Hypoprothrombinemia and Cephalosporins in Uremics

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Dear Sir,

The pathophysiology of coagulation in uremia is complex [1]. A special and probably underestimated problem is the interference of antibiotics with coagulative factors. Many authors observed bleeding episodes in patients with impaired renal function who had been given high doses of 3rd-generation cephalosporins, i.e., moxalactam, cefamandole and cefoperazone: the cause is hypoprothrombinemia and depression of other vitamin-K-dependent factors (factor VII, IX, X and protein C) [2]. Several lines of evidence point to an interaction between cephalosporins with an N-methyl-thiotetrazole side chain and hepatic vitamin K metabolism [3]. The reason why uremics are prone to this side effect is unknown, probably they are vitamin K depleted.

Here we refer to 2 cases of interference of cephalosporins of the first and 3rd generation with vitamin K factors in 2 hemodialytic patients. The first patient, 55, was transferred to our unit from the surgical department after an endoscopic papillotomy for common bile duct stones. She had a prothrombin time of more than 3 times INR which rose to normal after withdrawal of cefamandole.

The 2nd patient, 56, came from the cardio-surgery department after replacement of the mitral valve. His prothrombin time was more than 3 times INR after 10 days from withdrawal of dicumarol. Prothrombin time promptly rose after cephaloxin was stopped.

Cephalosporins only, which carry N-methyl-thiotetrazole, are claimed for hypoprothrombinemia; here we report a case of persistent low prothrombin time with the use of cephalosporin of the first generation with a thiodiazolyl side chain.

Our statement is that probably the problem is underestimated and may be cleared by a more extensive prothrombin time testing in uremics treated with cephalosporins.

References


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