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).6,7

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 regime. 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.


Figure 1.
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 deficiency is caused by a biallelic mutation either in cubilin or amnionless. Blood intrinsic factor receptor is a novel complex as well as cubilin and amnionless less genes cause IGS. Both proteins act as a receptor for intrinsic factor-vitamin B12 complexes as well as cubulin is an albumin binding protein important for renal tubular albumin reabsorption. Because of absence of glomerular damage in kidney biopsies progressive kidney disease is not usual. Broch et al enrolled 14 patients to a long term follow-up study and exhibited no deterioration in kidney function. Limited numbers of cases have been observed almost 50 years and renal prognosis is excellent. We aimed to announce our case with IGS who has a good renal prognosis over 20 years follow-up.

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