Aortic stenosis and angiodysplasia in the elderly: common things occur commonly?

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Abstract
An elderly lady with severe aortic stenosis presented with profuse bleeding per rectum, secondary to angiodysplasia of the small intestine. An emergency aortic valve replacement with a bioprosthesis led to the resolution of her symptoms. Although the association of aortic stenosis and angiodysplasia have been described previously, the patho-physiological mechanisms remain unclear.

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1. Introduction

Although there have been several reports [1] about the association between aortic stenosis and gastrointestinal (GI) tract angiodysplasia, there is still an ongoing debate as to whether this is a true association or just coincidental [2]. Moreover the mechanism through which these two pathologies are linked remains unclear. However, it is well recognised that aortic valve replacement in this setting will lead to the resolution of the GI blood loss in most patients. There is now emerging evidence that the link between the above two pathologies may be through von Willebrand Disease [3,4]. We present a case of severe aortic stenosis in an elderly lady with a short history of angiodysplasia of the small intestine and review the pathophysiology of the GI blood loss.

2. Case report

An 80-year old lady with a 3-year history of aortic stenosis presented with collapse. There were no prior history of chest pain, blackouts, and shortness of breath or palpitation. She was taking oral corticosteroid for poly-myalgia rheumatica. She denies any history of GI bleeding. Clinical examination was consistent with aortic stenosis. She was also noted to be tachycardic and pale. Transthoracic echocardiography confirmed severe aortic stenosis (gradient of over 100 mmHg across the valve). Routine blood test revealed a low serum hemoglobin level (7.6 g/dl). Platelet counts and clotting profile were normal. Reticulocyte count was 4.6%. The only abnormality in the haematinic studies was a low serum ferritin level (8 ng/ml). Colonoscopy and barium follow-through studies were normal. However enteroscopy revealed angiodysplasia in the gastric fundus (Fig. 1a) and the distal duodenum (Fig. 1b) which were actively bleeding. She was treated with argon beam photocoagulation. Unfortunately she continued to bleed and over a period of 3 weeks she required 18 units of blood transfusion. She ultimately underwent aortic valve replacement with a size 21 mm Carpentier-Edwards pericardial xenograft (Edwards Lifesciences, Irvine, CA, USA) and her GI bleed settled. Unfortunately because of her multiple blood transfusions prior to surgery, von Willebrand factor (vWF) levels were inconclusive.

3. Discussion

Angiodysplasia is the commonest reported cause of GI bleeding in the elderly population [5]. It is an acquired submucosal arterio-venous malformation. This develops during the ageing process due to the combination of high tension and weak supporting connective tissue (deficiency of collagen type IV) in the GI wall being worse in the caecal

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area of the colon. Additionally, the incidence of aortic stenosis (AS) also increases with age.

The first report of the association of AS and angiodysplasia was by Heyde in 1958 [1] and since then several authors have described similar cases in the elderly [6]. However, there is still ongoing controversy about the occurrence of these two clinical conditions concomitantly [2]. It is well recognised that aortic valve replacement in the setting of angiodysplasia does lead to the resolution of GI blood loss in the majority of the patients.

It has been reported that angiodysplasia is more likely to develop in patients with critical aortic stenosis as in the case described above. Several mechanisms have been postulated to account for the development of angiodysplasia in this setting. As early as 1971, Boss and Rosenbaum described distension of intestinal mucosal vessels in post-mortem studies as the cause for the GI blood loss [7]. This reflex vasodilatation is thought to be mediated via the sympathetic system in response to low-grade hypoxia. There is relaxation of the vascular smooth muscle which with chronicity causes ectasia. On the other hand, according to Boley et al., the elderly population develops intermittent intestinal submucosal venous outflow obstruction as part of the ageing process [8]. It has also been suggested that in AS there is a redistribution of the splanchnic blood flow with associated mesenteric ischaemia along with an increased in the intestinal intramural pressure [9]. Pulse wave arterial patterns in the terminal superior mesenteric artery have shown significant damping of the arterial pressure in patients with AS compared to controls [6]. The end result of all the above processes is mucosal ischaemia and hence bleeding due to the loss of gut integrity [8].

A supplemental theory involves the coagulation system. Initially it was thought to be due to platelet dysfunction due to mechanical damage as they are ejected from the heart through the abnormally tight aortic valve. However there is now emerging evidence that there may be a low-grade consumption coagulopathy involving the vWF [4], originally described by Warkentin et al. in three patients [3]. This theory has been further strengthened by the findings of Veradier et al. [10]. This acquired form of von Willebrand disease resolves within a few months following aortic valve replacement. The functional fraction of vWF levels (high-molecular-weight multimers of vWF) were significantly decreased prior to surgery and returned to normal post AVR. This polypeptide facilitates platelets adhesion and aggregation to the endothelium at vascular injury sites via an interaction between vWF and platelet membrane glycoproteins. Thus, abnormal vWF is associated with an increased incidence of bleeding.

4. Conclusion

Angiodysplasia is more likely to be associated with critical aortic stenosis. The mechanism involves a weakening of the supporting collagenous tissues due to old age in the setting of mucosal ischaemia. Bleeding is perpetuated by an acquired von Willebrand Factor abnormality. Aortic valve replacement improves the mucosal blood supply and corrects the haematological abnormality and remains the first line of treatment in this setting.

Fig. 1. (a) Endoscopic findings of the stomach showing the bleeding angiodysplasia (black arrows) and blood collection in the stomach. (b) Enteroscopy of the distal duodenum demonstrating the angiodysplastic lesion (white arrow).
References


