pressure 139/87 mmHg, weight 81 kg, and height 180 cm. The laboratory parameters were urea 61 mg/dL, creatinine 1.7 mg/dL, creatinine clearance 44.37 mL/min, microalbuminuria 11.75 mug/min and hemoglobin A1c 5.5. Other parameters were normal. In January of 2006 the patient began a macrobiotic diet, consisting of wholemeal cereals, like barley and wheat, see algae, and vegetables. He avoided eating meat, milk or milk derivatives. In October of 2006 he had blood pressure 138/82 mmHg and weight 79 Kg. Laboratory findings were urea 46 mg/dL, creatinine 1 mg/dL, creatinine clearance 81.57 mL/min, microalbuminuria 29,31 m/min, hemoglobin A1c 5.9, albumin 4.2 g/dL, total proteins 6.7 g/dL, total cholesterol 150 mg/dL, triglycerides 168 mg/dL, and the other parameters were normal. These findings were similar in further analyses three and six months later. He did not appear malnourished. No changes in the treatment were prescribed and no other cause could be found to explain the improvement in renal function.

Macrobiotic diet is an extreme form of vegetarianism combined with some thoughts derived from Buddhist Zen philosophy. Eating and quality of food are thought to affect the health.[3] Macrobioticdiet emphasizes the use of organic products, like cereals, fruits, algae, vegetables, fermented soy, combined according to the principles of the balance between the yin and yang properties.[4] Some reports demonstrate the anti-tumoral effect, particularly in breast tumors. Women who make a macrobiotic diet apparently excrete a greater amount of estrogens in stool and urine, have lower blood levels of estradiol and have lower risk of developing a breast carcinoma.[5,6] Some studies about the macrobiotic diet as treatment of different neoplasms, like melanoma or pancreas cancer, have been published. An improvement of the clinical picture and of the quality of life of the patients was observed.[6,7]

The effect of macrobiotic diet in diminishing cardiovascular risk has been reported.[8,9] An improvement in total cholesterol, an increase in HDL-cholesterol and an improvement in blood pressure were demonstrated, but no effect on the progression of the DN could be showed.

The potential danger of the diet is that if strictly followed can cause malnourishment, anemia due to vitamin B_{12} deficiency and scurvy.[10]

The reported patient presented a - CRF secondary to DN of six years. After 10 months of macrobiotic diet an improvement in renal function was observed, without signs of malnourishment and no other cause could be found to explain this improvement.

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Acute renal failure due to carnitine palmitoyl transferase deficiency

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To the editor: We present a male, who developed severe habdomyolysis after an infectious episode leading to acute oliguric renal failure that required hemodialysis. He was 18 years old and had a history of febrile episodes in the childhood and tonsillectomy. He referred «dark-colored urine after strenuous efforts» and consumed no toxics.

He was evaluated at the Emergency Room because of generalized muscle pain and odynophagia. Physical examination was unremarkable except for hyperemic pharynx and intense pain on muscle palpation.

Laboratory parameters were: Biochemistry: Urea 49.6 mg/dL, Cr 2.05 mg/dL, Na 132 mEq/L, K 4.9 mEq/L, ionic calcium 4 mEq/L, uric acid 4.5 mg/dL, CK 285.390 U/L; CK-MB 4353 U/L. Arterial blood gas analysis: pH 7.40; pCO2 33 mmHg; HCO3 20 mmol/L ; PO2 98 mmHg , Sat 97.4%. Blood cell counts: Hb: 17.5 g/dL; hematocrit: 47.6% ; MCV: normal, leucocytes: 13.210 (neutrophils: 88%), other parameters were normal. Urine: density 1010; pH: 5.5, leucocytes 25/ul; erythrocytes > 300 and normal sediment. Ions in urine: Na 77; K 25 ; FENa < 1. Toxics in urine: negative.

Renal ultrasound was normal. The scintigraphy showed abnormal distribution of radiotracer with intense deposits in the muscles of the thorax, abdomen and extremities (fig. 1). A diagnosis of acute renal failure due to severe rhabdomyolysis of unknown origin was made and the patient was admitted to the hospital. Treatment was initiated with intravenous fluid therapy, urine alkalinization and manitol. Forty-eight hours later he developed progressive increase of creatinine values to 5.3 mg/dL, oliguria and cardiac failure and hemodialysis was performed. A total of 6 sessions were required. In the 9th day the patient recovered diuresis. He was discharged with polyuria and decreasing creatinine.

