MENDELSON'S SYNDROME AND ITS SEQUELAE

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MENDELSON'S SYNDROME is a well recognized syndrome that may occur after the aspiration of acid gastric fluid under the influence of anaesthesia.¹ Various investigators have confirmed Mendelson's original hypothesis that the development of this syndrome is largely dependent on the pH of the aspirated fluid.^{2,3} Fluids with a pH of less than 2.0 instilled into the tracheobronchial tree of animals will predictably reproduce the clinical picture.^{4,5} Patients are predisposed to aspiration of acid gastric secretions under anaesthesia by delayed gastric emptying, raised intra-abdominal pressures and incompetence of the cardio-oesophageal junction. All these factors may occur in the later months of pregnancy. This explains the relatively high frequency with which this syndrome occurs in patients given general anaesthetics for surgical manipulations at term.^{1,6}

Death from aspiration during anaesthesia is among the commonest causes of preventable anaesthetic deaths.⁷⁻¹⁰ Unfortunately, there is often insufficient evidence to decide whether these deaths were associated with Mendelson's Syndrome or from vomiting and aspirating particulate food material.

Long-term follow-ups of patients developing aspiration pneumonitis have been few. The patient described below, who developed the classical signs of aspiration pneumonitis, was investigated closely, both during the acute phase of the illness and subsequently.

Case History. A healthy, twenty-seven-year-old primipara at term was admitted to the hospital in labour, twelve hours after spontaneous rupture of her membranes. Prior to this time she had received routine antenatal care, during which period no abnormalities had developed. On admission she was having weak contractions at 15-minute intervals and had a cephalic L.O.A. presentation. The foetal heart rate was 134 beats per minute. During her predelivery hospital stay, she was given nothing by mouth and various manœuvres were attempted to stimulate progress of labour.

Fifty-five hours after admission it was decided to perform a caesarean section since labour was not progressing due to cephalo-pelvic disproportion. There was 3+ molding of the caput, an oedematous cervix and the foetal head still lay above the iliac spines.

A nasogastric tube, 18 French gauge was inserted and a small amount of greenish fluid was aspirated. Atropine 0.6 mg was given intramuscularly. One hour later the patient was positioned horizontally on the operating table and pre-oxygenated after the nasogastric tube had again been suctioned (with no resultant aspirate) and its

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FIGURE 1. Progression of X-Ray Findings.

- (a) 50 mins. after aspiration
- (b) 12 Hours after aspiration
- (c) 10 Days after aspiration
- (d) 44 Days after aspiration

end left open. Induction was achieved with 200 mg of 2.5% thiopentone and 50 mg of succinylcholine following 2 minutes of preoxygenation. On cessation of muscle twitching and without prior lung inflation, the pharynx was exposed with a Macintosh laryngoscope and was found to be flooded with greenish fluid. Suction was performed rapidly, the larynx was exposed and an endotracheal tube was passed. Subsequent suction recovered only a few specks of greenish aspirate. Anaesthesia was continued with nitrous oxide: oxygen (6:3 L/min.), and d-tubocurarine 30 mgs. The airways were clear and IPPV was achieved with a Manley ventilator set to a tidal volume of 600 mls and a respiratory rate of 16 per minute. This tidal volume was achieved with an inflation pressure of 24 cms H₂O.

Twenty minutes after induction a ten-pound four-ounce female baby was delivered with an Apgar score of 9 at 1 minute. Forty-five minutes after induction the operation was completed, curare was reversed and the patient was extubated. She wakened promptly and her vital signs were consistent with preoperative values. Respirations were unlaboured without evidence of broncho-constriction. A chest X-ray taken at this time revealed diffuse, patchy opacification. The sequence of X-ray findings is depicted in Figure 1. The X-ray at 44 days was reported as normal.

One hour and thirty-five minutes after induction of anaesthesia, the patient developed a cough. Fine crepitations were present at both lung bases and diffuse wheezing was heard all over the lung fields. All signs were more evident on the right side of the chest.

Two hours and fifteen minutes after induction, respiratory signs had increased and became bi-laterally similar. The pulse rate had risen to 130 beats per minute. Oxygen by face mask was instituted and she was given 100 mg of hydrocortisone intravenously, which was continued at six-hour intervals for 3 days. Other treatments employed were intravenous digoxin 0.75 mg (continued for 8 days) Penicillin 1 mega unit intravenously (continued as crystalline penicillin 1 mega unit 6 hourly for 2 days and then Procaine penicillin 600,000 units b.d.) and streptomycin 0.5 gm. intramuscularly (continued as 0.5 gm bid).

Three hours and 55 minutes after induction her clinical condition was deteriorating. Her temperature had started to rise. She complained of dyspnoea for the first time and by 4 hours 25 minutes her blood pressure had fallen to 60/40 mm Hg, pulse rate was 150 beats per minute and respiratory rate 52 per minute.

