ANTERIOR RECURRENT EPISTAXIS FROM KIESSELBACH'S AREA

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Abstract

Vascular diseases are a major threat to human health nowadays. Hypertension, cardiovascular disease and varicose vain disease including hemorrhoids, are now increasingly recognized as inflammatory diseases.

The role of inflammation cytokines in the pathogenesis of these diseases is very important. The lamina propria in the nasal mucosa is rich in blood vessels and humoral mediators.

Recurrent epistaxis from Kiesselbach's area syndrome (REKAS) was first mentioned as early as 1985. It has been found that 90% of patients suffering from recurrent epistaxis from Kiesselbach area syndrome simultaneously suffered from hemorrhoids.

Clinical observations suggest a possible mutual pathophysiologic relationship between Kiesselbach's and anorectal venous plexus. This relationship is also suggested in the reverse direction: significantly more than two thirds of primarily hemorrhoidal patients (83.01%) showed simultaneous vascular dilatations within their Kiesselbach plexuses, but none of these patients had ever have recurrent nose bleeds.

There is one more thing they did not have (contrary to REKAS group) - anterior septal deformity. Furthermore, REKAS and hemorrhoidal disease, despite being different clinical entities, frequently appear in the primarily REKAS patients or their closest relatives (more than 90% out of all!). At the same time, all of REKAS patients did have a certain degree of the anterior septal deformity, which primarily hemorrhoidal patients did not have at all.

Therefore we consider that Kiesselbach's vascular plexus in the Little's area of the nasal septum belongs to the same group as anorectal venous plexus does (others of this group are brain, esophagus, and lower leg venous system).

We also consider that the anterior septal deformity is a crucial factor for the onset of the inflammation of the nasal vestibule skin (vestibulitis nasi), while vestibulitis nasi precipitates the onset of typical recurrent nose bleeds from the Kiesselbach's plexus.

Keywords: Nasal septum, recurrent epistaxis

Introduction

Vascular diseases are a major threat to human health nowadays.

Cardiovascular diseases, including coronary heart disease and a stroke, are the leading cause of death in the United States and Europe.Several risk factors (i.e. stressful life,

overweight, physical inactivity, smoking, hypertension, and diabetes mellitus, high levels of cholesterol and lipids) are associated with the development of cardiovascular disease. Hypertension and cardiovascular disease, including atherosclerosis, cardiac hypertrophy, and ischemic disease, nowadays have been increasingly recognized as inflammatory diseases. In recent years, this hypothesis has lead to heightened interest in studying the role of inflammation cytokines in the pathogenesis of these diseases [1].

Like artery, the veins are also a part of vascular system and have their own pathology.

Varicose veins are tortuous, twisted, or lengthened veins. The theory that varicose veins result from failure of valves in the superficial veins leading to venous reflux and vein dilatation has been superseded by the hypothesis that valve incompetence follows rather than precedes a change in the vein wall [2]. Thus, the vein wall is inherently weak in varicose veins, which leads to dilatation and separation of valve cusps so that they become incompetent.

Risk factors for varicose veins include increasing age and parity and occupations that require a lot of standing.

Hemorrhoids are a very widespread disease causing pain by thrombosis, fear by bleeding and be a burden by weeping and itching. Hemorrhoids occur when the external hemorrhoidal veins become varicose (enlarged and swollen), which causes itching, burning, painful swellings at the anus, dyschezia (painful bowel movements), and bleeding. Pain with bowel movements and bleeding are often the first signs of hemorrhoids. Taweevisit et al. propose that mast cells have a multidimensional role in the pathogenesis of hemorrhoids, through the actions of the chemical mediators and cytokines released from mast cell granules [3].

Some investigation focuses on caliber and flow changes of the terminal branches of the superior rectal artery supplying the corpus cavernosum recti in patients with hemorrhoids [4]. This fact implicates the unity of the vascular system and its pathophysiology.

Some pathophysiological aspects of the nasal blood vessels

The lamina propria in the nasal mucosa is rich in blood vessels.The arterioles are conspicuous by an absence of internal elastic membrane. Porosity of the endothelial basement membrane had been described as characteristic of nasal blood vessels. As a result of these structural characteristic, the subendothelial musculature of these vessels may be influenced more easily by agents, such as mediator substances, hormones and drugs, circulating in the blood stream.

