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ORIGINAL ARTICLE

Revised morphology and hemodynamics of the anorectal vascular plexus: impact on the course of hemorrhoidal disease

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Abstract
Purpose The pathogenesis of hemorrhoidal disease is based mainly on the vascular hyperplasia theory. The aim of this study was to reassess the morphology and the functional mechanisms of the anorectal vascular plexus with regard to hemorrhoidal disease.
Materials and methods The anorectal vascular plexus was investigated in 17 anorectal and five hemorrhoidectomy specimens by means of conventional histology and immunohistochemistry. Vascular corrosion casts from two fresh rectal specimens were used for scanning electron microscopy. Transperineal color Doppler ultrasound (CDUS) with spectral wave analysis (SWA) was performed in 38 patients with hemorrhoidal disease and 20 healthy volunteers.
Results The anorectal vascular plexus was characterized by a network of submucosal vessels exhibiting multiple thickened venous vessels separated by distinct sphincter-like constrictions. CDUS and SWA showed significant flow differences in peak velocities (6.8±1.3 cm/s vs. 10.7± 1.5 cm/s; P=0.026) and acceleration velocities (51±4 ms vs. 94±11 ms; P=0.001) of afferent vessels between the control group and patients with hemorrhoidal disease.
Conclusions Coordinated filling and drainage of the anorectal vascular plexus is regulated by intrinsic vascular sphincter mechanisms. Both morphological and functional

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failure of this vascular system may contribute to the development of hemorrhoidal disease.

Keywords Hemorrhoids · Anorectal vascular plexus · Vascular sphincter · Smooth muscle

Introduction

Although still under debate, the multifactorial pathogenesis of hemorrhoidal disease is based on two path-breaking theories: the prolapse of the anal cushions and the vascular hyperplasia theories [1]. The vascular plexus within the subepithelial space of the anal transitional zone (ATZ) has been described as "corpus cavernosum recti" and claimed to provide mechanical rather than nutritional functions resembling morphological features of erectile tissue [2].

Several anatomic investigations have demonstrated the existence of arteriovenous communications between the terminal branches of the superior rectal artery (SRA) and the subepithelial venous plexus [1, 3]. This subepithelial vascular plexus is known to be a complex system of thin-walled tortuous venous structures supported by smooth muscle and fibroelastic tissue scaffolding [4]. These vascular structures surrounded by fibro-muscular tissue have been described as so-called anal glomerula corresponding to the anal cushions [2]. The discovery of this morphologic symbiosis of subepithelial smooth muscle fibers and anorectal vascular plexus in the middle of the nineteenth century [5] and confirmation by several authors [6] were essential for the functional understanding of anal continence and defecation mechanisms.

However, the development of hemorrhoidal prolapse—especially from the third decade onward when replacement of the musculature by collagen tissue takes place—remained unclear. A loss of suspensory function of the so-called musculus submucosus ani located adjacent to the hemorrhoidal plexus has been suggested. The submucous muscle layer associated with the hemorrhoidal plexus was termed musculus submucosus ani [7] and must not be confounded with the muscular wall components of the veins draining the anorectal vascular plexus described by Stelzner as "throttling veins." Up to now, scarce reliable data exist on the regulatory mechanisms of the anorectal vascular plexus, which are supposed to be decisive for the pathogenesis of hemorrhoidal disease. Therefore, the aim of this clinical–anatomic study was to reconsider the morphology of the anorectal vascular plexus especially with regard to its functional role in filling and drainage in both normal and hemorrhoidal specimens. In the following, the term "anorectal vascular plexus" (AVP) will be used.

Material and methods

Morphological studies

Material

A total of 17 anorectal specimens from the Division of Clinical and Functional Anatomy, Innsbruck Medical University (seven women, ten men; mean age, 65 years ranging from 1.5–100 years), were investigated with regard to anorectal region using conventional and immunohistochemical staining methods. The specimens showed no macroscopic abnormalities nor was there any history of previous pathology of the anorectal region. Additionally, hemorrhoidectomy specimens from five patients with hemorrhoids grade IV following conventional Milligan–Morgan hemorrhoidectomy (three men, two women; mean age, 52 years ranging from 39–77 years) were obtained from the Department of Pathology, Innsbruck Medical University, after autopsy. Transmural colonic sections from a 1-year-old male individual and penile sections from a 38-week-old fetus served as material for comparative studies of the corpus cavernosum recti and the normal submucosal vascular plexus of the colon and the vascular architecture of the penile cavernous bodies.

Histology

Specimens were fixed in 7% formaldehyde in PBS (0.2 mol). After paraffin-embedding in a routine histological infiltration processor (Miles Scientific, Naperville, USA), the specimens were cut in 4-μm sections in planes parallel or transverse to the longitudinal axis of the anorectum using a Microm ERGO Star Rotations microtome (Microm, Walldorf, Germany). The sections were dried overnight, dewaxed with xylene, and rehydrated in graded alcohol series, and every tenth to twentieth section of a series was stained either with hematoxylin and eosin or with azan.

