The treatment of spleen abscess consists of appropriate systemic antibiotic and removal of pus by percutaneous catheter, splenotomy or splenectomy. E. corrodens is usually susceptible to a broad-spectrum antibiotic such as ampicillin, cephalosporins, fluoroquinolones and thienamycins. Finally, we choose radiographically guided percutaneous drainage for two reasons: the peripheral location of the abscess, which was easy to puncture for confirmation and placement of a drainage catheter, and is a successful procedure with very high cure rate and very low rate of complications (over 80% and less than 5%, respectively).

J.M. Ramos
E. Pacho
B. García-Valle
M. Cuenca
A. Franco
M.C. Pontes
Department of Internal Medicine, Medical Microbiology and Radiology, Fundación Jiménez Díaz, Madrid 28040, Spain.

References

Acute serum lipid changes in a renal transplant recipient on intravenous cyclosporin upon administration of an intravenous lipid solution

Sir,

We wish to report an acute increase in serum lipids on the administration of intravenous cyclosporin in a renal transplant recipient on intravenous cyclosporin and steroids. A 53 year old male developed perforated diverticular abscess 3 months after a renal transplantation. He underwent a Hartmann's procedure. This was complicated by a paralytic ileus and parenteral nutrition was initiated to establish nutritional support. He was commenced on a peripheral regimen of Vitrimix (Kabi Pharmacia) consisting of 500 ml of 20% Intralipid (fractionated soybean oil 100 g, egg-yolk phospholipids 12 g, glycerol 22.5 g/ml) and 1,600 ml of 0.9% glucose. Within 24 hours, his blood sample was turbid in nature and his serum total cholesterol concentration rose from 5.2 mmol/l to 10.0 mmol/l, and serum triglyceride concentration rose from 4.2 mmol/l to 15.1 mmol/l concurrently. His serum lipid abnormality persisted and the Vitrimix was discontinued after 48 hours. He was then commenced on an alternative parenteral nutrition formulation consisting of 500 ml of 20% Intralipid, 500 ml of 50% glucose and 1,000 ml of Vamin administered through a central vein. His lipid levels still remained moderately elevated with the serum appearance, however, becoming less turbid. Clinically, on day 4 he had become confused and on examination was generally hypotonic. On day 9 he developed recurrent grand mal seizures and his clinical condition deteriorated. He remained drowsy but responsive to verbal command, he suffered a fatal cardiac arrest on day 10.

Similar changes were reported in a renal transplant patient on intravenous methylprednisolone and intravenous cyclosporin, when on administration of propofol 10 mg/ml dissolved in Intralipid (fractionated soybean oil 100 g, egg-yolk phospholipids 12 g, glycerol 22.5 g in 1,000 ml water), the patient's serum triglyceride rose from 3.16 to 6.01 mmol/l and cholesterol from 3.63 to 5.75 mmol/l.

Both cases had similar acute changes in serum cholesterol and triglyceride upon infusion of intralipid-based solutions concurrently with cyclosporin. However, while the patient in de Klippel's report had a sudden increase in his whole blood cyclosporin concentrations (250–997 ng/ml), in our patient his whole blood cyclosporin concentrations were within the range of 300–600 ng/ml and no sudden increase was demonstrated. This difference between the two patients may be spurious as de Klippel's patient had his whole blood cyclosporin concentrations measured using a non-specific assay, while we used a specific EMIT method. It is well established that metabolites of cyclosporin interfere with the measurement of cyclosporin in the non-specific assays, and it is recommended that the parent drug whole blood assay is preferred.

Cyclosporin is lipophilic and 40% is taken up by erythrocytes and most of the rest is bound to lipoproteins with high (HDL) and low density lipoproteins (LDL), and very little binds to very low density lipoproteins (VLDL). The infusion of intralipid produces a sudden increase in VLDL, and fall in HDL and LDL, resulting in a rise in unbound cyclosporin concentrations. This unbound cyclosporin is postulated by de Klippel to have produced a cyclosporin-induced encephalopathy in his patient. Although our patient had some neurological symptoms similar to the described case, we were unable to perform a magnetic resonance imaging (MRI) scan and therefore unable to demonstrate white matter changes on MRI of leuкоencephalopathy. However, the common feature in both these cases is that they developed an acute mixed hyperlipidaemia on the administration of an intralipid solution whilst on cyclosporin.

We conclude that an interaction exists, which severely disrupted the patient's lipid metabolism. The altered neurological state observed was possibly mediated by abnormal distribution, and binding of cyclosporin and its metabolites. Until this interaction is fully understood, we believe that intralipid-containing solutions are best administered with caution in renal transplant recipients on intravenous cyclosporin.

J. Bolodeoku
G.J. Mellotte
Department of Chemical Pathology and Metabolism and South West Thames Renal Unit, St Heifer Hospital, Carshalton, Surrey, SM5 1AA, UK.
Rhabdomyolysis and renal failure following defibrillation

Sir,

The effect of electrical cardioversion on total creatine kinase (CK) elevation has been studied by many authors. The release of CK after elective cardioversion correlates with the cumulative energy delivered, indicating increased skeletal muscle damage with greater energy. A case of rhabdomyolysis and myoglobinuric renal failure following cardioversion was described by Minor et al.

A 54 year old male suffered a severe oppressive chest pain along with profuse sweating, nausea and marked skin pallor. One hour after the onset of pain he arrived at the emergency department of our hospital and almost immediately developed ventricular fibrillation. Despite electrical defibrillation and cardiopulmonary resuscitation (CPR), ventricular fibrillation kept on recurring. The patient received CPR for 65 minutes, 18 electrical countershocks of 360 J each and multiple boluses of epinephrine, bretylium, lidocaine, and sodium bicarbonate. After the 18th attempt at defibrillation, sinus rhythm ensued and an electrocardiogram revealed acute inferior wall myocardial infarction. Thrombolytic therapy was ruled out because of the prolonged CPR.

He was then transferred to the intensive care unit where mechanical ventilation was started. On arrival at the unit he was alert but confused, and agitated. His blood pressure was 80/50 mmHg, pulse rate 110 beats minute, the lungs were clear and cardiac auscultation did not reveal any S3 gallop, murmurs or pericardial rubs. The urine was dark and qualitative urine myoglobin detection was positive. The level of serum CK peaked 42 hours after onset of chest pain. Serum CK was 64,480 IU/l.

For the first 3 days he had oligo-anuria with progressive worsening of his renal failure. Fifty-four hours after the onset of the myocardial infarction, serum creatinine was 781 μmol/l and serum potassium 6.3 mmol/l.

The patient required peritoneal dialysis for 2 consecutive days. He was extubated on the 11th day. He recovered his normal mental status. His renal function became progressively normal.

The very marked elevation in CK with only 2–3% CK-MB release observed in our case, indicates the importance of the skeletal muscle damage produced.

Rhabdomyolysis and prolonged hypoperfusion during CPR, can be considered the cause of acute renal failure in this case, but it is difficult to say which one contributed more.

A very high serum CK level is predictive of acute renal failure in rhabdomyolysis.

F. Barrachina
J.J. Guardiola
P. Benito
Intensive Care Unit,
‘Joan XXIII’ Hospital,
University of Tarragona,
Spain.

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J. Bolodeoku and G. J. Mellotte

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