Posttraumatic, ischemic, toxic, infectious, endocrine and immunological etiologies were ruled out. A muscle biopsy

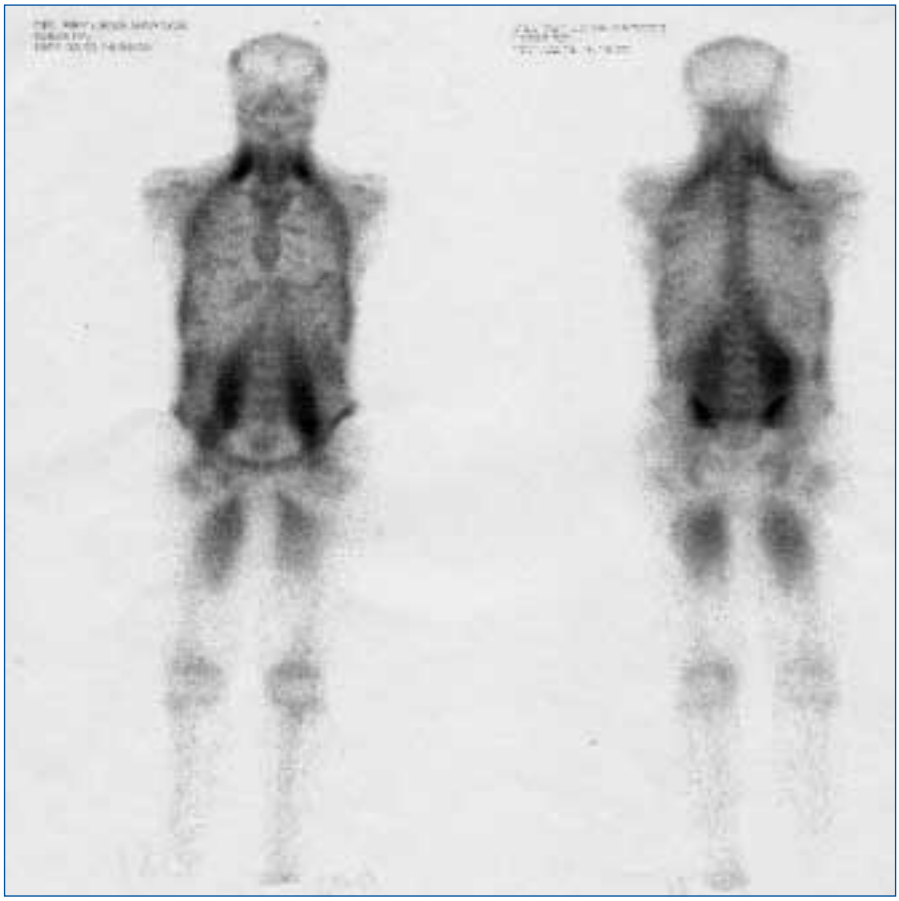

Figure 1. Scintigraphy with abnormal distribution of the radiotracer showing severe deposition at the abdominal muscle groups and the extremities compatible with rhabdomyolisis.

from the deltoid muscle was performed. The immunohistochemistry revealed type 2 carnitine-palmitoyl transferase (CPT) deficiency. The familial study disclosed that one sister was also affected.

Metabolic myopathies are a small percentage of rhabdomyolysis causes. However they are a preventable cause of acute renal failure, which very often goes unnoticed. CPT deficiency is the most frequent metabolic myopathy.[1,2] Lipids are an important energy source for resting muscles and during sub-maximal exercise. In case of CPT deficiency fatty acids do not enter in the mitochondria to be oxidized and no energy is obtained. The consequence is muscle destruction or rhabdomyolysis. This condition is the most frequent cause of recurrent myoglobinuria with no clear trigger and should be always suspected in these patients.[3]

The clinical picture consists of recurrent episodes of muscular weakness, myalgias, rhabdomyolysis or acute renal failure.[4,5] The episodes can be elicited by viral infections, exercise, prolonged fasting or fever. In two thirds of the patients the disease presents in the first or second decade.

The treatment is to avoid the factors that can trigger rhabdomyolysis, like prolonged fasting, to eating a low-fat high-carbohydrate diet, frequent meals and with excessive carbohydrates intake after exercise.

Renal acute failure is due to intratubular deposition of myoglobin. Early volume reposition,[6] and urine alkalinization[7] with calcium and potassium monitoring are fundamental to prevent it. Manitol use in acute renal failure is controversial, but in case of rhabdomyolysis it appears to reduce interstitial edema and to uptake free radicals. The beneficial effect has been demonstrated in patients with serum CK levels higher than 30,000 U/L.[8]

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Acute post-estreptoccocal glomerulonephritis in the elderly

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To the editor: Post-streptococcal glomerulonephritis (PSGN) is the prototype of acute glomerulonephritis. It is heralded by a group A b-hemolytic streptococcus infection.[1,8] The evolution is benign in children, and poorer with older age[2] and in case of renal acute failure.[5] Approximately in 95% of the cases renal function is recovers in within 3-4 weeks. The evolution in the elderly is less predictable.[1,4,5] Irreversible renal failure occurs in less than 1% of children and in a higher percentage of adults.[6,7,9] The treatment consists of antihypertensive drugs, diuretics and antibiotics, and sometimes hemodialysis may be required.[1,3,8] Immunosuppressive therapy is indicated in case of glomerulonephritis with crescent formation.[5] Steroids appear to be efficacious in adults with PSGN and nephrotic syndrome.[3]

We present a 78 years old male who was admitted because of cardiac failure of 48 hours and oligo-anuria, together with renal function worsening in the last week. The blood analysis revealed