Thrombotic thrombocytopenic purpura secondary to ABO group incompatible blood transfusion

Sir,

The recent report on thrombotic thrombocytopenic purpura (TTP) after transfusion of ABO incompatible blood is very interesting.[1] Solak et al. hypothesized that the TTP might be due to “transfusion of ABO incompatible blood” or “open cardiac surgery.”[1] Solak et al. also noted that “TTP secondary to ABO incompatible blood transfusion has never been reported in the literature until date.”[1] What the exact mechanism in the present case is still a myth and the data on confounding morbidity (such as occult cancer) in the present case is not well presented. In fact, it is no doubt that ABO incompatibility can induce TTP. The previous reports on TTP after ABO mismatched liver and renal transplantations are good example.[2,3] Hence, the cases of ABO blood transfusion should be no doubt for the pathogenesis of TTP.

Beuy Joob,
Sanitation Medical Academic Center, Bangkok, Bangkok-Thailand

Correspondence:
Dr. Beuy Joob, Sanitation Medical Academic Center, Bangkok, Bangkok-Thailand
E-mail: beuyjoob@hotmail.com

References
Sir,

The article by George et al. is indeed interesting.[1] However, a few aspects of their report require contemplation. The use of methylene blue continuous infusion in the management of methemoglobinemia due to insecticide poisoning is not congruent with current evidence. Methylene blue is a cationic thiazine dye and acts as an oxidant. It has assumed a significant role, with diverse applications in clinical practice, but is fraught with risks of side-effects.[2]

Methylene blue is reduced to leukomethylene blue by erythrocyte methemoglobin reductase in the presence of nicotinamide adenine dinucleotide phosphate (NADPH). Further, leukomethylene blue reduces methemoglobin to oxyhemoglobin. Therefore, large doses of methylene blue may result in higher levels of methylene blue rather than the expected leukomethylene blue, which could potentially induce acute hemolytic anemia, independent of preexisting methemoglobinemia.[3] This is strengthened by reports of the paradoxical induction of methemoglobinemia by methylene blue.

The possible mechanisms resulting in rebound methemoglobinemia include continued absorption of the inciting drug as well as prolonged half-life in the setting of renal or hepatic dysfunction.[4] For example, the hydroxylamine metabolites of dapsone responsible for the formation of methemoglobin have a half-life of over 30 h and may linger in circulation for up to 35 days. These agents are metabolized to reactive metabolites that oxidize 75

References