Immunohistochemistry

Immunostaining of paraffin sections (5 μm) was performed using antibodies for labeling smooth muscle cells (α-smooth muscle actin, SMA) and peripheral nerves (S-100). SMA is a contractile myofilament protein and shows extensive interspecies and intertissue conservation. The monoclonal mouse primary antibody (clone 1A4, Linaris, Histoprime, Wertheim-Bettingen) specifically identifies the alpha isotype of SMA. It reacts with normal and neoplastic smooth muscle cells and both vascular and visceral smooth muscle cells including myofibroblasts and myoepithelial cells. Protein S-100 is expressed in neuroectodermal tissues including nerves and melanocytes, and is expressed by Schwann cells and malignant melanomas. Peripheral nerves

were readily identified with the mouse monoclonal anti-S100 antibody (4C4.9 Ventana Medical Systems SA, Strasbourg, France).

Paraffin sections were mounted on Superfrost plus™ slides (Menzel, Germany) and dried at 60°C overnight. The specimens were deparaffinized and rehydrated according to standard laboratory procedures using xylene and a graded ethanol series (Merck, Darmstadt, Germany). For antigen retrieval, the tissue was boiled for 30 min in a commercial microwave oven using Antigen Retrieval Citrate Buffer™ (Dako A/S, Copenhagen, Denmark). After primary antibody incubation for 32 min, the slides were placed in the immunostainer. Chromogenic detection was performed with the basic DAB detection kit according to the manufacturer's recommendations. The slides were counterstained with hematoxylin for 4 min and Bluing Reagent for 2 min, and dehydrated in increasing ethanol and xylene series according to standardized laboratory procedures and mounted permanently with Entellan™ (Merck, Darmstadt, Germany). All immunohistochemical procedures were performed at 37°C using a Ventana Nexes™ automated staining platform (Ventana Medical Systems, Tucson, Arizona, USA).

Scanning electron microscopy of vascular corrosion casts

Two fresh anorectal specimens (one man, one woman; 74 and 41 years) were obtained from the Department of Pathology, Aachen University, 12–48 h postmortem following forensic autopsy. SRA and inferior rectal artery (IRA) in the man or SRA only in the woman were exposed, and 60 ml (50 ml via SRA and 10 ml via IRA) or 50 ml (female) of Mercox-Cl-2B diluted 4:1 with monomeric methylmetacrylic acid (Fluka, Basel, CH) was injected using slight manual pressure. After the injected resin had hardened at room temperature, specimens were submerged in hot water for tempering (60°C; 12 h), rinsed under running tap water, and transferred to 7.5% potassium hydroxide (40°C) for removal of soft tissue. Finally, macerated specimens were rinsed in several passages of distilled water, frozen therein, and freeze-dried. Dry specimens were mounted onto specimen stubs using the conductive bridge method [8], evaporated with carbon and gold, sputtered with gold, and examined with the scanning electron microscopes ESEM XL-30 (FEI, The Netherlands) or Stereoscan 250 (Cambridge, UK) at accelerating voltages of 10 kV [9, 10].

Hemodynamic studies

Patients

A total of 38 consecutive outpatients (12 women, 26 men; mean age, 52 years; range, 27–77 years) with symptomatic hemorrhoidal disease of Goligher grades I (n=5), II (n=11), III (n=16), and IV (n=6), who were referred to the Proctological Unit at the Department of General and Transplant Surgery, were included in this study. All patients underwent clinical inspection including digital exploration and anoscopy and, prior to intervention, were additionally examined with standardized transperineal color Doppler ultrasound (CDUS) including spectral wave analysis (SWA). Patients with other anorectal diseases such as colorectal cancer, chronic inflammatory bowel disease, perianal fistula/abscesses, or perineal trauma were excluded from the study.

Twenty healthy volunteers served as age-matched control group (ten women, ten men; mean age, 47 years; range, 22–69 years). Symptomatic hemorrhoids and other anorectal disorders were excluded by physical examination. Standardized transperineal CDUS and SWA were performed by a single experienced radiologist (H.G.), who was blinded to whether the respective subject belonged to the study group or the control group and also to the respective grades of hemorrhoidal disease. Informed consent was obtained prior to each inclusion in the study. Both the morphological and functional studies were approved by the local ethics committee.

