Review Article

Angiodysplasia: current concepts

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Angiodysplasia is a condition of unknown aetiology in which microvascular abnormalities are found in the mucosa and submucosa of the bowel wall. The lesions are found predominantly in the caecum and right side of the colon and are frequently associated with either intermittent acute or continuous chronic intestinal blood loss. There is no family history and no recognized association with vascular abnormalities of the skin or other organs. The lesions of angiodysplasia which are small (less than 5 mm) and usually multiple are diagnosed by selective visceral angiography and/or colonoscopy but cannot be detected on barium enema or by the naked eye at laparotomy. Localization by the histopathologist is greatly facilitated by special injection techniques demonstrating the blood vessels of resected colonic specimens prior to fixation and section. The lesions are thought to represent a distinct benign pathological entity characterized in their early stage by dilated tortuous submucosal veins. In the more advanced stages there is further dilatation of the submucosal veins and venules and capillaries. These characteristic features of right colonic vascular ectasias correspond to the definition given by Gentry et al. of telangectasias, i.e. dilatation of pre-existing vascular structures.

Galdabini in 1974 first used the term 'angiodysplasia' to describe the pathological abnormality found in a patient who presented with what is now recognized as a characteristic history and the angiographic features of the condition defined above.

There are two main clinical problems related to angiodysplasia; firstly, the fact that true prevalence of the condition in the general population is unknown and secondly the difficulty in detecting the disorder in patients with gastrointestinal bleeding of uncertain origin and, having made the diagnosis of angiodysplasia, establishing that it is the cause of the blood loss.

How common?

It has been stated that angiodysplasia may represent the commonest single cause of obscure gastrointestinal bleeding in the elderly population. Indeed a number of series have established that in patients undergoing visceral angiography in the investigation of obscure gastrointestinal bleeding the most commonly detected abnormality is angiodysplasia. In our own published series, angiodysplasia was diagnosed in 40% of patients examined. It must be remembered however that patients undergoing angiography represent a highly selected group since the cause of gastrointestinal haemorrhage will be identified by routine investigations in between 80% and 95% of cases. Angiodysplasia without doubt represents the commonest cause of bleeding in the residual 5 to 20% of patients. Extrapolating from these figures it can be seen that angiodysplasia accounts for between 2% and 8% of all patients presenting with gastrointestinal bleeding. This estimate is supported by the work of Richter et al. who diagnosed angiodysplasia colonoscopically in 26 patients, in a period when 1044 patients were colonoscoped for bleeding, a prevalence of 2.6%. The same group of workers detected the lesion in 13 patients out of a group of 1400 examined for reasons other than bleeding, a prevalence of 1.4%. Three principal in vitro studies have been performed in an attempt to address the question of the incidence of angiodysplasia in the general population. In one study employing an injection/radiography technique, no lesions were detected in 39 autopsy specimens. The other two studies employed an injection and clearing technique followed by examination using trans-illumination and microscopy and both groups found lesions in up to 50% of specimens examined.

We have recently reviewed over 450 visceral
angiograms performed for reasons other than gastrointestinal bleeding. Out of 166 patients in whom the caecum and the right colon were adequately visualized the characteristic features of angiodysplasia were detected in 6 (3.6%). It is clear from the wide variation in these figures that further research is necessary to establish the true prevalence of angiodysplasia in the non-bleeding population.

**Diagnosis**

The second major clinical problem relating to angiodysplasia is its recognition and this affects three groups of medical practitioner: clinicians (physicians and surgeons), radiologists and pathologists. These problems are summarized in Table I.

It would seem that when both angiography and colonoscopy are available to a clinician then it is advantageous to utilize both techniques to make a positive diagnosis of angiodysplasia and exclude other pathology.

The radiologist having accepted a patient for angiography must perform a 3-vessel study of high quality (i.e. coeliac, superior mesenteric and inferior mesenteric examinations). The characteristic features of angiodysplasia include vascular tufts visualized in the arterial phase, an early filling draining vein and a slowly emptying, dilated, tortuous intramural vein. In our institution we normally require at least two of these three features to be present before the diagnosis is suggested (Figure 1). Angiodysplasia can only definitely be implicated as the cause of blood loss if the lesions are seen to be actively bleeding at the time of the study. Some authorities advocate that the colon should be distended with air at the time of angiography and that direct serial magnification radiography should be used to improve the chances of detection of these lesions. We have not employed these techniques in our institution, finding that selective catheterization, the use of adequate volumes of contrast media and high quality radiography are the most important factors.

