Q: What are the current options for treatment of angiodysplasia of the gastrointestinal tract?

A: Angiodysplasia of the gastrointestinal (GI) tract is a vascular malformation that results from dilated veins and capillaries. These defects result in an increased risk of GI bleeding and may be responsible for up to 6% of all cases of lower GI hemorrhage. Abnormal growth alters vascular thickness and location and contributes to the onset of bleeding. Thrombocytopenia, coagulopathies, and compression (compartment) syndrome can also be consequences of these vascular malformations. Most patients with angiodysplasia are over the age of 60. The condition may be treated with endoscopic therapy, surgery, or pharmacological intervention. Endoscopic therapy (argon plasma coagulation, photoablation, etc.) and surgery often provide only temporary response as it is frequently difficult to locate all areas of hemorrhage. In addition, the lesions may reappear after employment of these procedures. Somatostatin analogs such as octreotide inhibit angiogenesis, decrease splanchnic blood flow, increase vascular resistance, and improve platelet aggregation. The drug has not only been effective in decreasing the recurrence of bleeding in the majority of patients with angiodysplasia, but also in reducing the need for administration of blood products. Most patients studied had acquired angiodysplasia while those with hereditary hemorrhagic telangiectasias (HHT) were excluded from the trials. Further study is necessary to identify the exact role of octreotide in this disorder. The effectiveness of hormonal therapy to reduce bleeding recurrence in angiodysplasia has long been debated. In 2001, a multicenter, randomized, placebo controlled trial showed no difference in bleeding recurrence when estrogen-progestin therapy was compared to placebo. The risk of angiodysplasia and GI bleeding appear to be based on one of two genetic mutations that result in increased expression of vascular endothelial growth factor (VEGF) and subsequent formation of abnormal vasculature. Newer therapies such as bevacizumab (Avastin) directly or indirectly suppress VEGF and interfere with angiogenesis. Significant adverse effects including additional bleeding complications appear to limit the widespread use of this agent. Thalidomide also possesses anti-angiogenic properties and has been used with some success in patients with angiodysplasia. It does not appear to increase the risk of bleeding, but its teratogenic potential and neurotoxic effects are well documented. In conclusion, the drug therapy or procedure chosen to control GI bleeding due to angiodysplasia should be based on the severity of the lesion(s) and the patient’s response to previous therapy.

References:

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