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Fever with acute renal failure due to body massage-induced rhabdomyolysis

Sir,

Body massage, known as complementary and alternative medicine, is very popular in the world [1,2]. [It is considered a relatively safe therapy for the senior population that attempts to relieve pain or improve quality of life.] However, in one incident, an 88-year-old man developed rhabdomyolysis in the aftermath of a body massage session.

The gentleman was quite healthy in the past, with a history of type 2 diabetes mellitus under well-controlled by diet. He presented with a fall-down accident due to weakness of four limbs, fever (~38°C), acute renal failure (ARF) (creatinine 1.7 mg/dl, urea nitrogen 22 mg/dl) and mild proteinuria (initial urinalysis: protein ++, occult blood +++, WBC 5–10/HP, RBC 0–2/HP), but no significant ecchymosis or swelling of whole body were found. Though urinary tract infection was once suspected at a local hospital for his fever, leucocytosis (WBC 21.5×10^3/μl, neutrophils 89%) with mild pyuria, it was excluded by repeated urinalysis (protein 100 mg/dl, pH 5.0, WBC 0–2/HP, RBC 0–2/HP) and finally sterile cultures of urine and blood. His blood biochemistry showed as follows: aspartate aminotransferase 322 U/l, alanine aminotransferase 72 U/l, lactic dehydrogenase 1224 U/l, creatine phosphokinase (CPK) 7940 U/l with 100% of CPK-MM form, potassium 6.5 mEq/l, creatinine 1.2 mg/dl and urea nitrogen 19 mg/dl. After adequate hydration with intravenous fluid, solute alkaline diuresis and rapid-acting insulin, his serum potassium level normalized within 6 h and his fever subsided with regained strength 3 days later. The time–concentration curve of CPK was almost parallel to that of WBC of peripheral blood and serum C-reactive protein (CRP) level (Figure 1), indicating that the extent of inflammation was closely related to rhabdomyolysis process.

An enquiry disclosed the habit of the patient: for more than 40 years, he had regularly received body massage for 1–2 h every other day. The afternoon before this accident, he received a body massage session for 2 h served by two new massagists at the same time instead of one. The strength of this massage session was significantly stronger than that of the past. He drank little water before and after the massage session. Generalized muscle pain and soreness developed that night but was not given attention.

Compression or pressure-induced rhabdomyolysis has been reported in coma or immobilized patients [3,4],

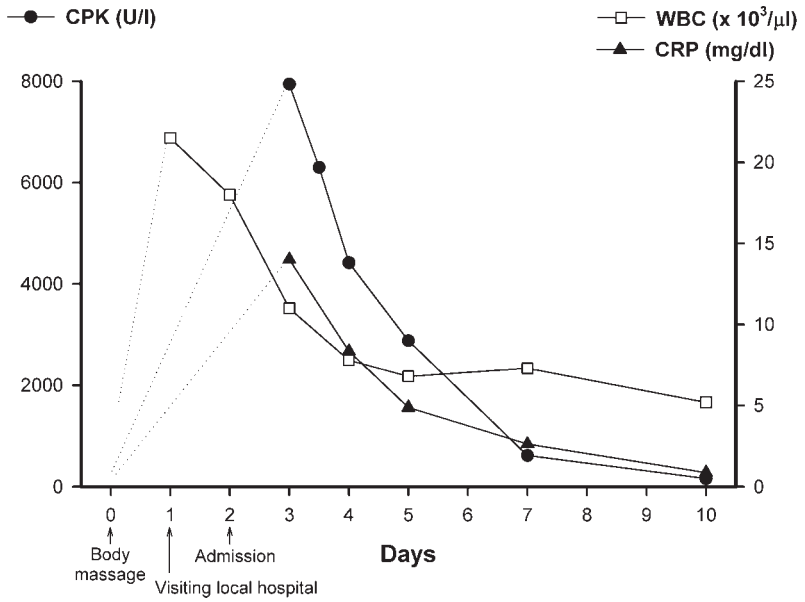

Fig. 1. Serum CPK level and corresponding white blood cell count (WBC) of peripheral blood and serum C-reactive protein (CRP) level in the patient with body massage-induced rhabdomyolysis. Note that the time–concentration curve of CPK was almost parallel to that of WBC and CRP, indicating that the extent of inflammation was closely related to the rhabdomyolysis process.

prolonged cardiopulmonary resuscitation [5] and obese men who received bariatric surgery [6], but it has never been associated with body massage. Senior and diabetic patients need to be warned that vigorous body massage may cause dangerous complications such as rhabdomyolysis. In addition, the people receiving body massage should drink adequate amount of water before and after the massage session so as to prevent unusual episodes of rhabdomyolysis-associated ARF, which is exacerbated by volume depletion [3,4].

