**Neurology**

**A bed bound patient**

**G G Hanna, G V McDonnell**

Answers on p 421.

A 62 year old retired woman was admitted to hospital with a 10 month history of pain and weakness in the muscles of both arms and legs. Her problems began with episodic weakness in both legs such that on occasions her legs gave way and she fell to the ground. She had a gradual deterioration in her muscle strength and after a fall five months before admission she became confined to bed.

Over the month before hospital admission her weakness had progressed to the extent that she was unable to lift her head off a pillow. She had also developed pain in the muscles of the arms and legs. Of note, she had no sensory, visual, speech, or swallowing disturbance. Relevant past history included epilepsy since the age of 43, hypertension, hypercholesterolaemia, and diet controlled type II diabetes mellitus.

Drug therapy was as follows: folic acid 5 mg once daily, lisinopril 5 mg once daily, aspirin 75 mg once daily, carbamazepine 200 mg twice a day, and cocodamol as required. She was a smoker of five cigarettes per day and was teetotal.

On examination she was dehydrated but apyretic and not distressed. She had marked proximal muscle weakness and tenderness but no fasciculation. There was grade 2/5 power in neck flexion and extension. Shoulder abduction, hip flexion, and knee extension were all weak bilaterally with 3/5 power. Finger abduction, power grip, ankle dorsiflexion, and plantar flexion were all modestly weak (4/5). The other muscle groups had full power. Upper limb reflexes were present with reinforcement, lower limb reflexes being absent, and the plantar responses flexor. There was no objective sensory deficit and the remainder of the examination was normal.

Investigations at this stage included full blood profile (haemoglobin 129 g/l, leucocyte count 11.2 × 10^9/l), erythrocyte sedimentation rate (ESR) 120 mm/hour, urea and electrolytes (sodium 141 mmol/l, potassium 4.7 mmol/l, urea 8.3 mmol/l, creatinine 83 µmol/l), creatine kinase 533 U/l, creatine kinase MB fraction 34, electrocardiography (normal), and chest radiography (normal).

**QUESTIONS**

(1) What is the differential diagnosis and what would you do next?

(2) How would you confirm the diagnosis?

(3) What are the associations of this condition?

(4) How would you manage this patient?

**Postgrad Med J 2003;79:418**

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**Diabetes**

**Low HbA1c levels in a poorly controlled diabetic**

**A R Vasudevan, S Ghosh, R Srivastava, LDKE Premawardhana**

Answers on p 421.

A 52 year old man was referred to the diabetic clinic because of weight loss and persistent osmotic symptoms. He was diagnosed three years before presentation and was on diet therapy. His general practitioner was concerned that he had a “sinister cause” for his symptoms because his glycated haemoglobin (HbA1c) levels were “near normal” when repeated on several occasions (measured using the Menarini high performance liquid chromatography method, with a reference range of 4%–6%). He was in remission from Hodgkin’s disease. Initial clinical examination was unremarkable. Laboratory data were: haemoglobin 155 g/l, white cell count 4.81 × 10^9/l (normal differential count), platelet count 214 × 10^9/l; liver enzymes, renal function, and microalbuminuria screen were normal. Other results are shown in table 1.

**QUESTIONS**

(1) What do the data demonstrate?

(2) What is the differential diagnosis and what would you do next?

(3) What is the pathophysiological basis of the discrepancies observed and how would you assess this man’s long term glycaemic control?

**Postgrad Med J 2003;79:418**

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Submitted 27 August 2002

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**Self Assessment Questions**

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A bed bound patient

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