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Cefepime-induced encephalopathy in patients with renal failure

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Dear Editor,

Cefepime is a fourth-generation cephalosporin that is widely used in hospital settings.[1] Since its approval, isolated cases of encephalopathy have been reported in patients with both normal[2] and impaired kidney function.[3,4] Nonetheless, the information about the clinical manifestations and the prognosis of this adverse reaction is scarce. Therefore, we believe it is important to report seven cases of cefepime-induced encephalopathy in patients with kidney failure. These cases corresponded to 4 males and 3 females with an average age of 63 years. All of the patients had acute or chronic renal failure when cefepime was prescribed. The average value of creatinine at the beginning of treatment was 3.6mg/dl and the initial dose of cefepime was 2.75g/day; in five patients the dose was adjusted for the degree of kidney function. The average time period between beginning of treatment and symptoms was 5.4 days. The most common clinical manifestations were a decreased level of consciousness (71.4%) and myoclonus (71.4%). The EEG was pathological in the six cases where it was carried out, demonstrating a non-convulsive epileptic status in three, slowed global activity with repetitive paroxysm in two, and diffuse affectation with a predominance of triphasic waves in one. The CT scan and the spinal tap were normal in all cases. After diagnosing the encephalopathy, treatment with cefepime was discontinued. Three of the patients received dialysis. Three patients improved (42.9%), one of whom required haemodialysis. The 4 remaining patients (57.1%) died from the encephalopathy.

The use of cefepime in patients with kidney failure, even at adjusted doses, may cause serious encephalopathy, and thus its administration should be avoided or used with close monitoring. The appearance of alterations in the level of consciousness and the myoclonus should alert us to the appearance of a non-convulsive status that requires an EEG as it is the most useful diagnostic test. Haemodialysis does not seem to modify the clinical outcome.

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Ileal intussusception by carcinoid tumour in patients with chronic renal failure

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Dear Editor,

In connection with the clinical case presented in number 4, volume 26, of this same journal, we would like to report a similar case of intussusception of the terminal ileum by a carcinoid tumour in a patient with chronic kidney failure as it is an infrequent and little referenced illness in patients with chronic kidney failure.[1]

A 54 year old female patient with a history of chronic renal failure, hyperuricaemia and nephrolithiasis. She attended Accident and Emergency with generalized abdominal pain, nausea, vomiting and diarrhoea of 48 hour duration.

She had distended and tympanic abdomen with diffuse pain and no signs of peritonism.

Air-fluid levels could be seen in the small intestine on plain abdominal x-ray. The CT of the abdomen showed a dilated jejunum-ileum with thickening of the terminal ileum and caecum wall and a 4cm mass.

With the impression of an acute intestinal obstruction, the patient underwent urgent laparotomy. The small intestine was dilated to the terminal ileum where a tumour measuring 5cm was found that had caused the intussusception of the small intestine and the obstruction. A right colectomy and an ileum-colonic anastomosis were performed.

The postoperative evolution was satisfactory. A carcinoid tumour of the ileum measuring 1.8 x 1.5cm was

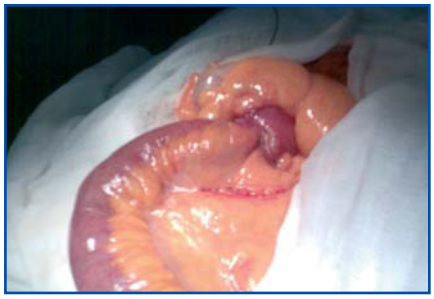

Figure 1.

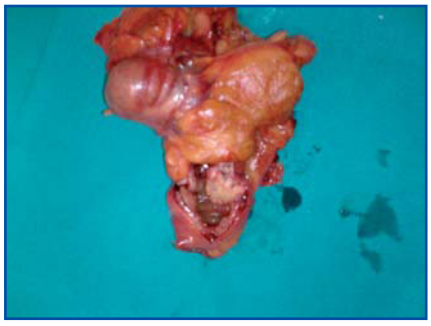

Figure 2.

infiltrating the adjacent muscle on anatomo-pathological examination; the tumour was serotonin-producing.

