A murmur and a bleed: the Heyde syndrome
Smita I Negi, Risheng Xu, Aashish Anand

Clinical presentation—A 75-year-old lady with long-standing severe aortic stenosis (AS) (Figure 1), coronary heart disease status post bypass grafting, history of stroke, and transient ischaemic attacks, presented with acute severe gastrointestinal bleed.

Figure 1. Calcific severe aortic stenosis on transthoracic echocardiogram

She underwent a colonoscopy and an oesophageoduodenoscopy and both showed no active source of bleeding. A capsule endoscopic examination of the jejunum revealed multiple angiodysplasias (Figure 2).

Figure 2. Images of angiodysplasia of the jejunum obtained on capsule endoscopy
These angiodysplasias were cauterized with good results. Upon calculation of her risk for open valvular replacement, she was deemed a candidate for percutaneous transcatheater aortic valve replacement.

Once stable, she underwent successful transcatheater implantation of an artificial aortic valve (Figure 3).

**Figure 3. Transcatheater aortic valve replacement seen on fluoroscopic imaging**

**Discussion**—Heyde syndrome refers to a triad of calcific aortic stenosis, acquired coagulopathy, von Willebrand syndrome type 2A, and iron deficiency anaemia of blood loss from intestinal angiodysplasia.

Angiodysplasia can occur anywhere in the gastrointestinal tract, but are most commonly seen in the ascending colon and the jejunum. The incidence increases with age.

It has been postulated that a low-grade chronic hypoxia drives a reflex sympathetic vasodilatation and smooth muscle relaxation, which ultimately leads to vessel ectasia and angiodysplastic changes in the vessel wall.

In addition, aortic stenosis has been shown to cause an acquired form of von Willebrand syndrome type 2A occurring from the degradation of von Willebrand factor multimers by the shear stress across the narrowed valve, leading to loss of large
multimers involved in haemostasis and bleeding in previously latent intestinal angiodysplasias.

Valve replacement has been shown to improve the haematological abnormality and a reduction in bleeding.

**Author information:** Smita I Negi, Clinical Fellow, Division of Cardiovascular Medicine, Department of Internal Medicine, University of Texas at Houston, Houston, TX, United States; Risheng Xu, Resident, Department of Internal Medicine, University of Texas at Houston, TX, United States; Aashish Anand, Resident, Department of Neurosciences, Baylor College of Medicine, Houston, TX, United States

**Correspondence:** Aashish Anand, MD, Department of Neurosciences, Baylor College of Medicine, Houston, TX 77030, United States. Fax: +1 713 7999410; email: aanand@bcm.edu

**References:**