Cardiorespiratory arrest with laryngeal oedema in pregnancy-induced hypertension

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A 17-year-old black female with pregnancy-induced hypertension (PIH) suffered cardiorespiratory arrest on arrival in the recovery room after Caesarean section under general endotracheal anaesthesia. Successful resuscitation included orotracheal intubation, complicated by severe laryngeal oedema. Causative mechanisms are discussed.

Key words

COMPLICATIONS: arrest, cardiac; arrest, respiratory; pregnancy-induced hypertension. ANAESTHESIA: obstetric; ANAESTHETIC TECHNIQUES: endotracheal.

Laryngeal oedema may develop in obstetrical patients with pregnancy-induced hypertension (PIH)[1–5] and in healthy parturients.[2,4] It may embarrass the airway and complicate endotracheal intubation[1,2,4] or even necessitate tracheostomy.[3] Only nine cases have been reported to date.[1–5] However, this complication is thought to be much more common than the few reported cases may reflect.[4] The present case is believed to be the first reported cardio-respiratory arrest in association with PIH-related laryngeal oedema.

Report

For three weeks prior to admission a 17-year-old black female, gravida 1 at term, had had some cough, rhinorrhoea and her voice had become slightly hoarse. The patient was 163 cm tall, weighed 102 kg, had a blood pressure of 140/102 mmHg and heart rate of 96/minute. Her extremities showed trace oedema. A slight rhinorrhoca was noted. She received intramuscular injections of magnesium sulfate for pregnancy-induced hypertension (PIH), but no analgesics. After 12 hours of slow progress of labour, low transverse Caesarean section for cephalopelvic disproportion was planned. After 30 ml of milk of magnesia p.o., the patient was placed in the supine position with a 15° left lateral tilt. Dextrose 5 per cent in lactated Ringer's solution, until then infusing at 80 ml/h, was replaced by plain lactated Ringer's solution. EKG lead II was monitored continuously. Blood pressure was measured with an automatic device every minute until delivery and every three minutes thereafter. After preoxygenation, anaesthesia was induced with a rapid sequence technique using 3 $mg \cdot kg^{-1}$ thiopental and 1.4 $mg \cdot kg^{-1}$ succinylcholine IV. Moderate muscle fasciculations were observed. A 7.0 Portex endotracheal tube was passed orally, without difficulty. When manual ventilation produced bubbling sounds from the patient's mouth, cricoid pressure was reinstituted and at laryngoscopy the tube was found intratracheally, with the cuff well below the vocal chords. Moderate laryngeal oedema was noted, with swollen false chords covering the vocal chords. After additional cuff inflation, clear breath sounds were heard bilaterally. Anaesthesia was maintained with 50 per cent nitrous oxide in oxygen, with controlled ventilation, until a viable male infant was delivered. Anaesthesia was then continued with 10 mg of morphine sulfate, 200 μg of fentanyl and 2.5 mg of Droperidol, all IV, as well as nitrous oxide and oxygen at 4:2 liters per minute. Twenty