Transperineal CDUS and SWA

Transperineal CDUS and SWA were performed with a standard iU22® ultrasound device (ATL Philips®, Washington, USA) using a curved array 9–4 MHz broadband transducer adjusted with a thermic index not exceeding 0.5 and a mechanical index not exceeding 0.8. For depiction of all necessary topographic landmarks, patients were arranged in left lateral position with flexed knees and hips. The probe was attached to the perineum always sagittally so that all relevant perineal structures (i.e., perineal body, deep and superficial portion of the external anal sphincter muscle, puborectalis muscle sling, and internal anal sphincter muscle) were sufficiently assessable in a sagittal plane. The settings for the CDUS were standardized wherever possible with gain settings at about 80% and a pulse repetition frequency of up to 1,700 Hz and for arterial structures and of up to 400 Hz for venous signals. The wall filters were set between 30 and 110 Hz. The additional gain settings for corresponding SWA were automatically adjusted to 30% to 40% with corresponding wall filters between 100 and 120 Hz. The purpose of all these adjustments was to optimize the signal-to-noise ratio in order to prevent ambient noise from masking signals from vessels of interest. All relevant measurements in SWA were done while always keeping the detection volume (DV) as low as possible and exactly within the depicted feeding and draining vessel of interest. The DV was always angulated less than 60°. All necessary calculations were performed with the QLAB® program (ATL Philips®, Washington,

USA) installed on the ultrasound device during the scope of the investigations. All vessels terminating at and draining the AVP with its submucosal branches and the previously described additional branches of the SRA were depicted (CDUS) and measured (SWA) at their perforation through the rectal wall right above the levator ani muscle [11]. The following data on the respective vessel perfusion were calculated: peak systolic velocity, end diastolic velocity, resistance index (describing the relative resistance of the vascular system distal to the point of measurement), and acceleration time (time elapsed between EDV and PSV).

Statistical analysis

Data obtained from ultrasound studies were expressed as range and means with standard deviations (SD). Data (peak flow and acceleration time) in the study group and the control group were compared by means of the two-tailed unpaired Student's *t* test for continuous normal distribution. The non-parametric Mann–Whitney *U* test was applied whenever indicated. Statistical significance was defined as $P<0.05$. SPSS for Windows 11.0 software (Chicago, IL, USA) was used for all analyses.

Results

Morphological studies

Histology and immunohistochemistry

The ATZ interposed between the distal rectal mucosa and the proximal squamous non-keratinized epithelium comprising mucosa, submucosa, and parts of the internal anal sphincter muscle was analyzed in all specimens. The AVP extended throughout the submucosal layer and was composed of a densely distributed network of tortuous venous capacity vessels. Most submucosal venous vessels of the ATZ showed considerably dilated lumina and were characterized by a thickened tunica media with up to 15 layers of smooth muscle cells (Figs. 1 and 2). These findings were consistent with regard to age and could be observed even in a specimen from an 18-month-old female (Fig. 3). In contrast, mucosal vessels located above the lamina muscularis mucosae did not show this distinct muscular layering as compared to the submucosal vascular network. Both the prominent venous dilatations and the thickening of the vascular wall were confined to the ATZ. Orally to the ATZ (colonic sections), the submucosal vascular plexus exhibited regularly sized vessels with a rather thin vascular wall.

The venous vessels were arranged in a cushion-like pattern. Between the ballooned vascular segments were constricted portions located at regular intervals (Fig. 4). These vascular constrictions at both ends of the dilated segments were characterized by a thickened tunica media containing an increased amount of smooth muscle cells (Fig. 5). The concentrically arranged smooth muscle cells resembled morphological features of small vascular sphincters, suggesting that the luminal narrowing originated from active muscular contractions of the vascular wall. Moreover, between dilated venous cushions, several collateral anastomoses with abruptly changing caliber were observed in the AVP (Figs. 2 and 4).

Hemorrhoidectomy specimens showed submucosal vessels to be remarkably increased in number and surrounded by sclerotic connective tissue. The vascular plexus con-

