studies up to 40% of renal transplant recipients. Such adverse events can extend along the entire GI tract, and can vary in severity from those which are mild (nausea, discomfort, appetite loss) and do not require altering immunosuppressive regimen to those which are more severe or even life threatening (severe diarrhea, GI tract ulcerations, hemorrhage and perforations).

The etiology of GI disorders following transplantation is not well understood. Because of enterocyte dependency for de novo purine synthesis MMF exposure could thus restrict the ability of intestinal epithelial cells to maintain normal barrier function, or decrease their capacity to recover from damage.7

Our patient has experienced a life threatening, severe lower GI bleeding which reoccurred within 2 days upon initial stabilization while on a stable immunosuppressive regimen. Upon dose reduction, the bleeding had stopped, indicating the possible adverse effect of MMF.

A database from the United States Food and Drug Administration’s (US FDA) Adverse Event Reporting System (AERS), containing more than 4,000,000 adverse events reported between 2004 and 2011, has a record of 9 cases of haematochezia (0.02%) associated with MMF treatment (www.drugcite.com; accessed Feb 1, 2012).

We have reported this case to the Croatian National Drug Agency and in feed-back letter have been informed that it is a serious, unexpected adverse drug reaction, possibly associated with MMF treatment. A total of 16 cases have been reported to the WHO Adverse Drug Reaction Monitoring Center with two fatal outcomes (WHO, UMC VigiBase, 29th November 2011).

Clinicians should be aware of possible, rare, but life threatening, lower GI bleeding associated with MMF treatment in renal transplant patients. Special caution should be given to patients with digestive system disease even if asymptomatic.

Conflict of interest

The authors declare that there is no conflict of interest associated with this manuscript.


A long-term follow-up of an Imerslund-Grasbeck syndrome patient with proteinuria

Dear Editor,

Imerslund-Grasbeck syndrome (IGS) is a rare autosomal recessive disorder characterized by megaloblastic anemia due to selective vitamin B12 malabsorption and asymptomatic proteinuria.1 IGS occurs in the first 1-2 years of the life and megaloblastic anemia is responsive to parenteral vitamin B12 treatment.2 It is thought that proteinuria is benign in IGS; however, there is no sufficient number of follow-up series in IGS.

Case report

A 22-year-old woman had been referred to our pediatric outpatient clinic with the complaints of pale skin, loss of appetite, ataxia and diarrhoea-constipation periods when she was 2-year-old. The clinical examination and laboratory studies revealed pallor of conjunctiva, megaloblastic anemia with vitamin B12 deficiency (serum vitamin B12 level <150pg/ml, hemoglobin: 6.5g/dl, MCV: 104fl and peripheral blood smear with hypersegmented neutrophils) and mild proteinuria (less than 0.5g/day) with absence of kidney function abnormality.
Two renal biopsies were performed because of persistent proteinuria, however, there was no remarkable histologically changes. She was diagnosed with IGS in the light of this clinical picture. Anemia and neurological symptoms were improved with vitamin B12 therapy in the next few weeks. Mild proteinuria remains persist with normal kidney function and she is being still followed-up with periodically for proteinuria.

IGS was firstly described in 1960 by Olga Imerslund and more than 300 cases have been published to date. In IGS, vitamin B12 is completely abolished and if untreated with parenteral therapy the disease is fatal. A recent study revealed some last genes cause IGS.

Conflict of interest
The authors declare that there is no conflict of interest associated with this manuscript.


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Reacción adversa por la administración intravenosa de hierro: ¿hipersensibilidad o efecto secundario?

Se. Director:
La reposición de hierro es necesaria en los pacientes de hemodiálisis debido a las pérdidas hemáticas crónicas que se producen con la técnica. La administración de hierro intravenoso de hierro mancofer, hierro sucrosa (Venofer*) se sitúa en el 1/100-1000 de los pacientes con la administración de diversos preparados férricos; cuadro compatible con efecto secundario. Las manifestaciones clínicas reaparecen de forma atenuada con las administraciones sucesivas de hierro, sin mayores implicaciones.

La tasa de efectos adversos relacionados con la administración de diversos preparados de hierro intravenoso (hierro dexcmalato, hierro carboximaltosa, hierro carboximida) se sitúa en torno a 30 por millón. El prurito asociado al hierro carboximida se describe de forma aislada como poco frecuente (hasta un 1/100-1000 de los pacientes);