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Bilateral phrenic nerve injury after neck dissection: an uncommon cause of respiratory failure

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Abstract
The case of a patient with carcinoma larynx who developed diaphragmatic paralysis and post-operative respiratory failure due to bilateral phrenic nerve injury is reported. The use of portable ultrasonography for an early diagnosis of diaphragmatic paralysis is discussed.

Key words: Respiratory insufficiency; Respiratory paralysis; Phrenic nerve; Post-operative complications

Introduction

Phrenic nerve injury is a known complication of neck surgery usually producing asymptomatic diaphragmatic paralysis (Coleman, 1986; Everts *et al.*, 1993). It gives rise to respiratory inadequacy only if there is an associated pulmonary problem (Beahrs, 1977). We report a case of carcinoma larynx that underwent bilateral radical neck dissection and sustained bilateral phrenic nerve injury causing diaphragmatic paralysis and respiratory failure.

Case report

A 46-year-old man weighing 84 kg presented to the hospital with a swelling in the right side of the neck for one year and hoarseness of the voice for three months. A right aryepiglottic growth with a fixed right hemilarynx was found on direct laryngoscopy. A diagnosis of carcinoma larynx was made and the patient admitted for surgical management. The patient was hypertensive and on treatment with atenolol, nifedipine and alprazolam. He was a habitual chewer of betel leaves and tobacco but was not a smoker. He had dyspnoea on moderate exertion. The pre-operative physical examination revealed no abnormality other than mild obesity with a body mass index (BMI) of 30.8. The haematological and biochemical investigations and the chest X-ray (Figure 1) were within normal limits. An electrocardiogram showed a left axis deviation.

The patient was given his regular morning doses of antihypertensive medication and premedicated with intramuscular pethidine and promethazine one hour prior to anaesthesia. A balanced narcotic-relaxant anaesthetic technique was employed using pancuronium, pethidine, oxygen, nitrous oxide and halothane. Total laryngectomy with right radical neck dissection, left modified neck dissection and primary tracheooesophageal puncture were performed. The intra-operative course of the 3.5 hour procedure was uneventful. At the end of the procedure, the neuromuscular block was reversed with neostigmine and atropine. However, the patient had a residual nondepolarizing neuromuscular block as demonstrated by a train of four and double burst stimulation (DBS) of the ulnar nerve. The patient was transferred to the ICU and ventilated mechanically with an inspired oxygen concentration (F_IO_2) of 0.4. His arterial blood gas

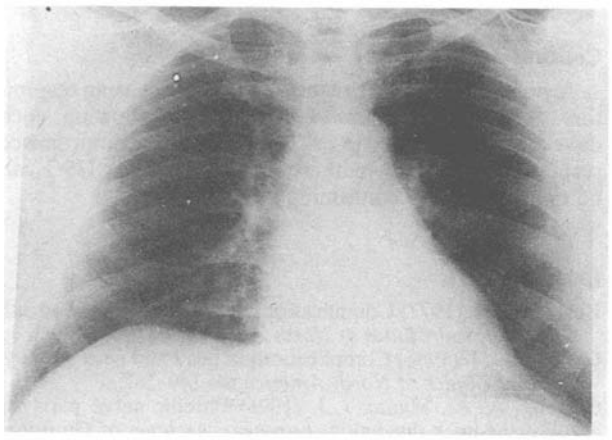

FIG. 1
Pre-operative X-ray chest, PA view.

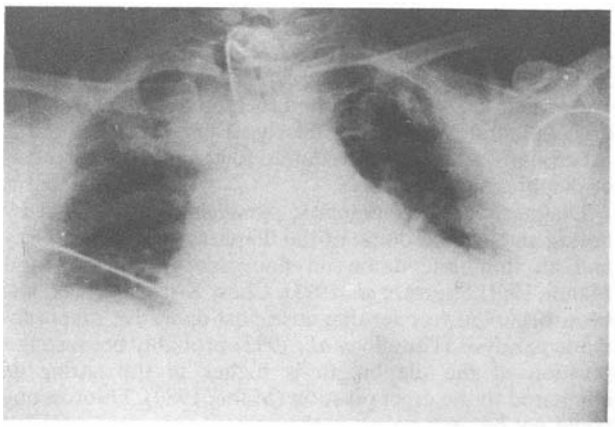

FIG. 2
Post-operative X-ray chest (day 2), AP view.