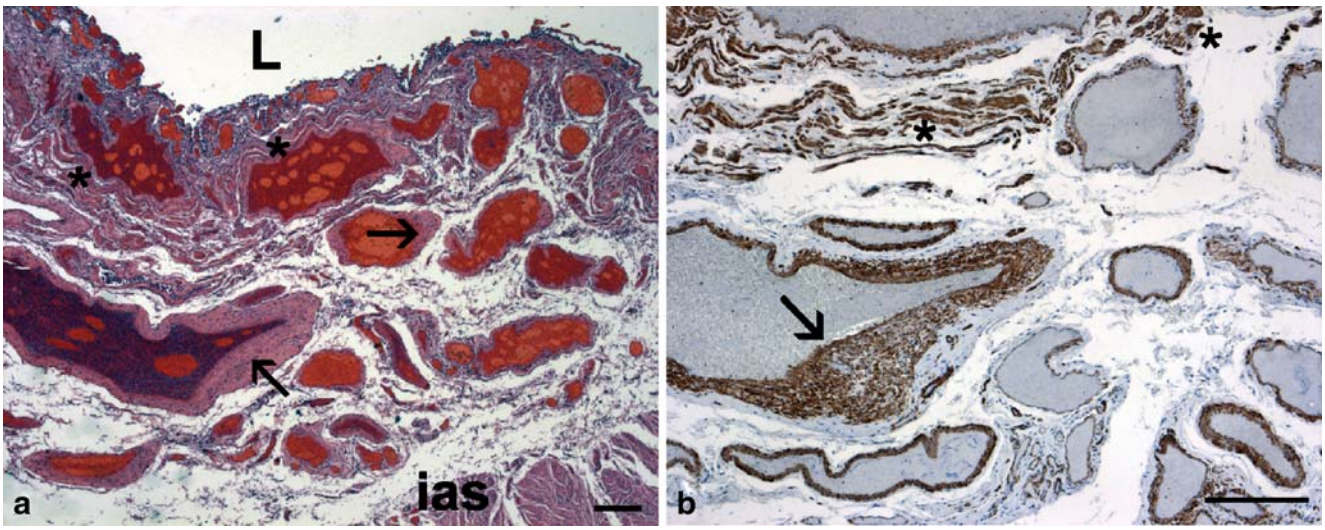

Fig. 1 Anorectal vascular plexus. Hematoxylin/eosin (**a**) and immunohistochemical (**b**) staining of a-SMA in a longitudinal section of an anorectal specimen (female, 67 years). Muscularis mucosae (*asterisks*), thickening of the muscular wall of dilated vessels (*arrows*), internal anal sphincter muscle (*ias*). *L* lumen of the rectum. *Bar* indicates 100 μm

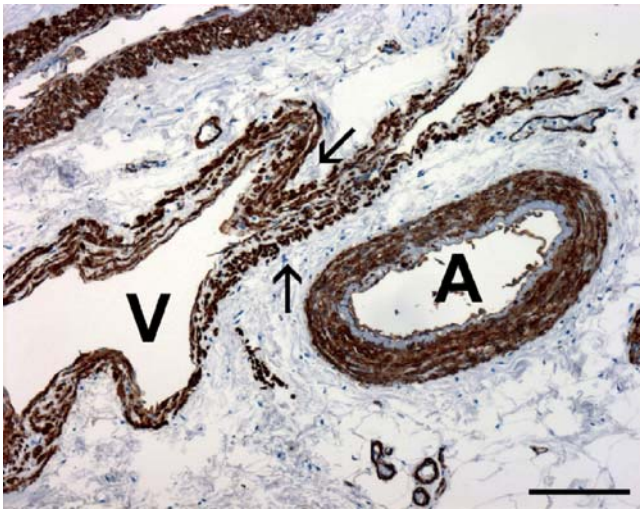

Fig. 2 Anorectal vascular plexus. Immunohistochemical staining of a-SMA in a longitudinal section of an anorectal specimen (male, 59 years). Dilated venous vessel (*V*) with collateral continuation of abruptly changing caliber (*arrows*). Arteriole (*A*). *Bar* indicates 50 μm

sisted of dilated vessels with thinned walls mainly composed of only two to three layers of smooth muscle (Fig. 6). However, there were interposed segments of regular wall thickness observed in the hemorrhoidectomy specimens (Fig. 6). In contrast to healthy anorectal specimens, a dense network of hypertrophied nerve fiber bundles was observed in the submucosa of hemorrhoidectomy specimens (Fig. 7).

Scanning electron microscopy study

In contrast to histological sections, scanning electron microscopy allowed a three-dimensional assessment of the architecture of AVP. At overview magnification, the

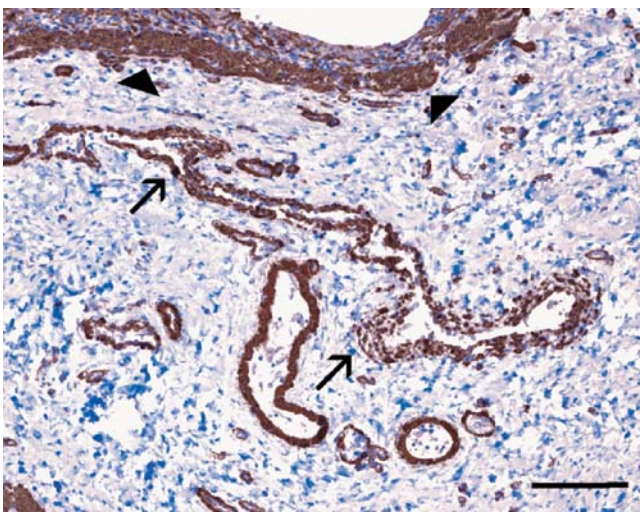

Fig. 3 Anorectal vascular plexus. Immunohistochemical staining of a-SMA in a longitudinal section of an anorectal specimen (female, 1.5 years). Dilated venous vessel with thickened smooth muscle layer (*arrows*); muscularis mucosae (*arrowheads*). *Bar* indicates 50 μm

