Case Report

The value of computed tomography and magnetic resonance imaging to diagnose rhabdomyolysis in acute renal failure

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Introduction

Rhabdomyolysis results from various clinical conditions such as drug abuse, alcohol, neuroleptic agents, extreme pyrexia, direct trauma, compression, immobilization, ischaemia, excessive muscular activity, polymyositis, and metabolic/genetic disorders [1]. Clinically it is accompanied by release of creatine kinase, myoglobin, lactate dehydrogenase, phosphate, and potassium from muscles, causing increased serum concentration of these substances. Rhabdomyolysis can be complicated by acute renal failure [2].

Diagnosis of rhabdomyolysis can usually be made easily based on elevated serum creatine kinase level. In some cases the presence of rhabdomyolysis is not immediately evident, e.g. when a patient is admitted several days after the event. In such patients the increase in creatine kinase, if any, is minimal because creatine kinase peaks within 24 h and subsequently decreases by 39% every day [1].

Recently magnetic resonance imaging (MRI) [3,4] and computed tomography (CT) [5] have been introduced to visualize rhabdomyolysis. Russ and Dillingham [5] suggested that CT can be helpful in diagnosing rhabdomyolysis in cases of acute renal failure of unknown aetiology. Here we report such a case where CT and MRI were helpful in documenting a history of rhabdomyolysis as the cause of acute renal failure of unknown origin.

Case

A 57-year-old male was admitted to our hospital for acute renal failure and immobilization. He had a history of depression and had been treated with sulpiride, thioridazine, and biperiden for about 37 years. On 13 June 1998 he complained of progressive weakness and fever. On 4 July his sister found him lying on the floor at home with extreme fatigue and inability to move. There was nobody who witnessed him having seizures. At a nearby hospital he was found to be febrile and was diagnosed as having acute renal failure with blood urea nitrogen of 143 mg/dl and serum creatinine 15.0 mg/dl. Other laboratory data included potassium 5.0 mmol/l, uric acid 14.8 mg/dl, and creatine kinase 295 IU/l (normal, 30–150). Treatment with cefoperazone/sulbactam and intravenous frusemide 60 mg/day was started. He was transferred to our hospital for the management of acute renal failure on 8 July 1998.

The patient had a history of familial adenomatous polyposis (Gardner syndrome) for which he underwent total colectomy in 1967. In 1992 he was found to have rectal cancer, pancreatic cancer, and diabetes mellitus when rectal resection and duodenopancreatectomy were performed. Osteoma of the skull and the mandible were resected in 1992. In 1994 he underwent partial hepatectomy and lipiodolization for multiple hepatic tumours. In 1996 he was diagnosed to have pigmentary degeneration of the retina. The patient had never previously consumed alcohol or tobacco.

On transfer, he was lethargic and mildly obtunded. Blood pressure was 142/78 mmHg, pulse ranging from 60 to 106/min, respiration 16/min, and temperature between 36.3 and 38.6°C. The chest and abdomen were unremarkable apart from the surgical scars. There was 2+ oedema in the upper extremities. Muscular tenderness was noted in the left upper arm and left inner thigh. Neurology showed nystagmus, sluggish speech, asterixis, and continuous rhythmic involuntary movement of the eyelids, head, mouth, and upper extremities, but reflexes were normal.

