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Letters

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H1N1 infection and acute kidney injury in the critically ill

Sir,

Acute renal failure due to viral infections rarely occurs. We assessed the development of acute kidney injury in critically compromised patients due to H1N1 influenza virus. All patients with PCR-confirmed diagnosis of H1N1 infection between May and July 2009 were retrospectively studied. Thereafter, the risk factors associated with the development of acute renal injury, the requirements of acute haemodialysis and death were analysed. Twenty-two subjects with H1N1 pneumonia were included: age: 52.91 ± 18.89 years; gender: males 11 (50%); chronic airway disease: 9 (41%); oncohaematological disease: 8 (36.7%); cardiovascular disease: 5 (22.7%); chronic renal insufficiency: 4 (18.2%); obesity: 3 (13.6%); concomitant pregnancy: 2 (9.1%); diabetes mellitus: 2 (9.1%); previous influenza A vaccination: 9 (41%). All patients received oseltamivir within 48 h of presumed diagnosis. Seventeen patients (77.3%) developed initial fever. Six patients (27.3%) required non-invasive ventilation assistance and 15 (68.2%) received invasive ventilatory support. The mean days on mechanical respiratory assistance were 11 ± 10.35. The arterial partial pressure of oxygen/fraction of inspired oxygen ratio was 140.11 ± 83.03 mmHg. Inotropic drugs were administered to 15 patients (68.2%). Fourteen patients (63.6%) developed acute kidney injury. The mean highest creatinine levels were 2.74 ± 2.83 mg/dL. Four patients (18.2%) needed renal replacement therapy with a mean duration of 15 ± 12 days. Six patients (42.9%) recovered renal function. Significant differences between patients with and without acute kidney injury included, respectively, pregnancy, 2 versus 0, $P < 0.05$; non-haematological immunosuppression, 6 versus 0, $P < 0.05$; APACHE score, 26.64 ± 2.51 versus 14.2 ± 1.63, $P < 0.01$; SOFA score, 9.21 ± 1.01 versus 4 ± 0.94, $P < 0.01$; MURRAY score, 0.55 ± 0.34 versus 1.34 ± 2.46, $P < 0.05$; mechanical respiratory assistance, 12 versus 2, $P < 0.05$; days on mechanical ventilation, 8.5 versus 25.66, $P < 0.05$; use of inotropic drugs, 12 versus 3, $P < 0.05$; and lower platelet levels, 91828 ± 18446 versus 149 250 ± 24 181, $P < 0.05$. Haemodialysis requirements were associated with elevated SOFA scores (12.25 ± 1.75 versus 6.22 ± 0.8, $P < 0.05$), elevated creatine phosphokinase (933 ± 436.6 versus 189.9 ± 79.3 U/L, $P < 0.05$) and alanine transferase levels (843.3 ± 778.8 versus 85.33 ± 17.4 U/L, $P < 0.05$). Twelve patients died (54.6%), 10 of whom had acute renal failure (83.3%) and 3 had been on acute haemodialysis (25%). Mortality was associated with higher APACHE, SOFA and Murray scores, a higher oseltamivir dose (253.1 ± 25.8 versus 183.8 ± 27.6 mg/day, $P < 0.05$), lower oxygen inspired fraction/alveolar pressure ratio (99.3 ± 12.2 versus 196.3 ± 33.9 mmHg, $P < 0.01$), thrombocytopaenia (88 966 ± 22 977 versus 141 200 ± 17 282 mm^3, $P < 0.05$), hypoalbuminaemia (1.82 ± 0.1 versus 2.61 ± 0.2 g/dL, $P < 0.01$), acute renal failure (10 versus 4, $P < 0.05$), oligoanuria (5 versus 0, $P < 0.05$) and lack of recovery of renal function (2 versus 4, $P < 0.01$). Three out of four (75%) haemodialysed patients died. In summary**,** in the critically ill due to H1N1 pneumonia, renal insufficiency was a frequent complication, demanding renal replacement therapy in 18% of cases. The necessity of haemodialysis was associated with an elevated risk of death. Mortality was mainly associated with multiple organ failure, oligoanuria, acute renal injury and a lack of recovery of renal function. Rhabdomyolysis may play a role in renal dysfunction, regardless of CK levels [1–4].

