Haemolysis, Acute Renal Failure and Haemolytic-Uraemic Syndrome

P.		Stratta
C.		Canavese
L.		Colla
A.		Vercellone

Department of Nephrology, University of Turin, Italy

Dr. Piero Stratta, Strada del Macallé 4, I-10131 Torino (Italy)

Dear Sir,

Having read the letter by Mijares et al. [1], we agree with the conclusion that acute renal failure is due to tubulo-necrosis during haemolysis.

Recently a case of acute renal failure in association with haemolysis after sniffing organic solvent was reported [2]. The authors stated that this was a case of relapsing haemolytic uraemic syndrome (HUS), even though they described the absence of schistocytes in the blood smear and acute tubular necrosis in the renal biopsy picture.

We have also observed a similar case: a 16-year-old boy with haemolytic anaemia, which was not microan-giopathic, disseminated intravascular coagulation and acute renal failure after organic solvent (Trielina) sniffing. The clinical picture developed with the same characteristics as acute tubular necrosis, and cleared up satisfactorily within a week (without the necessity of dialytic treatment), as did the haematological pattern.

We, therefore, do not agree with the definition of HUS in such cases inasmuch as, by using this term in such an unspecific and generalised way, one runs the risk of removing all the specific diagnostic significance attached to it.

It is well known that acute renal failure may occur during intravascular haemolysis due to various factors: mismatched transfusion [3]; G6PD deficiency [4], and paroxysmal haemoglobinuria [1]. These episodes are obviously accompanied by haematological signs of intravascular haemolysis (haptoglobin consumption, LDH and indirect bilirubin increase, reticulocytosis), and may also cause more or less direct signs of activating the coagulation pathway (uncommon in HUS) due to the release of red cell thromboplastinic factors. These are well-defined nosographic entities from a nephrologic stand point, which usually develop favourably and also have clinico-histologic characteristics of acute tubular necrosis. They are not usually combined with hypertension, nor do typical red cell deformation (schistocytes and burr cells) and the characteristic histopathologic pattern (thrombotic microangiopathy) appear. In our opinion, both the case reported [2] and ours fall under this category.

At the risk of being repetitive, we must stress that since HUS is as yet difficult to define, the use of this term in such a generalised way may create further confusion. We conclude that if we call any acute renal failure in association with haemolysis HUS, then we must find a more specific term for real HUS.

References

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