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analysis (ABGA) at that point was: pH 7.38, P_aCO_2 36.0 mmHg and P_aO_2 69.5 mmHg. At the end of an hour the DBS response was normal and a trial of spontaneous respiration was given but the respiration continued to be shallow, laboured and paradoxical. The ABGA showed normal pH and P_aCO_2 but P_aO_2 was 52.5 mmHg. Intermittent positive pressure ventilation (IPPV) was reinstituted. A trial of weaning after four hours failed again and the patient was ventilated overnight after sedation with buprenorphine 0.3 mg and diazepam 5 mg intravenously.

The patient's ventilatory pattern remained the same next morning. There were reduced breath sounds over the infra-axillary and infrascapular regions bilaterally. A chest X-ray in the sitting posture showed haziness in both the lower zones, more marked on the left side (Figure 2). Pleural effusion was suspected but a pleural tap in the left sixth intercostal space failed to drain any fluid. A reassessment of the chest X-ray at this time led to suspicion of elevated domes of the diaphragm, possibly due to diaphragmatic paralysis. A bedside ultrasonogram (USG) showed markedly elevated domes of the diaphragm without any appreciable movement on spontaneous respiration. There was no pleural effusion. A diagnosis of bilateral diaphragmatic paralysis due to bilateral phrenic nerve injury during neck dissection was made.

Ventilatory support was continued with a minute volume of 6–8 litres, an F_IO_2 of 0.4 and a positive end expiratory pressure (PEEP) of 6–8 cmH_2O. The P_aO_2 gradually increased from 60 to about 100 mmHg by the third post-operative day and weaning was begun using synchronized intermittent mandatory ventilation (SIMV). A repeat USG on the fourth post-operative day showed no improvement in the diaphragmatic movements. The patient however, continued to tolerate weaning and was on spontaneous respiration by the seventh post-operative day. An X-ray of the chest at this time showed the diaphragm to be in a normal position. The patient was transferred out of the ICU on the eighth post-operative day. Fluoroscopy of the chest performed on the tenth post-operative day showed a normally positioned and mobile diaphragm. The rest of the patient's stay in the hospital was uneventful and he was discharged with a vocal prosthesis.

Discussion

Respiratory failure is one of the causes of increased post-operative morbidity and mortality. In the present case, the respiratory distress noted at the end of anaesthesia was initially attributed to residual neuromuscular block. However, the respiratory failure persisted even when the neuromuscular block had completely worn off. A pleural effusion was falsely suspected on radiological grounds. A USG was required to confirm the diagnosis of diaphragmatic paralysis.

Diagnosis of diaphragmatic paralysis can be made by noting an elevated dome of the diaphragm on chest X-ray and an immobile dome on fluoroscopy (de Jong and Manni, 1991; Everts *et al.*, 1993). Chest X-ray however, has been observed to over-diagnose post-operative diaphragmatic paralysis (Fedullo *et al.*, 1992) probably because the position of the diaphragm is higher in the sitting as compared to the erect position (Milne, 1980). Fluoroscopy could not be undertaken in the present case as the patient was on ventilatory support and could not be moved out of the ICU. Ultrasonography is capable of detecting diaphragmatic movements (Harris *et al.*, 1983) and is more specific than chest X-ray for diagnosis of diaphragmatic paralysis (Fedullo *et al.*, 1992). Thus a portable USG machine could be used for detecting diaphragmatic paralysis in nonambulatory patients.

Phrenic nerve injury is a known complication of neck dissection (Coleman, 1986; Everts *et al.*, 1993). In a retrospective study of 176 unilateral neck dissections, its incidence was eight per cent (de Jong and Manni, 1991). Bilateral phrenic nerve injury following neck surgery however, is rare. Diaphragmatic paralysis following phrenic nerve injury does not usually result in respiratory failure, unless there is some concurrent pulmonary problem (Beahrs, 1977). A search through Medline database from 1981 to July 1994 and Cancer database from 1988 to April 1993 revealed only two case reports of respiratory failure due to phrenic nerve injury following neck surgery. Moorthy *et al.* (1983) reported two patients who developed unilateral phrenic nerve injury after neck dissection. These patients developed an increased alveolar-arterial oxygen difference and hypoxaemia requiring oxygen supplementation but no ventilatory support. Rosett (1987) reported the case of a patient who underwent bilateral supraclavicular rib excision and developed bilateral phrenic nerve injury requiring a brief period of post-operative ventilation.

