Case report

Acute thrombocytopenia after De-Nol

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SUMMARY We describe a case of severe acute thrombocytopenia in a 72 year old man treated with De-Nol for erosive biliary gastritis.

A 72 year old man is reported with a two day history of painful haemorrhagic mouth ulcers and recurrent epistaxes. In 1968 vagotomy and pyloroplasty had been performed for duodenal ulceration, but he continued to suffer intermittent dyspepsia, treated with antacids and on one occasion a short course of De-Nol, eight years previously. De-Nol is a bismuth chelate (tripotassium dicitrato bismuthate) used in the treatment of peptic ulcers. It works by binding to mucus and increasing the resistance to back diffusion of hydrogen ions, and is also thought to stimulate production of protective prostaglandins.^{1,2} Adverse reactions to De-Nol are infrequent and usually mild.

Case report

Two weeks before admission after an exacerbation of peptic symptoms, a gastroscopy showed extensive erosive biliary gastritis, with fresh blood loss. A full blood count at that time was normal: Hb 14.0 g/dl; WBC $7.5 \times 10^9/l$; and platelets $350 \times 10^9/l$. De-Nol (two tablets tid) was prescribed. He was taking no other medications.

On admission he was afebrile and had evidence of widespread bleeding: oral haemorrhagic vesicles; widespread petechiae; epistaxes; melaena and fresh blood per rectum; and microscopic haematuria. A full blood count revealed severe thrombocytopenia: the platelet count was $<10 \times 10^9/l$. The remainder of his FBC was normal (Hb 14.3 g/dl, WBC $9.0 \times 10^9/l$). Morphology of the blood film, full coagulation screen, and biochemical profile (electrolytes, LFTs) were also normal. Acute and convalescent viral titres

were not raised. Bone marrow showed hypoplasia with absent megakaryocytes, although morphology of other cell lines was normal and no infiltrations were seen. The De-Nol was stopped and he received oral prednisolone (5 mg tid) and daily platelet transfusions. Four days after admission the bleeding stopped and no new petechiae developed. On the 10th day the platelet count had risen to $62 \times 10^9/l$ and two days later to $320 \times 10^9/l$. A repeat bone marrow biopsy showed normal haemopoietic tissue with increased numbers of megakaryocytes. No further bleeding occurred and the platelet count remained within the normal range at subsequent follow up.

Discussion

It is a common misconception that De-Nol is not absorbed from the gastrointestinal tract. Bismuth has been shown to be absorbed after De-Nol therapy in minute quantities, reaching a mean blood concentrations of approximately $7 \mu g/l$ after four weeks' treatment. Although this is well below the proposed toxic threshold of $100 \mu g/l$, it is possible that the widespread erosive lesions in this patient facilitated increased absorption of bismuth. Alternatively, absorbed bismuth even in a low concentration may be enough to induce an idiosyncratic drug reaction.

Acute thrombocytopenia has not previously been reported as an adverse reaction after De-Nol treatment.³ The temporal relationship of drug administration, the severity of symptoms, and the rapid recovery of the bone marrow after withdrawal of the De-Nol in this patient suggests an idiosyncratic drug reaction.

This reaction has been reported to the manufacturers and to the CSSM.

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