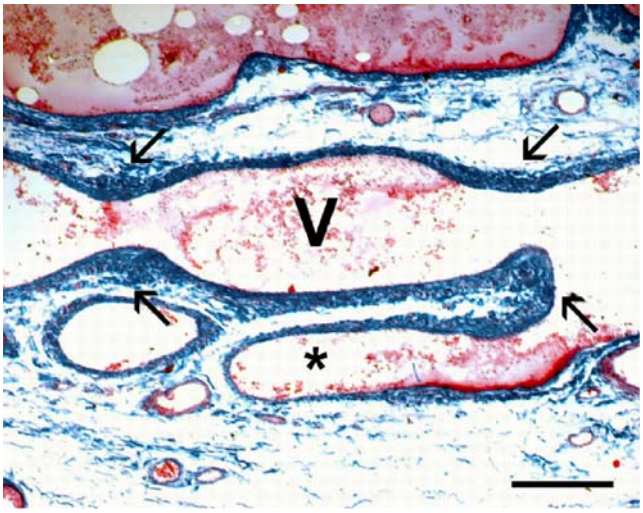

Fig. 4 Anorectal vascular plexus. Azan staining of a longitudinal section of an anorectal specimen (control group). Dilated venous vessels (*V*) appear at regular intervals and are separated by sphincter-like constrictions (*arrows*). Small-sized collateral vessel (*asterisk*). Bar indicates 50 μm

AVP was composed of densely distributed, tortuous vessels exhibiting remarkably cushion-like dilatation (vascular glomerula) at regular intervals (Fig. 8). The dilated vascular segments reached diameters of up to 1 mm and were frequently interconnected by small-sized collaterals with diameters alternating between 100 and 35 μm. The venous nature of these dilated vessels was confirmed by their typical endothelial cell nuclear imprints seen on the surface of the casts. These imprints appear as ovoid to round impressions with no preferred orientation (Fig. 8).

As observed in histological slides, venous dilatations were arranged in a pearl necklace fashion and were narrowed at their beginning and end by sphincter-like constrictions. The abrupt luminal narrowing between venous dilatations was easily discernible from circumferential impressions of the vascular wall. In some instances, circular impressions were also observed at the center of venous dilatations (Fig. 8). A clear morphological distinction between the afferent or efferent nature of vessels relative to the dilated vascular glomerula, however, could not be made with certainty. While veins of various caliber were found to drain into the dilated venous plexus, no arterial feeders were found. At different levels of the anorectal submucosa arterio-arterial as well as veno-venous, shortcuts were found.

Functional studies

All vessels supplying the AVP including submucosal branches and the previously described external arterial branches perforating the rectal wall right above the levator

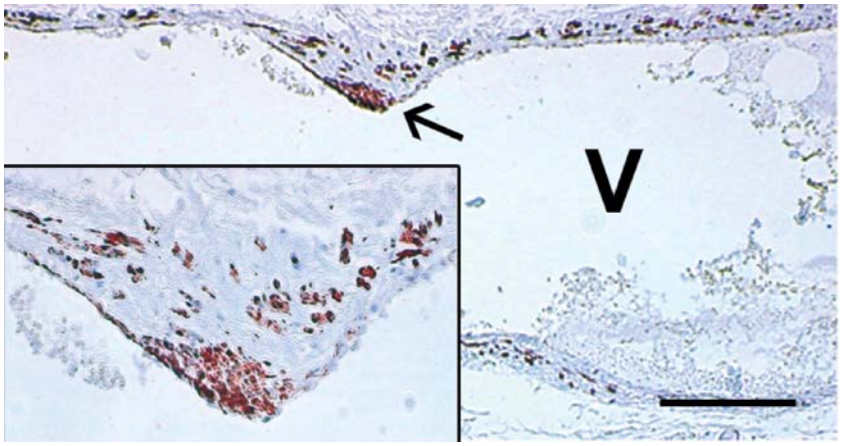

Fig. 5 Anorectal region. Immunohistochemical staining of a-SMA in a longitudinal section of an anorectal specimen (control group). Between venous dilatations (*V*), sphincter-like constrictions are discernible containing increased amount of smooth muscle cells (*arrow*). *Bar* indicates 25 μm (magnification, *insert*)

ani muscle [11] were assessed by means of CDUS and SWA. Non-pulse synchronous cyclic (10–15 min^{-1}) filling and drainage flow within the vascular plexus was recorded both in the control group and in the patients with hemorrhoids (Fig. 9a,b).

A subtle distinction between the direct afferent and efferent loop of the dilated vascular glomerula could not be clearly defined by CDUS/SWA according to the microscopic dimensions of the plexus (Fig. 8). However, significant differences in peak velocities (6.8±1.3 cm/s vs. 10.7±1.5 cm/s; P=0.026) and systolic acceleration velocities of the cumulative arterial afferent blood flow of the AVP (51±4 ms vs. 94±11 ms; P=0.001) were evident between the control group and patients with hemorrhoidal disease, depending on their grade (Fig. 9a,b).

