engaged and foetal heart sounds were regular, 140/minute.

Vaginal examination showed cervix to be fully dilated, lower pole of head at 'O' station, hand prolopsed in the vagina, no loop of cord felt.

OPERATION

Examination under anaesthesia was done followed by manual reposition of the

prolapsed hand inside the uterus and mid-forceps delivery. Placenta and membranes expelled spontaneously. Uterus retracted well and there was no post-partum haemorrhage.

ANAESTHESIA

The last feed of the patient was noted to be 24 hours before the start of anaesthesia. Preanaesthetic general condition of the patient was good, dehydration was corrected, respirations were regular, 15 per minute, pulse was regular, 100/minute and B.P. 110/70 mm Hg.

Premedication given was 1/100 gr. of atropine sulphate intravenously 2 minutes before induction. The total duration of anaesthesia was half an hour. Induction was done by intravenous Thiopentone sodium (250 mgm) and Suxamethonium (100 mgm.). Intratracheal intubation was done with No. 8 cuffed intratracheal tube. Nitrous Oxide (4 litres)) and oxygen (2 litres) were used for maintenance of anaesthesia in a closed circuit. The patient behaved well throughout the operation. Pure oxygen inhalation was given for 5 minutes after the anaesthetic was stopped. Patient quickly recovered from anaesthesia and endotracheal tube was removed after suction of orophranygeal secretions. Patient was shifted to the labour room half an hour after stoppage of anaesthesia.

The patient suddenly developed wheezing and intense expiratory dyspnoea, at 7.30 A.M. (1 hour 45 minutes after stoppage of anaesthesia). At this time, pulse was regular, 96/minute and respirations were 32/min. On auscultation, there were coarse crepitations and rhonchi in both the lungs. X-ray chest did not reveal any abnormality. She was put in a propped up position, pure oxygen inhalation started and injection aminophylline (0.5 gms) given intravenously. At 8.15 A.M. respiration rate rose to 40/min., pulse was 144/min., regular and the lung findings were exaggerated. She was given injection digoxin (.25 mg), lasix (40 mgm.) and dexamethasone (8 mgm.) intravenously. Oropharynx was clear and intratracheal suction did not bring out any material. However, the patient gradually improved with the treatment. At 4 P.M., the lung findings subsided to a great extent and the patient did not have any breathlessness. Oxygen inhalation was continued till next day and the patient was shifted to the ward. By 4th day, the crepitations in the lungs were absent. On 8th day, the patient had tubectomy under spinal anaesthesia and was subsequently discharged in a fit condition.

Discussion

Many inter-related predisposing and precipitating factors are responsible for the relatively high incidence of vomiting or regurgitation and consequent aspiration of gastric contents in the obstetric patients. The important ones are,

(1) The emergency nature of obstetric anaesthesia and the higher frequency of patients with full stomach.

(2) The presence of factors which delay gastric emptying.

(3) The generally high incidence of vomiting in obstetric patients.

(4) Increased intragastric pressure and distention.

(5) A relatively high incidence of hiatus hernia.

(6) The effects of anaesthesia.

The first five are predisposing factors while anaesthesia is the precipitating one.

Usually aspiration of vomitus or regurgitation of fluid can be prevented by avoiding the factors mentioned above. The obstetrician should explain this problem during antenatal check up and impress on the patient the importance of abstaining from eating and drinking during labour. If the patient is likely to receive general anaesthesia, no fluid of any kind should be allowed. A few ice chips or a sip of water may be given to prevent dryness of mouth. If dehydration or acidosis is the problem, that should be treated with 5% dextrose drip.

Every patient should be treated as a potential case of aspiration of vomitus and every precaution should be taken to pre-

vent it. Theoretically, the chance of aspiration can be avoided by emptying the stomach before administration of anaesthesia. This can be done by injection of apomorphine or by a large bore stomach tube. A Ryle's tube is absolutely useless in this regard.

Role of administering antacids as a routine in patients in labour who are potential cases for general anaesthesia is also frequently stressed (Mendelson, 1946; Taylor and Pryse-Davis, 1966). Since the main factor in Mendelson's syndrome is the hydrochloric acid content of the gastric juice, (Bannister and Satillaro, 1962), it is a good practice to give antacids in the form of tablet or powder of aluminium hydroxide (every two to three hours) to patients who may require general anaesthesia. In patients who come as an emergency and require general anaesthesia within a short time it will be worthwhile to give the antacid about half an hour before the operation. Other preventive measures which have been advocated from time to time are:

(1) Mechanical blockage of the oesophagus by an inflated baloon. (Macintosh, 1951).

(2) Endotracheal intubation of conscious patient under topical anaesthesia (Bannister and Satillaro, 1962; Weaver, 1964).

(3) Preliminary oxygenation with 100% oxygen for 10 minutes before induction causing denitrogenation of the patient's lung, thus avoiding I.P.P.R. with oxygen after induction, which increases the intragastric pressure.

(4) Cricoid pressure to prevent regurgitation.

(5) Proper position—a head up or a head-down tilt is used, though the latter is preferred.

(6) A rapid smooth induction.

(7) Induction with cyclopropane and oxygen where the patient goes quickly in stage three and there is less chance of vomiting (Bonica, 1967).

