A 67-year-old man was transferred to the Emergency Department of our hospital for emergent evaluation of paraplegia and oliguria, from the local hospital of the nearby town, where he was admitted complaining from sudden, painless, progressive bilateral leg weakness and oliguria 4 days earlier. He gave no history of hypertension, diabetes mellitus or hyperlipidaemia, and had a negative family history of aortic diseases. On initial evaluation, the patient had a blood pressure of 131/71 mm Hg. His oral temperature was 36.4°C, pulse rate was 82 beats/min and respiratory rate was 20/min. He presented complete flaccid paraplegia with oliguria (urinary output <400 ml/d) and urinary retention, loss of pain and temperature sensation, vibration and position sense below the TH7 level bilaterally. Other general physical examinations were unremarkable. Laboratory tests showed a white blood cell count of 19.80×10^9/l, haemoglobin concentration of 109 g/l, blood urea nitrogen concentration of 50 mmol/l, blood creatinine concentration of 820 μmol/l, sodium concentration of 114 mmol/l, and potassium concentration of 4.6 mmol/l. The liver function tests were normal and other observations were unremarkable. Later thoracic and lumbar MRI revealed swelling of thoracolumbar spinal cord, with no enhancement on T1-weighted images (wi) and increased signal on T2-wi at the TH9-TH12 levels, suggesting cord ischaemia. At the same MR sequences, the double lumen of the descending aorta involving bilateral renal arteries indicated dissection in both sagittal and axial images. The diagnosis of Stanford type B acute aortic dissection was confirmed. When patients present with or develop signs and symptoms of paraplegia without obvious cause, aortic dissection should be considered, even without the presence of characteristic thoracic pain.

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Conclusions A 67-year-old man was transferred to the Emergency Department of our hospital for emergent evaluation of paraplegia and oliguria, from the local hospital of the nearby town, where he was admitted complaining from sudden, painless, progressive bilateral leg weakness and oliguria 4 days earlier. He gave no history of hypertension, diabetes mellitus or hyperlipidaemia, and had a negative family history of aortic diseases. On initial evaluation, the patient had a blood pressure of 131/71 mm Hg. His oral temperature was 36.4°C, pulse rate was 82 beats/min and respiratory rate was 20/min. He presented complete flaccid paraplegia with oliguria (urinary output <400 ml/d) and urinary retention, loss of pain and temperature sensation, vibration and position sense below the TH7 level bilaterally. Other general physical examinations were unremarkable. Laboratory tests showed a white blood cell count of 19.80×10^9/l, haemoglobin concentration of 109 g/l, blood urea nitrogen concentration of 50 mmol/l, blood creatinine concentration of 820 μmol/l, sodium concentration of 114 mmol/l, and potassium concentration of 4.6 mmol/l. The liver function tests were normal and other observations were unremarkable. Later thoracic and lumbar MRI revealed swelling of thoracolumbar spinal cord, with no enhancement on T1-weighted images (wi) and
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