At this time she was admitted to the intensive care unit. Her temperature was 103.80°F, respiratory rate 52 per minute, tidal volume 300-400 mls (as measured by Wright respirometer), pulse rate 150 per minute and blood pressure 80/50 mm Hg.

Blood gases drawn with a six-liter oxygen flow into a face mask (FIO_2 approximately 0.5) showed a PaO_2 57 Torr, PaCO_2 30 Torr, pH 7.43, standard bicarbonate 19.5 mEq/L and base excess -5. Other results were: Hct. 48, haemoglobin 15.5 gms %, WBC 4700/cu mm, Na 134 mEq/L, K 4.6, mEq/L, Cl 104 mEq/L and urea 36 mgs %. An EKG monitor was attached and continuously displayed on an oscilloscope. An intra-arterial line was placed in the femoral artery and attached to a pressure transducer. A central venous catheter was inserted through the subclavian vein. It passed without arrhythmias into the right ventricle and was left in this position for measurements on mixed venous blood. Forty mg of lasix was given intravenously and 250 mgs aminophylline intravenously over 10 minutes. Cooling was obtained with ice bags, alcohol rubs and fanning.

It was decided not to reinstitute IPPV as long as the patient's blood gases could be kept within an acceptable range and as long as her clinical condition did not deteriorate further. Indeed, over the next four hours she was subjectively much improved although by 9 hours 55 minutes after induction of anaesthesia, bronchial breathing was heard over the middle and lower zones.

By 8 p.m. on the first post-operative day her temperature was normal, blood pressure 100/60 mm Hg, pulse rate 110, respiratory rate 40 per minute. Frequent physiotherapy, humidified oxygen by face mask and other supportive therapy were continued. Sputum cultures taken at intervals grew normal commensals only.

FIGURE 2. Vital Sign Recordings.

FIGURE 3. Arterial O₂ Values.

FIGURE 4. Cardiac Output Values.

FIGURE 5. Pulmonary Shunt Values.

Figure 2 summarizes changes in vital signs recorded during the first 9 post-operative days. Arterial oxygen values are shown in Figure 3.

At daily intervals for the first 4 days and then at the 46th post-operative day cardiac output and pulmonary shunting were determined (Figures 4 and 5). The dye dilution technique was employed for measuring cardiac output using a Beckman densitometer (Beckman Instruments, Palo Alto, California) and cardiogreen as the indicator. Oxygen contents were derived from PO_2 (suitably corrected for electrode factor and patient temperature) and haemoglobin values of arterial and mixed venous blood. At the follow-up study, a central venous catheter was positioned but was not passed into the right ventricle and mixed venous oxygen content was assumed to be 6 mls.% different from the arterial values, for shunt determinations.¹¹ These results were always obtained both breathing 100 per cent oxygen and room air. In the early phase of the patient's illness she was closely observed for any clinical deterioration when breathing room air.

When she left the intensive care unit on the 6th post-operative day, PO_2 on room air was 74 mm Hg and on 100 per cent oxygen 500 mm Hg. Total pulmonary shunting at this stage represented 22 per cent of cardiac output of which, 7 per cent was accounted for by VQ mismatching. Cardiac output was 5 liters per minute. All these parameters had returned to normal by the 46th post-operative day, when she was investigated as an outpatient.

On the 5th, 9th, and 126th post-operative day, further pulmonary function studies were performed (see Table I). The forced expiratory volume in one second and the forced vital capacity were measured with a dry spirometer with the subject rested. On each occasion values were obtained from a mean of three technically satisfactory performances after two practice attempts. Total lung capacity (TLC), vital capacity, residual volume and functional residual capacity were measured with the spirometer, using the closed circuit helium dilution method. The apparent volume of distribution of a single breath of helium taken up to TLC, expressed as a fraction

TABLE I
RESULTS OF LUNG FUNCTION TESTS. ALL VOLUMES EXPRESSED IN LITRES B.T.P.S.

Parameter	Normals	Post-operative day		
	Standard ± deviation	5	9	126
FEV 1.0	2.58 ± 0.41	1.02	1.40	1.93
FVC	3.18 ± 0.47	1.42	1.94	2.51
FEV 1.0/FVC %	81.7 ± 7.6	71.8	72.2	76.9
TLC	4.88 ± 0.46	2.97	2.89	4.30
VC	3.35 ± 0.44	1.16	1.69	2.44
RV	1.41 ± 0.24	1.81	1.20	1.45
RV/TLC %	30.6 ± 5.4	60.9	41.5	33.7
FRC	2.96 ± 0.49	—	1.96	2.49
Index	0.9 ± 1.0	—	1.03	1.00
TF, ml min ⁻¹ TORR ⁻¹	26 ± 2.1	13	15	27
DM, ml min ⁻¹ TORR ⁻¹	48 ± 6.2	—	24	52
V _a , ml	50-100	—	42	63

Index - Index of distribution of ventilation.

