To the Editor:

We describe a 54-year-old woman with systemic sclerosis (scleroderma, SSc) of the limited subtype. Diagnosis in 1994 was based on tightness of the skin distal to the metacarpophalangeal joints, calcinosis, telangiectasia, esophageal dysmotility, and severe Raynaud’s phenomenon (RP) complicated by recurrent digital ulcers, gangrene and autoamputation. Her serology is significant for positive antinuclear antibody, and anticentromere antibody.

During her disease course, she received different regimens of vasodilators including nifedipine, losartan, and topical nitroglycerin in addition to aspirin, with inadequate response. In May 2010 she presented with a refractory ulcer in the right third digit. Despite increase in her vasodilator therapy and addition of pentoxifylline, it progressed to diffuse ulceration of the digit, extending proximal to the distal interphalangeal joint. Magnetic resonance imaging revealed no evidence of osteomyelitis. It was decided to admit her to hospital for intravenous (IV) prostaglandin.

After she received a 3-day course of continuous IV alprostadil, the ulcer size and pain improved significantly. On Day 3 of the alprostadil infusion, she developed coffee-ground emesis and a diminished hemoglobin, from 127 to 96 g/l. Endoscopy revealed severe esophagitis, esophageal ulceration, and appearance of the stomach consistent with gastric antral vascular ectasia (GAVE). A previous endoscopy in February 2008 at the same institution had not shown any of these findings. She was treated with transfusion of 2 units of packed red blood cells, intravenous pantoprazole, and discontinuation of the alprostadil. The bleeding stopped and her hemoglobin stabilized at a level of 120 g/l. She was discharged in stable condition.

SSc is an autoimmune disease characterized by progressive fibrosis of the skin and internal organs associated with vascular dysfunction. RP is one of the cardinal features of SSc, which can be disabling and difficult to treat. IV prostaglandins have been shown to be effective in treating resistant digital ulcers related to RP1. The precise mechanism by which clinical benefit is produced is unclear. Prostaglandins produce inhibition of platelet aggre-
fibrinolysis. Identification of high-risk patients with chronic anemia or previous GI bleeding, close monitoring for occult blood loss, and early intervention is strongly recommended prior to use of these agents.

MOHAMMED A. OMAIR, MD; SINDHU R. JOHNSON, MD, Division of Rheumatology, Department of Medicine, Mount Sinai Hospital, University Health Network, and University of Toronto, Toronto, Ontario, Canada. Address correspondence to Dr. S. Johnson, Division of Rheumatology, Ground Floor, East Wing, Toronto Western Hospital, 399 Bathurst Street, Toronto, Ontario M5T 2S8. E-mail: Sindhu.Johnson@uhn.on.ca.

Dr. Johnson has been awarded a Canadian Institutes of Health Research Clinician Scientist Award and is supported by the Norton-Evans Fund for Scleroderma Research.

ACKNOWLEDGMENT
The authors thank Dr. Peter Lee for providing the photograph of GA VE.

REFERENCES

J Rheumatol 2011;38:4; doi:10.3899/jrheum101173