An important point to note is that the angiographic features of angiodysplasia can be mimicked by other conditions including inflammatory bowel disease, such as Crohn's disease, and malignancy and the possible existence of these alternative pathologies should not be ignored. If angiography suggests the diagnosis of angiodysplasia then other disease must be excluded by colonoscopy, barium enema or even laparotomy.

**Treatment**

Once the diagnosis of angiodysplasia has been made the clinician is faced with the question of treatment. Having excluded malignant disease, and if the blood loss is not severe, simple supportive treatment for the anaemia may be adequate. If the lesions are small in number colonoscopic fulguration is, in experienced hands, readily performed, relatively non-invasive and easily repeated. When the disease is more extensive, or other methods are failing to control the anaemia then surgical resection is the treatment of choice. Laparotomy also allows the surgeon the chance to detect or exclude the presence of other lesions which may be the cause of the bleeding. In our own

**Table I** Problems relating to the diagnosis and management of angiodysplasia

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series, over 20% of patients who underwent laparotomy were found to have a second lesion which could have been responsible for the blood loss.11

Pathological confirmation of angiodysplasia in a resected specimen may be difficult. The lesions are very small, not detectable by the naked eye and it is impossible for a pathologist to section the entire specimen. Injection of the specimen with either a barium gelatin mixture or latex material allows the lesions to be readily visualized by either direct vision (Figure 2) or transillumination. Radiography of a specimen12 which has undergone barium injection will often reveal far more extensive disease than was originally suspected in vivo (Figure 3).

Pathological examination of the lesions of angiodysplasia readily reveals why they bleed so easily. Dilated vascular channels are present in the submucosa and mucosa, and the latter are separated from the bowel lumen by only one or two cell layers.

Non-colonic angiodysplasia

Traditionally the term angiodysplasia has only been used to describe lesions found in the colon. Recently similar vascular abnormalities have been diagnosed by both endoscopy and angiography in the stomach13,14 and small intestine.15 The occurrence of the condition at these sites is less common and it is important to be certain that histologically the lesions represent the same condition and that they fulfil the criteria set out in the definition at the beginning of this article.

Aetiology

There has been a great deal of speculation as to the aetiology of angiodysplasia. The most widely quoted theory is that proposed by Boley4 which is that it represents a degenerative condition related to ageing and that repeated partial intermittent low grade obstruction of submucosal veins occurs. This
Figure 2 The dissecting microscope appearances of angiodysplasia in a resected specimen that has been injected with a barium gelatin mixture. The normal honeycomb mucosal pattern is seen to be disrupted by enlarged tortuous vessels which have filled with barium.

Figure 3 Microradiograph of an area of angiodysplasia. The cluster of abnormal vessels is clearly seen and the very large draining veins can be identified.

eventually results in dilatation of submucosal veins which in turn progresses to incompetence of the precapillary sphincters and development of small arteriovenous communications.

Other theories are based on the supposition that the caecal mucosa is subjected, by a variety of mechanisms, to chronic ischaemia which damages the mucosa and causes arteriovenous shunts to develop. The often reported association between aortic valve disease and angiodysplasia\textsuperscript{16} has been used to support the theory of an ischaemic aetiology.

None of these theories explains the occurrence of angiodysplasia in young people. In our own series we have seen the condition in teenagers\textsuperscript{12} and a number of patients under the age of 40. We have also seen the condition in association with Meckel's diverticulum\textsuperscript{17} and have postulated that the condition may have a congenital aetiology or that patients may have a congenital predisposition to its development. More recently we have undertaken a detailed analysis of the distribution of angiodysplastic lesions found following resection. In all patients lesions are clustered in the mucosa in the immediate vicinity of the ileocaecal valve and the possibility that the small bowel contents exert a chemical effect on the mucosa in this region which predisposes to the development of arteriovenous shunts has been proposed. This theory has been supported by the discovery that in three patients new angiodysplastic lesions have developed in the colonic mucosa close to the ileocolic anastomosis in patients who have previously undergone hemicolectomy for the condition.
Conclusions

In conclusion it is clear that colonic angiodysplasia represents a major cause of chronic gastrointestinal blood loss and hence morbidity, particularly in the elderly population. Although the lesions of angiodysplasia occur most commonly in the colon, similar abnormalities occur, but less frequently, in the stomach and small intestine. The most appropriate means of diagnosis is dependent on local expertise and facilities but is ideally provided by a combination of colonoscopy and angiography. The choice of treatment depends largely on the confidence with which other diseases can be excluded and the severity of blood loss. The exact aetiology of angiodysplasia remains unknown but almost certainly both congenital and acquired factors (possibly multifactorial) are implicated.

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References

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