The most frequent location of the intestinal carcinoid tumours (ICT) is the caecal appendix (50%), followed by the ileum (25%), as in our case. The symptoms usually appear late and in a non-specific manner leading to a late diagnosis. 60% of the patients present hepatic metastases at the time of diagnosis. The carcinoid syndrome is found in only 5% of the patients and is related to the presence of hepatic metastases.[2]

In the diagnosis,[3] aside from the conventional imaging tests, the Octroscan is useful as it provides information on the localisation of tumours larger than 0.5cm and the expression or not of serotonin receptors. The differential diagnosis includes other intestinal tumours, ileo-caecal Crohn's disease and systemic mastocytosis.

Treatment should include surgical removal provided there are no metastases. Epsilon-aminocaproic Acid or somatostatin should be administered during the intervention to avoid carcinoid crisis. When there is metastatic dissemination, the patient should be managed conservatively. To reduce the metastases, 5-Fluorouracil or Adriamycin have been combined with Streptozotocin. Different drugs have been used for the treatment of the carcinoid syndrome with varied results including Chlorophenylalanine, serotonin antagonists, somatostatin, octeotride and interferon.

Although the prognosis for metastatic carcinoids is poor, survival is greater in patients with similar degrees of tumoural dissemination from other solid tumours.

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Haemodialysis management for salicylate intoxication

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Dear Editor,

Accidental overdose or suicide by salicylates provokes metabolic alterations and organ failure that can be fatal. [1,2] Haemodialysis must be used appropriately in these cases.[3,4] We present a case where haemodialysis was used in a potentially lethal overdose of Acetylsalicylic Acid (ASA).

We describe the case of a 24 year old female patient, with no morbid history, that presented in the Emergency Room 18 hours after having ingested 50 pills of ASA of 500mg, 25g total or 400mg per kilogram of weight, with suicidal intention. When hospitalized, she presented lethargy, confusion, vomiting, hypoacusia and tinnitus. Vital signs were normal and there were no positive findings in the physical examination. Laboratory tests on admission included: arterial gasometry: pH: 7.42, PO_2 115mmHg, PCO_2 14mmHg, HCO_3 9.2mmol/l. Creatininaemia: 0.89mg/dl, kalaemia: 2.1mEq/l, natraemia: 138mEq/l. The anion gap was 18. There were no alterations in liver function tests or in cardiac enzymes. A gastric lavage was performed, saline solution and sodium bicarbonate were administered. She was transferred to the Intermediate Care Unit with a salycilate level of 682mg/l.

Haemodialysis was carried out for four hours, with a polysulphone filter, and 39mEq/l bicarbonate, 3.5mEq/l of potassium and 140mEq/l of sodium bath. Fluid balance during haemodialysis was positive at 2,500cc. The patient was stable with progressive improvement of state of consciousness and slow correction of acid-base alterations. Salycilate levels of 99 and 1mg/l were obtained 12 to 20 hours after dialysis, respectively.

The ASA is absorbed in the stomach and small intestine as salicylic acid. It is conjugated in the liver and excreted in bile and urine. Therapeutic levels are of 100 to 300mg/l. The classic triad of intoxication by salicylate is hyperventilation, gastric irritation and tinnitus.[1] There may be liver, kidney, central nervous system and cardiovascular organ damage. Salicylate, above therapeutic levels, stimulates the respiratory centre and causes respiratory alkalosis.[5]

The toxic levels also produce a separation in the oxidative phosphorylation and accumulation of lactic acid, resulting in metabolic acidosis.[6] This mixed acid-base syndrome, with an increased anion gap, should lead one to suspect an intoxication by ASA in a patient where the antecedents of what has been ingested is unknown. The treatment of salycilate poisoning includes vital support, gastric lavage, the use of activated charcoal and alcalinization of the urine to encourage the excretion of the drug.[3] Salicylate has a distribution volume