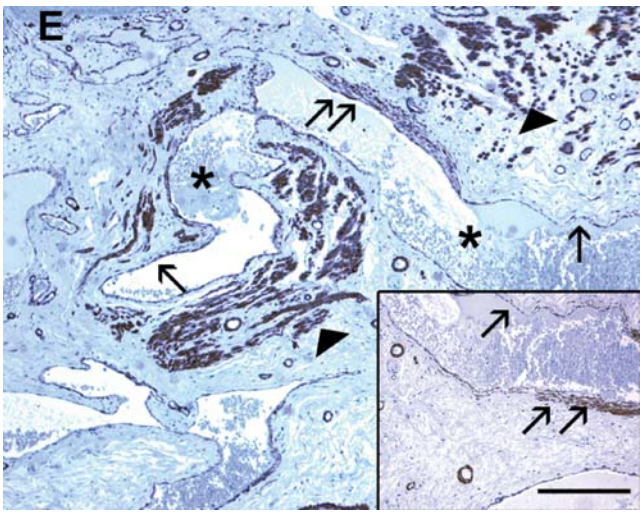

Fig. 6 Anorectal vascular plexus (*asterisks*). Immunohistochemical staining of a-SMA in a longitudinal section of an anorectal specimen (female, 39 years, hemorrhoidal disease IV° following open hemorrhoidectomy). Dilated, thin-walled vessels (*arrows*) within the submucosa are surrounded by dense connective tissue. The vascular wall is characterized by sparse smooth muscle cells with interposed segments of multi-layered smooth muscle tissue (*double arrow*, *insert*). Muscularis mucosae (*arrowheads*) and anal epithelium (*E*). *Bar* indicates 100 μm (detail view, *insert*)

Discussion

Hemorrhoidal disease still represents a major problem in colorectal practice, affecting approximately 5% of the general population with a peak prevalence noted between 45 and 65 years of age [12]. Its etiology and pathophysiology are based mainly on the vascular hyperplasia and the anal canal sliding theories [1], whereas some authors assign an additional causative impact of mucosal prolapse to the pathogenesis of hemorrhoidal disease [13]. Recent anatomical studies confirmed the arterial hyperplasia theory [14,

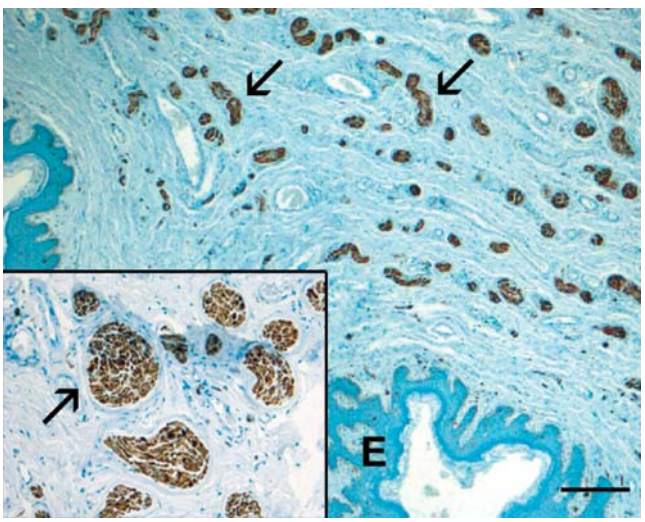

Fig. 7 Anorectal region. Immunohistochemical staining of protein S-100 in a longitudinal section of an anorectal specimen (female, 39 years, hemorrhoidal disease IV° following open hemorrhoidectomy). Densely distributed and hypertrophied nerve fiber bundles (*arrows*); anal epithelium (*E*). *Bar* indicates 100 μm (magnification, *insert*)