As a significant number of these aspiration accidents occur as the patient emerges from anaesthesia, it is important that the patient is to be left on the delivery table and observed until she regains pharyngeal and laryngeal reflexes and becomes conscious. Constant vigilance and prompt action by the anaesthesiologist are important factors in preventing this complication.

Although preventive measures for vomiting and regurgitation are very important to minimise the incidence of Mendelson's syndrome, still it is to be kept in mind that there may not be any overt vomiting in some of these cases. This has been observed in the second case reported in this paper. Hence the best way to deal with this problem is to bear in mind the possibility of such type of anaesthetic complication, specially in obstetric patients and to institute the treatment as soon as it is clinically diagnosed.

Most of the therapeutic procedures in such cases as have been advocated from time to time have been adopted in our cases specially the first case. These procedures may be summarised as follows:

(1) Head down tilt.

(2) Cleaning of the pharynx and larynx.

(3) Laryngoscopy.

(4) Endotracheal intubation and suction.

(5) Pulmonary ventilation using pure oxygen.

(6) Bronchoscopy, if needed.

(7) Tracheo-bronchial lavage with normal saline, (5-10 ml) has been recommended (Bannister and Satillaro, 1962). Lavage with a large quantity of normal saline, on the contrary, may do harm to

the patient by lowering the pulmonary surfactant (Bonica, 1967).

(8) Use of broncholytic agents to reduce the bronchospasm. Administration of corticosteroids has been widely accepted as it may inhibit bronchiolitis and may reduce bronchospasm (Bannister, *et al* 1961; Lawson, *et al* 1966; Nicholl, *et al* 1967).

(9) The institution of intermittant positive pressure respirations by way of endotracheal tube or tracheostomy has been shown to be of great help in the management of pulmonary oedema, relieving patients' exhaustion and dyspnoea and improving the alveolar ventilation by reducing the dead space (Nicholl, *et al* 1967).

(10) Treatment of shock by intravenous fluids and blood transfusion.

(11) Treatment of pulmonary and cerebral oedema by rapid digitalisation, diuretics and mannitol (Brossard, 1967).

(12) The views regarding endotracheal steroid therapy are very conflicting. Lewinski (1965) and Wamberg and Zeskov (1966) suggested on the basis of animal experiments that direct instillation of hydrocortisone into the tracheobronchial tree had the most inhibiting action on acid-pulmonary-aspiration syndrome. But almost similar experiments done by Taylor and Pryse-Davis (1968) showed that endotracheal hydrocortisone or fluocinolone did not affect the course of pulmonary pathology and hence did not recommend direct instillation.

The immediate cause of death in the first case may be the development of coagulation failure manifested clinically by oozing from the needle pricks as well as vaginal bleeding. To combat this complication, fresh blood transfusion was started but the patient died before the coagulation defect could be corrected.

Coagulation failure secondary to Mendelson's syndrome has not been reported so far, but it is a well-recognised complication after operations on certain organs like uterus, lung and prostate. The factors which are of importance in this particular case may be as follows:

(1) Operation on the uterus.

(2) Lung injury caused by the aspiration of acid gastric contents.

(3) The possibility of amniotic fluid embolism, another well known cause of hypo-fibrinogenaemia and subsequent coagulation failure cannot also be altogether ruled out in this particular case as the detailed haematological investigations to pinpoint the exact cause could not be done.

Summary and Conclusions

1. Two cases of Mendelson's syndrome have been reported. The first case developed coagulation failure during the symptomatic management and ultimately died. The second case was of less severity and could be managed successfully.

2. Relevant literature has been reviewed.

3. Preventive measures and treatment of this important anaesthetic complication have been discussed.

4. It has been concluded that preventive measures starting from the antenatal clinic, to keep in mind the possibility of such complications in the postoperative period and to institute the treatment as soon as it is clinically suspected are the best ways to deal with this problem.

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References

1. Bannister, W. K. and Stillaro, A. J. —Anaesthesiology, **23**: 251, 1962.
2. Bonica, J. J.: Obstetric Analgesia and Anaesthesia, F. A. Davis Co. Philadelphia, Vol. 1, 1967.
3. Brossard, G.: quoted in Survey Anaesth. **11**: 156, 1967.
4. Hall, C. C.: J.A.M.A. **114**: 728: 1940.
5. Irons, E. E. and Apfelbach, C. W.: J.A.M.A. **115**: 584, 1940.
6. Lawson, D. W., De falco, A. J., Phelps, J. A., Bradley, B. E. and McClenatnam, J. E.: Surgery. **59**: 845, 1966.
7. Lewinski, A.: Anaesthesiology. **26**: 37, 1965.
8. Macintosh, R. R.: Brit. Med. J. **2**: 545, 1951.
9. Mendelson, C. L.: Am. J. Obst. & Gynec. **52**: 191, 1946.
10. Nicholl, R. M., Holland, E. L. and Brown, S. S.: Brit. Med. J. **2**: 745, 1967.
11. Taylor, G. and Pryse-Davis, J.: Lancet. **1**: 288: 1968.
12. Teabeaut, J. R.: Am. J. Path. **28**: 51, 1952.
13. Vandam, L. D.: New Eng. J. Med. **273**: 1206, 1965.
14. Wamberg, K. and Zeskov, B.: Anaesth. Analg. **45**: 230, 1966.
15. Weaver, D. C.: J.A.M.A. **188**: 971, 1964.