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Occult hepatitis B virus infection in a Sicilian chronic dialysis population

Sir,

Occult HBV infection with undetectable HBsAg is a risk factor for hepatic disease development. Among the general population, HBV circulation is a public health concern [1]. The DOPPS II study showed a considerable prevalence of HBV infection in dialysis populations [2]. In an Italian subset of DOPPS II, the prevalence was 6.3% [3].

Today, the increased life expectancy of chronic dialysis patients allows HBV to manifest all biological effects it can produce: asymptomatic antigenaemia, cirrhosis, hepatocellular carcinoma and viral reactivation post-renal transplant immunosuppressive therapies. Recently, some studies reported occult HBV infection in haemodialysis patients [4,5]. We would like to communicate our experience about HBV presence among dialysis patients.

From January to May 2005, we screened 101 chronic dialysis patients who were consecutively admitted to the Civic Hospital Palermo, Italy. We did not include individuals with a history of alcohol abuse, illicit drug use, HIV infections or malignancy. Among the subjects examined, 1 was of Black African origin and 1 of Asian ethnicity; 99 were from Sicily; none had previous renal transplant or immunosuppressive therapy; 58 were males and 43 females with a mean age of 51 years. The major causes of IRC were diabetes mellitus and hypertension; 75 patients underwent haemodialysis and 26 peritoneal dialysis. The mean time on dialysis was 28 months. We identified four HBsAg-positive and DNA-HBV-positive patients (from 6–170 000 UI/ml). We found 97 HBsAg-negative patients: among these subjects, 22 were vaccinated for HBV infection, 29 were non-vaccinated and 46 had natural HBV infection; 79 were HCV negative and 18 HCV positive; in these patients, hepatic function tests always showed normal values. There were no echographic signs of liver disease. Among the HBV-negative patients, the DNA-HBV detection was always negative. In our experience, no occult HBV infection was found in the 97 HBsAg-negative patients. Nowadays, we would not recommend DNA-HBV screening as a routine virological control in dialysis services. Instead, we suggest DNA-HBV screening for patients on the kidney transplant waiting list and in patients with abnormal liver function tests.

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Thalidomide-induced heart block in a dialysis patient

Sir,

We report the case of a patient with thalidomide-induced third-degree heart block when used in context of myeloma and acute renal failure. An 88-year-old female patient was commenced on thalidomide 50 mg daily along with pulsed dexamethasone for treatment of newly diagnosed multiple myeloma. After 3 weeks of treatment, the dose of thalidomide was increased to 100 mg daily. Electrocardiogram (ECG) at baseline presentation and on 50 mg od showed normal sinus rhythm with a ventricular rate of 83/min. There was no evidence of delayed atrioventricular conduction (PR interval 130 ms) and normal QRS. After 3 days of 100 mg thalidomide, the patient started feeling light-headed on minimal exertion. A repeat ECG showed third-degree heart block with a ventricular rate of 31 beats per minute and left bundle block.

The patient had no history of ischaemic heart disease. Serum electrolytes, thyroid function tests, cardiac enzymes and chest x-ray were within normal limits. We observed this patient for 24 h, and the ECG abnormality did not improve. After counselling about the need for ongoing treatment with thalidomide treatment for her myeloma and the risks of infections with a permanent pacemaker, she went on to have a permanent pacemaker fitted.

Due to its anti-angiogenesis activity thalidomide has been used for the treatment of multiple myeloma. The combination of thalidomide and dexamethasone, often in combination with cylcophosphamide, is now one of the most common regimens for patients with newly diagnosed multiple myeloma [1].

As thalidomide predominately undergoes pH-dependent spontaneous hydrolysis in all body fluids into multiple metabolites and is passively excreted, its pharmacokinetics are not expected to change in patients with impaired liver or kidney function. Hence, no dose reduction is recommended for patients with renal impairment or those on dialysis.

Thalidomide has a number of well-recognized side effects such as teratogenicity, skin rash, peripheral neuropathy, pneumonitis and venous thromboembolism (VTE). However, the incidence of heart block has been rarely reported. There have been similar case reports of conduction abnormalities with thalidomide [2].

We conclude that cardiac monitoring should be instituted and screened for when treating patients with this drug. Regular ECGs should be performed when rate disturbance is noted on this therapy.

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