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units of oxytocin were added to the intravenous infusion. Muscle relaxation was ensured with a titrated infusion of succinylcholine, monitored with a nerve stimulator. Gastric contents were suctioned via an orogastric sump tube. The operation lasted 52 minutes, urine output was 76 ml, fluid intake was 1200 ml. After skin closure, the patient had recovered full muscle strength as evidenced by response to tetanic stimulus, train-of-four test, head-lift and hand-squeeze test. The patient was breathing at a regular rate with normal chest excursions when she was extubated, after oral and pharyngeal secretions had been suctioned. She responded to verbal communication, however, spoke with a hoarse voice and appeared to be disoriented. Initially, the patient produced snoring breath sounds but no wheezing or stridor. She was taken to the recovery room while lying on her left side, in a head up position. On arrival there, her blood pressure was 160/110 mmHg and her heart rate 64 beats/minute. During counting of her respiratory rate, the patient had a respiratory arrest with subsequent cardiac arrest. Immediate resuscitation included chest compression and ventilation by mask and Ambu-bag, while cricoid pressure was reinstituted. Laryngoscopy revealed severe tongue and laryngeal oedema, obstructing direct vision of the larynx. A 7.0 Portex tube was passed through the severely oedematous glottis, with difficulty. Three minutes after initiation of resuscitation, the EKG monitor showed spontaneous restoration of electrical heart activity at a rate of 145 bpm with multifocal ventricular extrasystoles. Lidocaine 100 mg IV restored normal sinus rhythm. Spontaneous respiration returned. Naloxone 0.8 mg IV cleared the patient's obtunded sensorium and restored full orientation. The serum magnesium level was 3.3 meq/L. The endotracheal tube was tolerated well by the patient, without sedation. After 12 hours of breathing humidified 40 per cent oxygen the oral pharynx was sprayed with lidocaine four per cent and awake laryngoscopy, revealed minimal swelling of the larynx and no tongue oedema. With the endotracheal tube removed, the patient was able to breathe without difficulty and speak with a normal voice. Breath sounds were clear over all lung fields. Humidified 40 per cent oxygen was supplied by face mask for 2½ uneventful hours under direct observation. There were no more respiratory problems during the hospital stay.

Discussion

PIH is frequently associated with oedema. In fact, the term used for this disease in Europe is "Edema-proteinuria-hypertension (EPH)-gestosis." Laryngeal oedema has been recognized as a serious complication of obstetrical anaesthesia.[1-5] Mackenzie[2] believes that even in the absence of PIH, prolonged labour with sustained effort may lead to increased venous pressure and thus cause laryngeal oedema. In three[2,4] of the nine previously reported cases PIH was absent, but strenuous labour had taken place. One PIH patient had also had an upper respiratory tract infection.[5] The patient described in the present report suffered from PIH, prolonged and slow progress of labour, as well as an upper respiratory tract infection. It is striking that no stridor was observed prior to the respiratory arrest. The absence of stridor has specifically been noted in three cases[1,2,4] whereas in four other cases absence of stridor, although not mentioned, may be assumed.[2,4] One patient initially presented with stridor,[5] whereas another patient developed post-intubation stridor.[3] The most impressive finding in the present patient was the dramatic increase in laryngeal and tongue oedema. Whereas, at induction of anaesthesia only moderate swelling was noted, the oedema one hour later, at the time of resuscitation, was so severe that the glottis could not be visualized. Previous reports[1,2,4] only described the finding of laryngeal oedema but no progression. One case of postintubation subglottic oedema was reported to develop after three days.[3] The rapidity of oedema formation in the case reported here reminds one of epiglottitis in children. The pre-existing oedema could have been aggravated by the repeated instrumentation of the oral cavity (two attempts of laryngoscopy, placement of orogastric sump tube, suctioning of the oropharynx). Fluid therapy, namely balanced salt solution 1200 ml within one hour may have contributed to the increased oedema formation. Fluid therapy was identified as the cause of cerebral oedema in three cases of PIH.[6] One patient, described in that report, also suffered respiratory arrest. The fact that the patient in this present report appeared to have an obtunded sensorium and was not fully oriented may be attributed to cerebral oedema, which may also have caused respiratory depression. On the other hand, naloxone cleared the patient's obtunded sensorium. Thus, even though

only small doses of narcotics were employed, they may also have contributed to respiratory depression. Hypermagnesemia of 35.1 mg/dL (28.9 meq/L) has been described as a cause for cardiopulmonary arrest.[7] Our patient, however, had a serum magnesium level of 3.3 meq/L, thus even below the therapeutic range of 3.5–6.0 meq/L.[8] Magnesium can also augment the muscle relaxation obtained with either depolarizing and non-depolarizing muscle relaxants[9,10] and thus possibly embarrass postoperative respiration. Since our patient objectively showed recovery of muscle strength, that possibility can be ruled out. In cases of PIH such as presented here, rapidly increasing laryngeal oedema may lead to a cardio-respiratory arrest. Cerebral oedema and/or residual narcotics, given either to alleviate the pain of labour or, as here, to maintain anaesthesia, may contribute to respiratory depression. If general anaesthesia is chosen for a PIH patient, instrumentation of the oral cavity should be kept minimal.

