

AN UNUSUAL CASE OF ATAXIA

Pages with reference to book, From 89 To 90

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INTRODUCTION

Intensive investigations including high sophisticated technology is helpful in reaching a diagnosis in many cases but equally important is a thorough detailed history and vigilant examination for the patient. Indeed a simple review of treatment charts may on occasion find the answer to a problem where high technology fails to do so, as evidence from the following case.

CASE REPORT

A 72 year old lady was admitted with 2 weeks history of increasing cough and breathlessness. She was a known case of chronic obstructive airways disease but had a very good quality of life with no mobility problems. Her condition worsened just before admission. She was moderately obese, drowsy, centrally cyanosed with flapping tremors of her fingers. Her blood gases confirmed ventilatory failure, PH was 7.37, PCO₂ 68.4 mmHg. PO₂ 29.5 mmHg and standard bicarbonate 33.2. She was also mildly hypothyroid with TSH of 7.31 IU/L (0.3 - 3.8 IU/L) and T₄ 37 nmol/liter (70-140 nmol/liter). Her blood sugar was elevated at 15.8 mmol/liter and initially needed a small dose of oral hypoglycaemic agent. She was treated with nebulized bronchodilators, antibiotics and graduated O₂ inhalation but her condition deteriorated. She was transferred to the Intensive Therapy Unit where she was started on Dopram infusions. In addition, she was given WAcetazolamide with subsequent oral replacement i.e. Dichlorphenamide which was continued. This increases alveolar ventilation by inducing metabolic acidosis and thus decreasing CO₂ retention¹⁻⁵. With this treatment she improved and her medication on discharge was Salbutamol, Beclomethasone inhaler, with tailing off of oral steroid, Dichlorphenamide, Thyroxine and Frusemide. She was re-admitted 2 weeks later with worsening of her breathing but this time she had an unsteady gait which became more obvious when her breathing improved with treatment as before. Neurological assessment at this stage, showed a relatively well patient, who had a very unsteady gait. Despite adequate replacement of Thyroxine, her ataxia did not improve. Other non invasive investigations including ultrasound of the abdomen and CT scan did not reveal any cause to account for her ataxia. She was transferred to a rehabilitation ward. There was slight improvement but quite extensive domiciliary and social arrangements were made solely due to her ataxia. It was then realised that Dichlorphenamide might be the cause and hence it was discontinued. Prior to discharge her gait improved to a great extent. She was reviewed 4 weeks after her discharge and she was fully recovered with a normal gait, despite continued vitalographic evidence of fairly severe airway obstruction.

DISCUSSION

In a case of ataxia, apart from hypothyroidism, one has to consider paraneoplastic syndrome affecting cerebellum, but in our case there were no other cerebellar signs except ataxia. There was no history of excessive alcohol intake. Her chest x-ray was normal, so was abdominal ultrasound, with no ovarian or pelvic mass. Ataxia is reported as a side effect of Dichlorphenamide^{6,7}. The case demonstrates that extensive investigation is no substitute for simple clinical vigilance.

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