TF - Transfer factor.

DM - Diffusion capacity of alveolar-capillary membrane.

V_a - Alveolar-capillary blood volume.

of TLC measured by the rebreathing method, was used as an index of the distribution of ventilation. Gas volumes were expressed at B.T.P.S.

The single breath CO transfer factor was measured with an automatic apparatus¹² and partitioned into its two components, the diffusion capacity of the alveolocapillary membrane and alveolocapillary volume by the method of Roughton and Forster.¹³ Predicted average normal values for the indices of lung functions were calculated from multilinear regression equations derived from a study of healthy Jamaican females.¹⁴

DISCUSSION

Many therapeutic procedures have been described by others as to their relative values in preventing aspiration during induction of anaesthesia.^{10, 15-17} If general anaesthesia is a common practice for obstetrical operations, the most logical single procedure is probably the routine administration of antacid to all obstetrical patients in labour.⁹ Antacids were not given prior to induction in our patient and, aside from preoxygenation, absence of IPPV prior to intubation and the presence of a nasogastric tube (an argument can be made for taking any nasogastric tube out immediately prior to induction) other preventive measures were not used either. The pH of the aspirate was not measured but the ensuing syndrome was characteristic of the inhalation of low pH fluid.

Treatment and supportive therapy after the occurrence was generally along the lines suggested for these patients.^{18,19} However, the hydrocortisone should have been given immediately after aspiration was recognized.²⁰

The haemodynamic and oxygenation measurements made on this patient all returned to normal within 46 days. The hypoxaemia was the result of VQ abnormalities and to shunting through the lungs. These two effects parallel each other in their return to normality. The high cardiac output seen initially was probably a response to her elevated temperature and hypoxaemia. Normal values were seen by the 6th post-operative day.

During the early stages of recovery, there was restrictive lung disease with reduction in total lung capacity, transfer factor, the diffusion capacity of the alveolocapillary membrane and the alveolocapillary blood volume. Initially the patient was too breathless to make a full expiratory effort, hence the vital capacity was small and the fraction of the total lung capacity occupied by residual volume (RV-TLC%) above normal. Breathlessness was diminished by the second lung function study when the RV-TLC% was at the upper limit of normal for this patient.

Lung functions were substantially improved 4 months after the event. Lung volumes were in the low normal range and the indices of gas transfer were close to predicted average normal values for the patient. There was never any evidence of airway obstruction in these studies and the fraction of the vital capacity expired in the first second and the index of distribution of ventilation were normal on each occasion.

These findings are not dissimilar from those reported by Sladen and Adams^{21,22} although the degree of reversal of the pathological changes seems to be more complete in the patient we have described. This situation is probably related to the

degree of initial damage. Although prophylactic IPPV has been shown in animals to have a marked beneficial effect following aspiration pneumonitis,²³ such effect must be weighed against the iatrogenic damage that long-term IPPV may cause in patients.²⁴ Certainly some of the Sequelae seen in Adams' patient was a result of such iatrogenic complications. It may well have been only because we could avoid reintubating our patient that super-added lung infection did not complicate the situation.

SUMMARY

The history is described of a 27-year-old primipara who developed Mendelson's Syndrome, following aspiration of liquid gastric contents on induction of anaesthesia for a Caesarean section. The progress of the Syndrome was documented by X-rays, vital signs, blood gases, cardiac output, pulmonary shunting and lung function tests. Lung function tests included an index of distribution of ventilation and the single breath carbon dioxide transfer factor.

In spite of seriously deranged cardiorespiratory parameters during the first few days following aspiration, all measurements were normal by the 126th post-operative day. It is suggested that the sequelae seen by others following aspiration may have resulted from a more severe initial lung response and/or complications of therapy.

RÉSUMÉ

C'est l'histoire d'une primipare de 27 ans qui a présenté un syndrome de Mendelson à la suite de l'aspiration du contenu gastrique lors de l'induction de l'anesthésie pour une césarienne. L'évolution de la maladie a été confirmée par les radiographies, par les signes vitaux, par les gaz sanguins, par le débit cardiaque, par le "shunting" pulmonaire et par les tests de la fonction pulmonaire. Les tests de la fonction pulmonaire incluent l'index de la distribution de la ventilation et le facteur transport du dioxyde de carbone pour une seule respiration.

Malgré une modification importante des paramètres cardiorespiratoires durant les quelques premiers jours suivant l'aspiration, lors du 126^e jour après l'opération, tous les résultats étaient normaux. Il faut croire que les séquelles observées par d'autres, à la suite d'une aspiration, ont été accompagnées d'une réponse initiale pulmonaire plus sérieuse et/ou de complications des traitements.

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