References

1 *Brock-Utne JG, Downing JW, Seedat I.* Laryngeal oedema associated with pre-eclamptic toxaemia. Anaesthesia 1977; 32: 556–8.

2 *Mackenzie Al.* Laryngeal oedema complicating obstetric anaesthesia, three cases. Anaesthesia 1978; 33: 271.

3 *Keeri-Szanto M.* Laryngeal oedema complicating obstetric anaesthesia, yet another case. Anaesthesia 1978; 33: 272.

4 *Jouppila R, Jouppila P, Hollinen A.* Laryngeal oedema as an obstetric anaesthesia complication. Acta Anaesthesiol Scand 1980; 24: 97–8.

5 *Seager S, MacDonald R.* Laryngeal oedema and preeclampsia. Anaesthesia 1980; 35: 360–2.

6 *Benedetti TJ, Quilligan EJ.* Cerebral edema in severe pregnancy-induced hypertension. Am J Obstet Gynecol 1980; 137: 860–2.

7 *McCubbin JH, Sibai BM, Abdella TN, Anderson GD.* Cardiopulmonary arrest due to acute maternal hypermagnesemia. Lancet 1981; 1 (8228): 1058.

8 *Pritchard JA.* The use of magnesium sulfate in preeclampsia-eclampsia. J Reprod Med 1979; 23: 107–14.

9 *Morris RE, Giesecke AH Jr.* Potentiation of muscle relaxants by magnesium sulfate therapy in toxemia of pregnancy. South Med J 1968; 61: 25–8.

10 *Giesecke AH Jr., Morris RE, Dalton MD, Stephen CR.* On magnesium, muscle relaxants, toxemic parturients and cats. Anesth Analg 1968; 47: 689–95.

Résumé

Une adolescente de 17 ans, de race noire, souffrant d'hypertension de grossesse a présenté un arrêt cardio-respiratoire à son retour de la salle d'opération à la suite d'une opération césarienne sous anesthésie générale avec intubation trachéale. La patiente a été réanimée par les manœuvres habituelles incluant l'intubation trachéale, celle-ci s'est compliquée d'un œdème laryngé sévère. Le travail discute des divers éléments possiblement en cause.

L'œdème laryngé est reconnu comme une complication parfois sérieuse de l'anesthésie obstétricale. En l'absence d'hypertension de grossesse, le travail prolongé avec les efforts violents qu'il peut comporter contribue à élever la pression veineuse qui à son tour peut causer de l'œdème laryngé.

D'autre part, l'hypertension de grossesse est fréquemment associée à de l'œdème. La patiente que nous décrivons dans ce travail souffrait d'hypertension de grossesse et en plus son travail était prolongé et elle présentait une infection des voies respiratoires supérieures. Un des éléments particulièrement impressionnants chez cette malade était l'augmentation spectaculaire de l'œdème du larynx et de la langue. En effet à l'induction l'œdème était tout à fait modéré alors qu'une heure plus tard, au moment de la ressuscitation il était impossible de visualiser la glotte.

L'œdème pré-existant a pu être aggravé par les manipulations instrumentales dans la cavité orale, soit deux tentatives de laryngoscopie, la mise en place d'un tube oro-gastrique, la succion de l'oro-pharynx.

L'administration de soluté salin à raison de 1200 ml dans une heure a pu contribuer à l'augmentation de l'œdème.

Il n'est pas impossible qu'un certain degré d'œdème cérébral ait pu exister et contribuer à la dépression respiratoire en association avec les petites doses de morphiniques employés.

Ce travail illustre donc que dans des cas d'hypertension de grossesse, un œdème laryngé peut se développer très rapidement et mener à l'arrêt cardio-respiratoire. L'œdème cérébral et/ou l'effet résiduel des morphiniques peut contribuer à la dépression respiratoire. Si on choisit une technique d'anesthésie générale chez ces malades, les manipulations instrumentales dans l'oro-pharynx se doivent d'être maintenues au minimum.