

CASE REPORT

Myxoedema coma

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THE MECHANISM of coma in myxoedema is uncertain. Hypothermia is not invariably present (Hyams, 1963). There is good evidence that CO₂ narcosis, consequent upon alveolar hypoventilation, is important in some cases and mechanical ventilation may result in prompt lightening of the coma (Nordquist *et al.*, 1960; Buchanan, McKiddie & Reid, 1967). However, two cases have been reported in which there was neither CO₂ narcosis nor hypothermia (Neilson & Ranlor, 1964). The importance of the present case is that serial measurements of arterial oxygen tension have also been made, and some remarkably low levels observed.

Case report

Mrs F.M., aged 60, was admitted to Manchester Royal Infirmary on 11 February 1968. She was confused and unable to give much history but said that she had been unwell for several weeks with tiredness and unsteadiness on her feet. Ten days before admission she had a fall and seemed to deteriorate further. On direct questioning she said that she had suffered from the cold for a long time and had noticed dryness of her skin and a change in her voice. Her son had noticed increasing lethargy and confusion for 1 week. There was no known previous medical or family history.

On examination. She had a bloated facies with periorbital oedema and bruising. She had a slow croaky voice and was mentally confused. Her skin was pale, dry and coarse, and her tongue was enlarged. Clubbing of the fingers was present. She was able to stand but had a grossly unsteady gait.

Temperature, 95°F; pulse, 64/min, regular. BP 130/90 mmHg; JVP, normal. There was oedema of the lower limbs and grossly delayed relaxation of the ankle jerks. She had a very slow respiratory rate and central cyanosis.

Investigations. Hb, 11.1 g/100 ml; PCV, 32%. MCHC, 35%; WBC, 5,200/mm³. Serum cholesterol, 255 mg/100 ml. Serum proteins: albumin, 3.8 g/100 ml; globulin, 2.3 g. Serum electrolytes: Na, 142; K, 3.8; Cl, 101 mEq/l. Blood urea on admission, 60 mg/100 ml. Serum amylase, 70 U/100 ml; serum lipase, 1.0 U/100 ml. PBI, 3.8 µg/100 ml. Thyroid antibodies: Antiprecipitin test, negative; CF test, positive 1 : 5; Tanned red cell agglutinating anti-thyroglobulin titre, positive 1 : 10. Chest X-ray: gross cardiomegaly; ECG: low voltage T waves 1; AVL, V4-6, prominent U waves.

Progress and treatment

On the morning after admission, therapy with L-thyroxine sodium 0.05 mg daily was started. The following day at approximately 15.00 hours the nursing staff found that she was unrousable and gave her oxygen by polymask. Her temperature was 94°F. The respiratory rate was noticed to be very slow and shallow. Her tendon reflexes were unobtainable. She was given triiodothyronine, 60 µg and hydrocortisone, 100 mg i.v. An Astrup reading showed that her Pco₂ was 95 mmHg and pH 7.04, so she was intubated and given 8.4% sodium bicarbonate, 200 mEq. She recovered consciousness approximately 30 min after commencing the above emergency treatment.

TABLE 1. Blood gas measurements

Observation No.	Day	Time	Oxygen	Type of blood sample	Po ₂	Pco ₂	pH	Standard bicarbonate	Buffer base	Base excess	Remarks
Onset of coma at 15.00 hours 13 January 1968											
1	1	15.30	Air	A	40	95	7.04	18.3	38.5	- 7.3	Unrousable
2	1	15.30	Polymask	A	50	115	6.99	18.3	38.5	- 7.3	Unrousable
3	1	16.30	O ₂ via ET*	A	65	100	7.19	27.5	52.5	+ 4.5	
4	1	18.00	IPPV (100% O ₂)	A	—	62	7.28	26.5	48.0	+ 3.0	
5	2	09.30	IPPV (70% O ₂)	A	55	40	7.48	29.0	54.0	+ 6.0	Conscious level unchanged
6	2	12.15	IPPV (70% O ₂)	C	52	48	7.43	29.0	54.0	+ 6.0	
7	2	12.20	Air (2 min)	A	30	51	7.41	29.0	54.0	+ 6.0	Alert
8	2	12.23	Air (5 min)	C	30	53	7.40	29.0	54.0	+ 6.0	
9	3	09.30	IPPV (70% O ₂)	A	115	31.5	7.52	27.0	51.0	+ 3.5	
10	3	09.40	Air (10 min)	A	50	37.0	7.46	26.3	50.5	+ 3.0	
11	3	12.00	Air (30 min)	C	55	35.0	7.48	26.3	50.5	+ 3.0	
12	3	16.00	Endotracheal tube removed	C	60	44.0	7.42	27.0	50.0	+ 3.5	
13	4	10.15	Off O ₂ 4 hr	C	55	40.0	7.45	26.8	51.0	+ 3.5	Fully conscious
14	5	10.00	Off O ₂ 24 hr	C	60	52.0	7.39	28.0	52.5	+ 5.0	Fully conscious
15	6	10.00	Off O ₂ 48 hr	C	60	52.0	7.39	28.0	52.5	+ 5.0	Confused during evening
16	7	10.05	Air	No reading obtained							Confused at times
17	7	10.05	O ₂ Ventimask	C	50	62.0	7.35	29.5	52.5	+ 6.5	Blood urea 67
18	8	02.00	O ₂ Ventimask	C	—	45.0	7.31	21.0	40.0	- 4.0	Deterioration, unrousable
19	8	10.00	Off O ₂ (10 min)	C	40	70.0	7.30	28.5	52.0	+ 5.5	Unrousable
20	9	15.45	Off O ₂ (10 min)	A	38	62.0	7.35	28.5	53.5	+ 5.5	Unrousable
21	9	10.15	Off O ₂ (10 min)	C	25	95.0	7.22	28.5	54.0	+ 5.8	Stuporous
22	10	10.00	Off O ₂ (10 min)	C	20	72.0	7.26	25.5	49.5	+ 2.3	Stuporous
23	10	10.00	Off O ₂ (10 min)	A	20	63.0	7.30	25.8	49.5	+ 2.3	Stuporous, oliguric
24	11	14.45	Off O ₂ (10 min)	A	17	60.0	7.33	26.7	51.5	+ 3.5	Stuporous
25	11	10.15	O ₂ Ventimask	C	45	75.0	7.1	18.3	—	C. - 5.0	Coma, intubated blood urea 260
26	12	10.10	IPPV 70% O ₂	A	150	77.0	7.14	19.7	—	C. - 3.0	Coma
27	13	12.45	Patient died								

