Lesson of the Week

Papilloedema and respiratory failure

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Papilloedema is a rare but well recognised complication of respiratory failure that was first described in 1933 in a patient with pulmonary emphysema.¹ Several cases have been reported of patients with respiratory failure and papilloedema who developed optic atrophy and blindness.^{2,3} We report on a patient who presented with loss of vision and papilloedema secondary to the obesity hypoventilation or pickwickian syndrome.

Case report

An obese 58 year old woman presented to her general practitioner with a week's history of blurring of vision. She was found to have papilloedema and was admitted as an emergency under the care of ophthalmologists. Apart from misting of vision, which she described as a film developing over her eyes, she had no other visual or neurological symptoms. She denied having headaches. She had recently had a chest infection treated with tetracycline, but she had no relevant medical history. She had smoked 20 cigarettes a day for 40 years, but she denied having a regular productive cough or breathlessness.

On examination she was noted to be obese, weighing 112 kg; her height was 155 cm. Her pulse rate was 80 beats/minute and regular, with blood pressure 170/100 mm Hg. She had bilateral ankle oedema but no other signs of heart failure. Auscultation of her chest elicited scattered bilateral expiratory wheeze.

Visual acuity was 6/9 in both eyes, and she had gross bilateral papilloedema with haemorrhages and venous congestion (fig 1). Visual fields were full to confrontation.

An intracranial tumour was suspected, and she was referred for a medical opinion. She showed clinical signs of respiratory failure with central cyanosis, a bounding pulse, and a flapping tremor. She also had a rattling cough. Her obesity and plethoric appearance suggested the obesity hypoventilation or pickwickian syndrome. She was noted to be somnolent in the ward, and she admitted on questioning to being drowsy at home.

Her haemoglobin concentration was 15.5 g/dl, white cell count $9.3 \times 10^9/l$, and erythrocyte sedimentation rate 27 mm in the first hour. Plasma urea and electrolyte concentrations, calcium and thyroxine concentrations, results of liver function tests, and blood glucose concentration were normal. Radiographs of the chest showed an increased cardiothoracic ratio as a result of poor inspiratory effort. No great changes were evident in either lung field. The arterial blood gas tensions confirmed hypercapnia and hypoxia: carbon dioxide tension was 7.2 kPa (54 mm Hg) and oxygen tension 8.3 kPa (62 mm Hg) pH 7.37. An electrocardiogram showed sinus rhythm. Tests of lung function showed a mild obstructive impairment of ventilatory capacity, but the predominant defect was restrictive and consistent with her obesity: forced vital capacity 1.80 l (60% predicted) and forced expiratory volume in one second 1.28 l (54% predicted), giving a ratio of forced expiratory volume in one second to forced vital capacity of 71% (86% predicted). The expiratory reserve volume was 0.32 l (30% predicted). The total lung

Respiratory failure may produce papilloedema resulting in loss of vision

capacity was minimally reduced at 3.59 l (75% predicted), and the residual volume was normal at 1.55 l (90% predicted) using the helium dilution technique. The single breath transfer factor was also normal at 92% predicted.

Radiographs of the skull showed decalcification of the posterior clinoid process. A computed tomogram of the head showed no evidence of a space occupying lesion; the cerebral cortex was oedematous. Ear oximetry

FIG 1—Appearance of right fundus on admission: papilloedema with haemorrhages and venous congestion.

(Hewlett Packard 47201 A oximeter) confirmed that her oxygen saturation decreased from about 80% when she was awake to 40% during periods of somnolence. Further analysis of arterial blood gas tensions after voluntary hyperventilation on air showed that she could correct both hypoxia and hypercapnia; oxygen tension rose to 12.4 kPa (93 mm Hg) and carbon dioxide tension fell to 4.8 kPa (36 mm Hg).

After a week in hospital her visual acuity had fallen to 6/36 in the right eye with no change in the left eye (6/9). Mapping of visual fields showed considerable constriction of the right field and a normal left one (fig 2). Visual evoked responses showed considerable delay and reduced amplitude in the right eye and an abnormal pattern but no delay in the left eye.

She was given oral medroxyprogesterone 20 mg thrice daily as a respiratory stimulant for her hypoventilation, and a weight reducing diet was begun. In view of the cerebral oedema and the risk of further loss of vision she also received a 10 day reducing course of dexamethasone. Her

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symptoms and eyesight improved with treatment. Her visual acuity rose to 6/9 in the right eye within a week, and the papilloedema was thought to be subsiding. She lost 7 kg in weight during her three weeks in hospital. Further ear oximetry before discharge showed that her blood oxygen saturation did not fall as often when she was asleep. She was discharged taking progesterone and a reducing dose of dexamethasone.

FIG 2—Right visual field on admission, with considerable constriction.

FIG 3—Right visual field one month after admission.

She was reviewed two weeks after discharge. Her vision continued to improve, as did her visual fields (fig 3) and the appearance of her discs (fig 4). She had lost a further 2 kg in weight. She continued to take medroxyprogesterone 20 mg thrice daily, having finished a course of corticosteroids 10 days previously.

Discussion

Despite the well known association of papilloedema and respiratory failure the importance of the underlying respiratory disorder has often been overlooked or underestimated.^{4,5} The diagnosis has been missed because the floridity of the fundal changes is often not appreciated, and patients often undergo extensive neurological observation before the aetiology is recognised.

This case was unusual because misting of vision was the only symptom at presentation and the patient did not have a notable

FIG 4—Appearance of fundus after five weeks of treatment.

respiratory history. Although the clinical diagnosis of obesity hypoventilation syndrome was suspected and confirmed by analysis of blood gas tensions and ear oximetry, it was not initially accepted that this was the only cause of her papilloedema.

Papilloedema has been reported previously in association with the pickwickian syndrome.^{6,8} Although there are no reports of patients with the pickwickian syndrome and loss of vision as the primary problem, in patients with serious hypoventilation the history may be limited and only the complications tend to be recognised.⁹

The pathophysiology of papilloedema in respiratory insufficiency is not well understood. Hypercapnia, hypoxia, increased venous pressure, and polycythaemia have all been implicated.^{3,4,5,10} Hypercapnia is generally recognised as the most important factor.^{3,5,10} Raised cerebrospinal fluid pressure is common,¹⁰ and cerebral congestion and oedema have been documented both at necropsy⁶ and on computed tomography of the head.³ We did not perform lumbar puncture, but radiographs of the skull showed changes consistent with raised intracranial pressure.

The treatment is of the underlying respiratory disorder; corticosteroids were used in one case without any change in fundal appearances,⁷ which resolved after respiratory function improved. Progesterone is thought to increase the sensitivity of the respiratory centre drive to carbon dioxide.¹¹ It therefore gives rise to hypoventilation and lowers the tension of carbon dioxide in the arterial blood, which was one of our principal objectives in managing this patient.

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