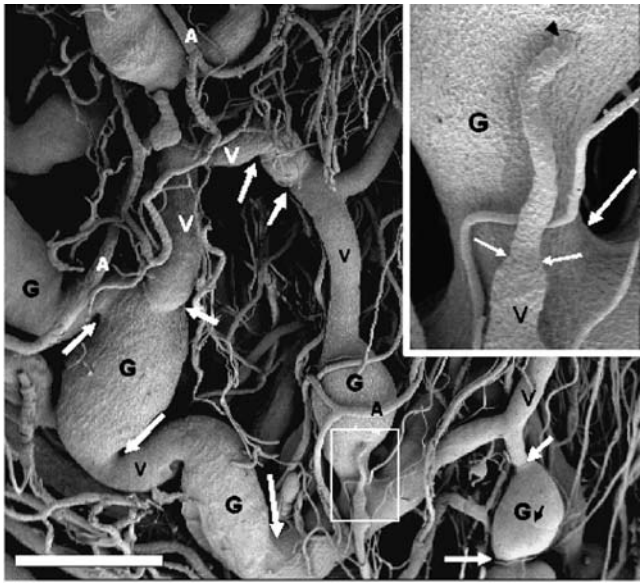

Fig. 8 Anorectal vascular plexus replicated by injecting the polymerizing resin Mercox-Cl-2R into SRA. Scanning electron micrograph. The vascular plexus consists of dilated venous vessels (vascular glomerula, *G*) with circular narrowings of various depths at their proximal and distal ends (*arrows*). Vessels interconnecting vascular glomerula are straight, wavy, or strongly kinked. *A* arteriole, *V* venous vessel. Detail from overview (*insert*). A small venule (*V*) drains into a vascular glomerulus (*arrowhead*). The diameter of the terminal portion of the venule is significantly reduced (*arrows*). *Bar* indicates 1,000 μm

15], and modern surgical approaches for treatment of hemorrhoidal disease aim to interrupt the submucosal arterial inflow [16], on the one hand, and to improve the venous drainage of the hemorrhoidal plexus [17], on the other hand.

Increased arterial inflow alone, however, does not explain the development of prolapsing hemorrhoids under the assumption of cyclic drainage of the AVP. This is also confirmed by previous observations made by our group that arterial hyperplasia of the plexus-supplying vessels [14] is unchanged following stapled hemorrhoidopexy. Stapling of the rectal mucosa therefore attempts to improve venous drainage *by resolving the hemorrhoidal prolapse or simultaneous rectal mucosa prolapse* [18] rather than to interrupt the arterial inflow as originally assumed. Albeit hypothetically, we suggest that hypertrophy of the hemorrhoidal cushions is caused by a temporary outflow insufficiency. As demonstrated both by scanning electron microscopy and histological studies, the AVP contains smooth muscle sphincters that may reduce the luminal diameter upon contraction and induce a temporary reduction in arterial inflow, thereby favoring venous drainage. If this mechanism fails, dilatation of submucosal anorectal vascular glomerula initiates a negative vicious circle with progressive vascular dilatation and insufficiency and, thus, hemorrhoidal hyperplasia. A basically similar mechanism can be found in the testis where concentric sphincter mechanisms exist within arteriovenous collateralizations reflecting the importance of regulated shortcuts in terms of functional blood supply [19].

We were not able, however, to distinguish morphologically between afferent and efferent vessels related to the AVP. According to our ultrasound studies, we can only indirectly assume such vanishing vascular sphincter mechanisms in the context of venous filling and drainage, as patients with hemorrhoidal disease—in contrast to healthy individuals—present significantly increased acceleration velocities in the cumulative arterial feeders of the AVP (Fig. 9b). However, this novel concept of hemorrhoidal blood drainage and filling is primarily based on the morphological findings.

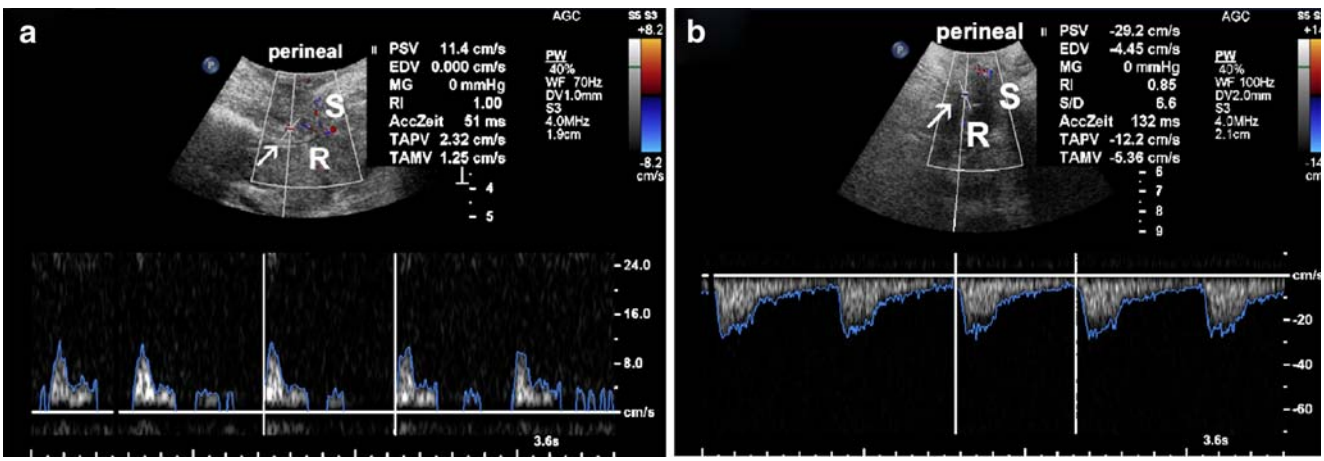

Fig. 9 Transperineal color Doppler ultrasound **a** in a male 31-year-old healthy individual (peak velocity, 11.4 cm/s; systolic acceleration velocity, 51 ms) and **b** in a male 66-year-old patient with hemorrhoidal disease III° (peak velocity, 29.2 cm/s; systolic acceleration velocity, 132 ms). Increased acceleration velocity in an afferent vessel (*arrow*) to the anorectal vascular plexus in the patient with hemorrhoidal disease (*bottom wave*). *S* anal sphincter muscles, *R* rectum