* Endotracheal tube.

The patient was referred to the Respiratory Unit and maintained by intermittent positive pressure ventilation (IPPV). Details of subsequent blood gas measurements are set out in Table 1. pH and P_{CO_2} estimations were obtained by the micro-Astrup technique using capillary or arterial blood as indicated. P_{O_2} was measured with a Clarke type platinum electrode (Radiometer, Copenhagen).

On the following day she was conscious although restless and confused. When IPPV was interrupted she became severely hypoxic (observations 5–8). Maintenance therapy consisted of triiodothyronine 10 μ g b.d. (subsequently increased to 40 μ g 6-hourly), hydrocortisone, 20 mg b.d. and tetracycline, 250 mg 6-hourly. On day 3 she was less stuporous and very conscious of the endotracheal tube. She breathed normally when off the respirator and was adequately oxygenated (observations 10–12) so the tube was removed. On day 4 she remained alert and conversational, breathing normally when off the respirator and was adequately oxygenated on air (observation 13). A chest X-ray was normal. On the evening of the 5th day she became confused and was given oxygen. The following day she was again hypoventilating and intermittently confused, but was adequately oxygenated on 28% oxygen by ventimask (observation 16). On day 8 she became extremely drowsy responding only to painful stimuli. Hypercapnia and hypoxia became more marked (observations 18 and 19). Further deterioration of blood gas measurements occurred on the following day. On day 10 she remained extremely drowsy. She was noted to be oliguric. She was profoundly hypoxic when oxygen was interrupted (observations 21–23). On day 11 she was unrousable and artificial respiration was restarted. At this stage her blood urea had risen to 260 mg/100 ml. Multiple bruising was apparent. Despite adequate correction of the blood gases, and maintenance of fluid balance, no improvement in conscious level was obtained and she died 2 days later.

Necropsy

Macroscopically there was complete atrophy of the right lobe of the thyroid and an encapsulated mass in the atrophic left lobe. Histologically the mass was a thyroid carcinoma of mixed papillary follicular type. There was no evidence of invasion or metastases. Elsewhere the thyroid tissue had been

completely replaced by fibrous tissue. Interspersed among connective tissue there were focal collections of thyroid cells showing Askanazy cell (Hürthle cell) change, with surrounding cuffs of lymphocytes.

Other findings were cardiomegaly with hypertrophy of both ventricles and mild coronary atherosclerosis. There was a moderate pericardial effusion and small bilateral pleural effusions.

In the lungs there was a small infarct in the left upper lobe. PAS stains did not reveal any mucopolysaccharide infiltration of the alveolar septa as described by Naeye (1963) in four out of fourteen cases of myxoedema. There was acute pancreatitis and some adjacent fat necrosis. The kidneys showed a few small congenital cysts. Microscopically there was mild diffuse interstitial fibrosis and hyalinization of many glomeruli and small vessels. The renal tubules were normal. The brain was normal.

Discussion

Hypothermia was clearly not an important factor in the present case. There would seem to be good evidence that hypercapnia precipitated the onset of coma. Later, remarkably low oxygen tensions were recorded.

In the present case IPPV was continued for only 48 hr. In both cases reported by Nordquist *et al.* (1960), IPPV was required for 2 weeks. In view of the gradual relapse of this patient which was initially heralded by quite mild hypoventilation, it may be advisable to perform early tracheostomy and be prepared to maintain strictly normal ventilation for a prolonged period in similar cases.

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