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Effect of ascorbic acid supplementation on plasma isoprostanes in haemodialysis patients

Sir,

We would like to report the effect of ascorbic acid (AA) supplementation on plasma F_2-isoprostanes in haemodialysis patients with anaemia and hyperferritinaemia. The F_2-isoprostanes, free-radical oxidation products of arachidonic acid, have been quantified in human models of increased oxidative stress [1], including haemodialysis patients [2], and are a useful measure of *in vivo* lipid peroxidative damage [3]. While AA supplementation may improve anaemia in haemodialysis patients with iron-overload and normal iron status [4,5], the effects on parameters of oxidative stress are conflicting. Studies have shown an extra-cellular pro-oxidant effect *in vitro* with bolus doses of AA [6], but intracellular anti-oxidant effects *in vivo* over 8 weeks of AA supplementation [7]. Recently, Fumeron and colleagues have demonstrated that there is no effect of short-term oral AA administration on oxidative stress markers [8]. As discussed in their paper, these paradoxical effects may relate to increased presence of catalytic transition metal ions such as iron and oxalate which favour pro-oxidant activity, AA doses used, whether AA was administered orally or intravenously (IV), and differing assays for Reactive Oxygen Species detection. We have extended the findings of Fumeron *et al.* by examining the effect of both oral and i.v. AA administration on plasma F_2-isoprostanes and serum oxalate levels, as the latter may counteract the antioxidant effects of ascorbic acid.

In a sequential study, we randomly assigned 21 haemodialysis patients with mild anaemia (mean Hb 114 g/l ± SE 2.2) and hyperferritinaemia (mean ferritin 632.0 ug/l ± 59.4) to either 250 mg of oral ($n = 10$) or IV ($n = 11$) AA three times a week post-dialysis [9]. We measured plasma F_2-isoprostanes before treatment with AA, after 8 weeks of AA and finally following a 4 week washout period. Plasma F_2-isoprostanes were measured on EDTA plasma samples collected into cold tubes protected from *in vitro* oxidation by the addition of 20 µg of butylated hydroxytoluene, and assayed using a combination of silica and reverse-phase cartridges, high-performance liquid chromatography and gas chromatography mass spectrometry using electron capture negative ionization [10]. As previously reported [9], plasma ascorbic acid and serum oxalate levels increased significantly following treatment with oral and IV AA, and the increase was not significantly different between the two routes of administration. We did not observe a significant change in the plasma F_2-isoprostane level with either oral or IV AA from baseline to week 8 [week 0: 1992.0 pmol/l (95% CI 1603.8–2472.3) *vs* 1908.4 pmol/l (95% CI 1473.9–2470.6), $P = 0.60$]. Additionally, plasma F_2-isoprostane level was not statistically different after the washout period [week 8: 1908.4 pmol/l 95% CI (1473.9–2470.6) *vs* week 12: 1979.7 pmol/l (95% CI 1586.0–2471.3), $P = 0.62$]. Furthermore, the route of AA administration, IV or oral, had no effect on plasma F_2-isoprostane level when adjusted for baseline values ($P = 0.68$).

We acknowledge that the lack of effect on oxidative stress may relate to the low dose of AA used, short duration of therapy and lack of co-administration with vitamin E. The lack of observed antioxidant activity with AA supplementation may also be due to the associated elevation in serum oxalate levels [11] and hyperferritinaemia, which are known to increase pro-oxidant activity. Finally, although we did not specifically measure for markers of inflammation, ferritin, a known acute-phase protein, was unchanged during the study period.

In summary, our study provides further supportive evidence that short-term treatment with either oral or IV AA has no effect on markers of oxidative stress despite an increase in plasma ascorbic acid levels.

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