The distinct circular impressions and narrowings at both ends of the venous dilatations demonstrated with scanning electron microscopy (Fig. 8) clearly prove the obvious availability of structural prerequisites for regulating both arterial inflow and venous drainage. Immunohistochemical data support this putative function, revealing increased smooth muscle cell layers concentrically grouped around the sphincter-like regions adjacent to the vascular glomerula. In contrast, patients with hemorrhoids of Goligher grade IV clearly lacked this specific vascular architecture (Fig. 6).

Obviously, venous dilatations combined with sphincter-like thickening of the tunica media are confined to the submucosal vascular plexus of the ATZ. Neither mucosal vessels located within the lamina propria nor submucosal vessels located in the colorectum exhibited such distinct morphological features. A similar thickening of the tunica media was found in the corpora cavernosa of the penis (data not shown). Physiologically, relaxation of the internal anal sphincter muscle permits the dilated venous cushions to drain during defecation. We therefore suggest a possible autonomic co-innervation between the internal anal sphincter muscle and the intramural sphincters of the AVP. This has already been proven for the intermingling canalis ani muscle as the "levator et compressor haemorrhoidalis" [4] and hypothesized by our own group with regard to the longitudinal smooth muscle fibers within the rectogenital septum [20]. Small bundles of perivascular smooth muscle fibers were found in the ATZ and were determined to be identical to the Treitz muscle or the musculus submucosae ani [21]. In this context, we clearly demonstrate perivascular nerve fibers adjacent to the AVP including a considerable increase in and hypertrophy of these nerve fibers in hemorrhoidectomy specimens (Fig. 7) as compared to healthy individuals, as described elsewhere [22]. We assume that perivascular nerve fibers are a standard finding consistently found adjacent to the AVP, most likely regulating the vascular diameter and thus the blood flow velocity. Hypertrophy of these nerve fibers in hemorrhoidal disease may be explained by higher mechanical exposure to the prolapsing cushions, thus resulting in higher incidence of pain in hemorrhoids grades III–IV.

SWA could be an important diagnostic tool for functionally characterizing different stages of hemorrhoidal disease that are presently under definition: changes in arterial and venous spectra—all found in various grades of hemorrhoidal disease—may even resemble shunt flow patterns usually detected in arterio-venous shortcuts of intrahepatic vascular malformations. With regard to hemorrhoidal disease, we suggest that the described vascular sphincter mechanisms vanish, as demonstrated in our microscopic studies. Here, regular wall thickness appeared to be disturbed by the extensive dilatation of the AVP (Fig. 6), resulting in detectable alterations of blood perfusion pattern of the AVP (Fig. 9). It is important to underline the functional relevance of these vascular sphincter mechanisms demonstrated in the AVP in terms of gas-tight sealing of the anal canal, thereby contributing to the preservation of fecal continence. Alterations in the anorectal continence system for instance are likely to be associated with progressive hemorrhoidal disease [23]. It is well known that morphologic symbiosis of the AVP and subepithelial smooth muscle fibers, with the latter originating from the internal anal sphincter muscle, is crucial for the erection of the AVP and, finally, for the passive maintenance of the anorectal sphincter complex. This natural spontaneous activity in the adjacent musculature with a special constitution of ganglia cells can be clearly confirmed by combined positron emission and computed tomography [24] and is important for both function and failure of the anorectal sphincter system.

In summary, our findings suggest the existence of a specialized functional vascular network at the anorectal region, similar to that of the penile corpora cavernosa. Even though Stelzner et al. [2] hypothesized the presence of some kind of regulating veins in the corpus cavernosum recti, the present study provides clear morphological and functional evidence for distinct vascular glomerula equipped with sphincter-like constrictions most likely responsible for regulating the filling and drainage of the AVP. The present data suggest that the AVP possesses an intrinsic active contractile mechanism able to ensure effective blood transport through the corpus cavernosum recti [2]. Disruption of this intrinsic blood flow regulation and concomitant replacement of smooth muscle tissue with connective tissue appear to be key factors in the pathogenesis of hemorrhoidal disease. Moreover, the distinct morphological peculiarities of the hemorrhoidal cushions as demonstrated in the present study may also contribute to better understand the impact of the AVP in functional anorectal disorders like fecal incontinence.

Conflict of interest The authors declare that they have no conflict of interest.

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