Laboratory data showed leukocytes 14 290/µl with 88% neutrophils, haemoglobin 9.4 g/dl, haematocrit 27.9%, platelet 192 000/µl, total protein 5.8 g/dl, albumin 3.1 g/dl, blood urea nitrogen 141 mg/dl, creatinine 14.8 mg/dl, uric acid 13.5 mg/dl, sodium 130 mmol/l,
potassium 4.7 mmol/l, chloride 97 mmol/l, bicarbonate 15.6 mmol/l, calcium 7.9 mg/dl, phosphate 6.9 mg/dl, aspartate aminotransferase 30 IU/l, lactate dehydrogenase 776 IU/l (261–483), alkaline phosphatase 825 IU/l (115–359), gamma-glutamyl transpeptidase 136 IU/l (10–47), creatine kinase 159 IU/l (62–287), aldolase 8.8 IU/l/37 C (1.7–5.7), gamma-globulin 18.7%, and C-reactive protein 5.0 mg/dl. Urinalysis revealed negative protein, 3+ blood, and trace sugar, with no evident proteinuria, 3+ leukocytes. Urinary sodium excretion had not been clinically diagnosed in 27% of the cases. This is due to the fact that (i) because serum creatine kinase is not always significantly elevated in ARF, especially when cases are admitted later, (ii) a history is difficult to obtain in patients with altered mental status, and that (iii) muscular symptoms may be minimal, particularly in cases with non-traumatic rhabdomyolysis. Therefore, non-traumatic rhabdomyolysis is an underdiagnosed cause of acute renal failure [6,9].

Recently it has been reported that CT and MRI are useful in the diagnosis of rhabdomyolysis. Muscle tissue having undergone rhabdomyolysis appears as a low-density area in CT [10,11], but this finding is not specific and can be seen in pyogenic myositis, abscess, and neoplasm [5]. On the other hand, Russ and Dillingham [5] found that on CT, four of eight patients with rhabdomyolysis had had high-density areas in the affected muscle. Plain muscle X-rays were negative in all the cases. The affected muscles were initially swollen, then calcified, and finally shrank while calcifications disappeared [5]. Therefore, high-density areas on CT may point to rhabdomyolysis as well. Indeed, Towers et al. [9] reported that calcification in the psoas muscle had led to the recognition of rhabdomyolysis in a case of acute renal failure which had initially been attributed to sepsis and hypovolaemia.

It has been reported that MRI is useful in the diagnosis of rhabdomyolysis as well. Zagoria et al. [4] reported hyperintensity in T2 images in two cases with rhabdomyolysis. Shintani and Shiigai [3] also described...
Fig. 2. T2-weighted images of magnetic resonance imaging showing high signal intensity in the left obturator, pectineus, and adductor muscles.

Fig. 3. Significant uptake in the right shoulder, right elbow, left upper arm, front chest, and left femoral regions in (A) gallium scintigram, and (B) 99mTechnetium hydroxy methylene diphosphonate bone scan.

that in four cases of rhabdomyolysis of various aetiologies, high-intensity lesions in T2-weighted images were observed as early as 2 days after admission and had disappeared by 17–46 days. Although MRI findings may not be entirely specific and may have represented reversible oedema and inflammation [3,4], MRI is still considered to be invaluable in the early diagnosis, because its sensitivity is definitely superior to that of CT or ultrasonography [10].

99mTechnetium pyrophosphate bone scan was able to detect tissue necrosis as early as 18 h after the onset [12] and was also sensitive in detecting muscle injury when physical examinations were negative [13]. While diuretic-phase hypercalcaemia makes physicians suspect the previous presence of rhabdomyolysis [14], the changes in CT, MRI and bone scan should be seen well before hypercalcaemia. It is suggested that CT and MRI should be performed in patients with acute renal failure of unknown aetiology.

Our patient was considered to have rhabdomyolysis, presumably associated with a long-term use of antipsychotic agents. The patient appears to fulfil criteria of neuroleptic malignant syndrome [15] because he had pyrexia, extrapyramidal signs, and consciousness disturbance. However, some of the symptoms could also have been due to uraemia or medications. The reason why our patient experienced rhabdomyolysis after 37 years of chronic antipsychotic treatment remains to be determined. The neuroleptic malignant syndrome may be triggered by infection or dehydration [16], either of which could have been present in our case. Although there is little information regarding preferential involvement of specific muscle groups in the neuroleptic malignant syndrome, muscle lesions in psoas and adductor have also been described in alcoholics and drug abusers [13,17].

References

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