The capillaries just below the surface epithelium and those surrounding the glands are of the fenestrated type. These capillaries are well suited for rapid movement of fluid through the vascular wall [5]. This will allow water to escape into the airway lumen and to vaporize to take place in conditioning the inspired air. Large venous cavernous sinusoids, mainly localized in the inferior turbinate, are characteristic of nasal mucous membrane. They are normally found in a semi-contracted condition as a result of the influence of the sympathetic nerve-mediated smooth muscle tonicity.

The cavernous sinusoids are regarded as specialized vessels adapted to the functional demands of the nasal airway with respect to heating and humidification of inhaled air. When they distend by blood, the nasal mucosa becomes swollen and tends to block the airway lumen, either in part (which is normal) or completely (in nose disease).

The extravasation of plasma through the walls of postcapillary venules takes place during inflammation of the mucosa. The process runs through the gaps in the intercellular junctions between the endothelial cells. This leads to an increase of the interstitial liquid volume and pressure, which, in addition, tends to force transfer of plasma-like liquid as an exudate. The humoral mediators that cause extravasation of plasma are many, and include

histamine, bradykinin, various prostaglandins, and sensory nerve neuropeptides such as substance P [5].

Recurrent epistaxis from Kiesselbach's area and anorectal venous plexus: do they have anything in common?

Epistaxis, whether spontaneous or otherwise, is experienced by up to 60% of people in their lifetime, with 6% requiring medical attention [6]. The etiology of epistaxis can be divided into local and general causes, however most (80–90%) are actually of unknown etiology [7].

Kiesselbach's plexus, which lies in Kiesselbach's area, or Little's area, is a region in the anteroinferior part of the nasal septum where four arteries anastomoses to form a vascular plexus of that name.

Recurrent epistaxis from Kiesselbach's area syndrome (REKAS) was first mentioned as early as 1985 [8]. This syndrome was found to be the result of a simultaneous interaction between the following four constant factors: (a) specific anterior septal deformity; (b) dilated vessels of Kiesselbach venous plexus; (c) infection of the nasal vestibule skin, and (d) heredity.

Clinical observations suggested also a possible mutual pathophysiologic relationship between Kiesselbach and anorectal venous plexus. He also found a high correlation of REKAS nose bleeds and the incidence of lower leg venous system disorders (varices cruris, for instance) [8].

Regarding hemorrhoidal disorders, a large number of REKAS patients, i.e. 90% of them, were found to suffer from hemorrhoids [8]. Local chronic infection was suggested to be a causative factor for both hemorrhoids and REKAS patients [9,10]. Furthermore, the symptoms of hemorrhoidal diseases (and also of varices cruris, cerebral strokes) were found in the closest relatives of 90% of REKAS patients or even in themselves, strongly suggesting a hereditary predisposition for venous plexus disorders [8], as is the case with hemorrhoids [11]. Finally, dilated vessels and direct arteriovenous communications are a usual finding in both hemorrhoidal [12] and REKAS patients [13].

A pathophysiologic relationship between Kiesselbach and anorectal venous plexus in REKAS patients is suggested by the high incidence of REKAS patients or their closest relatives suffering from hemorrhoidal problems. According to our previous results [14], this relationship is also suggested in the reverse direction: more than two thirds of primarily hemorrhoidal patients showed simultaneous vascular dilatations within Kiesselbach plexus of the nasal vestibule (83.01%). Surprisingly, none of these patients had recurrent nosebleeds [14]. This suggests that vascular dilatations within Kiesselbach venous plexus are not per se an exclusive, crucial factor for the onset of the REKAS.

Thus, the question arises here about which factors exactly were missing as to produce the onset of REKAS also in our primarily hemorrhoidal patients: anterior septal deformity, or vestibular infection, or perhaps both?

Anterior septal deformity exhibited a very low incidence (7.5%), whereas the signs of a slight vestibular infection were also very seldom seen (3.8%) [14].

Since neither vestibular infection nor REKAS appeared in primarily hemorrhoidal patients despite dilated vessels in their Kiesselbach venous plexuses and a positive hereditary factor, we believe that this situation was due to absence of the anterior septal deformity in these patients at the first place. The septal deformity has been generally recognized as a provocative factor in epistaxis [7]. As we mentioned before, inflammation also play a role in the pathogenesis of arterial and venous vascular diseases [1,3].

Conclusion

Kiesselbach's vascular plexus in the Little's area of the nasal septum belongs to the same group as anorectal venous plexus does.

The anterior septal deformity is a crucial factor for the onset of the inflammation of the nasal vestibule skin (vestibulitis nasi), while vestibulitis nasi precipitates the onset of typical recurrent nose bleeds from the Kiesselbach's plexus.

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