Diaphragmatic paralysis causes the domes of the diaphragm to lie high in the thorax resulting in atelectasis and hypoxaemia (Iverson *et al.*, 1976). In experimental animals it has been observed that bilateral phrenicotomy results in hypoxaemia and hypercarbia (Ninane *et al.*, 1989; Nachazel and Palecek, 1992). The hypoxaemia due to diaphragmatic paralysis may be accentuated in obese patients. Obesity causes ventilation-perfusion mismatch (Havlik *et al.*, 1983) and decrease in the functional residual capacity leading to atelectasis (Grant, 1990). All of these lead to hypoxaemia. Marrone *et al.* (1989) have reported a patient with bilateral diaphragmatic paralysis in whom obesity precipitated respiratory failure. Our patient had mild obesity, BMI being 30.8, and developed hypoxaemia following diaphragmatic paralysis but could maintain normocarbia.

Injury to the phrenic nerve may cause neurapraxia or neurotmesis and thus result in transient or definitive diaphragmatic paralysis respectively (Iverson *et al.*, 1976). Transient diaphragmatic paralysis may take three days to six months to recover (Iverson *et al.*, 1976; Rosett, 1987). In the present case, the patient could be successfully weaned from the ventilator and had normal diaphragmatic movements within 10 days of surgery, indicating phrenic neurapraxia.

Conclusion

A possibility of diaphragmatic paralysis causing respiratory failure should be kept in mind following neck dissection especially in a patient with compromised respiratory function. In these cases a portable USG can be of great help in confirming the diagnosis.

References

Beahrs, O. H. (1977) Complications of surgery of the head and neck. *Surgical Clinics of North America* **57:** 823–829.

Coleman, J. J. (1986) Complications in head and neck surgery. *Surgical Clinics of North America* **66:** 149–167.

de Jong, A. A., Manni, J. J. (1991) Phrenic nerve paralysis following neck dissection. *European Archives of Otorhinolaryngology* **248:** 132–134.

Everts, E. C., Cohen, J. I., McMenomey, S. O. (1993) Surgical complications. In *Otolaryngology – Head and Neck Surgery*. 2nd Edition. (Cummings, C. W., Fredrickson, J. M., Harker, L. A., Kranse, C. J., Schuller, D. E., eds.), Mosby Yearbook Publishers, St Louis, pp 1673–1690.

Fedullo, A. J., Lerner, R. M., Gibson, J., Shayne, D. S. (1992) Sonographic measurement of diaphragmatic motion after coronary artery bypass surgery. *Chest* **102:** 1683–1686.
Grant, I. S. (1990) Intercurrent disease and anaesthesia. In *Textbook of Anaesthesia.* 2nd Edition. (Aitkenhead, A. R., Smith, G., eds.), Churchill Livingstone, UK. pp 645–676.
Harris, R. S., Giovannetti, M., Kim, B. K. (1983) Normal ventilatory movement of the right hemidiaphragm studied by ultrasonography and pneumotachography. *Radiology* **146:** 141–144.
Havlik, R. J., Hubert, H. B., Fabsitz, R. R., Feinleib, M. (1983) Weight and hypertension. *Annals of Internal Medicine* **98:** 855–859.
Iverson, L. I. G., Mittal, A., Dugan, D. J., Samson, P. C. (1976) Injuries to the phrenic nerve resulting in diaphragmatic paralysis with special reference to stretch trauma. *American Journal of Surgery* **132:** 263–269.
Marrone, O., Sanci, S., Cibella, F., Pipitone, P., Milone, F., Bellia, V. (1989) Obesity as a possible cause of respiratory failure in bilateral diaphragmatic paralysis. *Italian Annals of Internal Medicine* **4:** 408–412.
Milne, E. N. C. (1980) Chest radiology in the surgical patient. *Surgical Clinics of North America* **60:** 1503–1518.
Moorthy, S. S., Gibbs, P. S., Losasso, A. M., Lingeman, R. E. (1983) Transient paralysis of the diaphragm following radical neck surgery. *Laryngoscope* **93:** 642–644.
Nachazel, J., Palecek, F. (1992) Hypoventilation after acute phrenicotomy of the urethane anaesthetized rats. *Physiological Research* **41:** 375–380.
Ninane, V., Farkas, G. A., Baer, R., de Troyer, A. (1989) Mechanism of rib cage inspiratory muscle recruitment in diaphragmatic paralysis. *American Review of Respiratory Diseases* **139:** 146–149.
Rosett, R. L. (1987) An unusual cause of post-operative respiratory failure. *Anesthesiology* **66:** 695–697.

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