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Endosonographic variations in thickness of anal sphincters in patients with hemorrhoids and normal population

Ahmed FARAG, Hany MS MIKHAIL, Mohamed Yehia ELBARMELGI, AbdRabou N MASHHOUR, Osama Refaie

General and Colorectal Surgery, Cairo University Kasr Alainy Faculty of Medicine, Giza, Egypt

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ABSTRACT

Objectives: The literature lacks enough research about the condition of the anal sphincters in patients with hemorrhoids. The aim of this study was to assess the thickness of internal and external anal sphincters (EAS) in patients with hemorrhoids using 3-dimensional endoanal ultrasonography.

Materials and Methods: This cross-sectional study non-randomized comparative study included 75 patients with internal hemorrhoids of 3rd and 4th degree and 75 healthy individuals as a control group. Endoanal ultrasound examination was done for all participants. The mean thickness of the internal anal sphincter (IAS) and EAS was measured (in mm) at the mid part of the anal canal at rest and during straining (pushing).

Results: Concerning IAS, its thickness during rest was apparently higher in the hemorrhoids group compared to the control group (p=0.071), and comparable in the two groups during push (p=0.175), so It did not change significantly with push in the two groups. The hemorrhoids group had significantly thicker EAS compared to the control group during rest and on push (p=0.025, and 0.022, respectively). Pushing resulted in a significant increase in the thickness of EAS in the control group (p=0.004), but not in the hemorrhoids group (p=0.132).

Conclusion: In hemorrhoidal disease, IAS and EAS are thickened during rest, but the EAS doesn't increase in thickness as occurring in control group in face of increased tension inside the anal canal during pushing. This may suggest a new pathophysiology for hemorrhoids as being a mechanism which developed to increase the thickness of the wall to protect anal canal wall from increased tension during push.

Keywords: Hemorrhoids; sphincters; endoanal ultrasound; pathophysiology

INTRODUCTION

Hemorrhoidal disease is one of the most common colorectal complaints in gastroenterology clinics. Worldwide, the prevalence of symptomatic hemorrhoids is estimated at 4.4% in the general population.[1] One-fourth of those patients consult a surgeon. Hemorrhoids are normally present in healthy individuals as fibrovascular cushions lining the anal canal. These cushions are considered sinusoids because some do not contain muscular

Address for Correspondence: Mohamed Yehia Elbarmelgi, General and Colorectal Surgery, Cairo University Kasr Alainy Faculty of Medicine, Giza, Egypt
E-mail: dr.yeho@yahoo.com **ORCID ID:** orcid.org/0000-0002-3903-3884
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walls. Physiologically, hemorrhoids protect the anal sphincter muscles and enhance anal canal closure during increased abdominal pressure.[2] The term "hemorrhoid" is commonly used to describe the pathologic hemorrhoid disease instead of the normal anatomic structure.[3] In about 40% of cases, hemorrhoids are asymptomatic.[4] In symptomatic cases, patients commonly complain of bleeding solely or with defecation, swelling, rectal pain, perianal irritation, hemorrhoidal protrusion and mucous discharge.[3] The exact pathophysiology of hemorrhoidal development is not clear. One theory, the sliding anal canal lining, suggests that hemorrhoids are an abnormal downward displacement of the anal cushions causing venous dilatation due to weakening of their supporting tissues.[5] In patients with hemorrhoids, some physiological changes of the anal canal have been observed. The resting anal pressure was higher in patients with hemorrhoids without significant change of the internal sphincter thickness.[6] Before hemorrhoidectomy, patients with hemorrhoids had lower rectal compliance, and more perineal descent in addition to high resting anal pressures. These changes reverted to the normal range within 3 months after surgery.[7] Farag suggested that the presence of an abnormality in the anal sphincter during push can lead to hemorrhoids development, as a protective mechanism to resist the increased anal canal wall tension.[8] Later it becomes pathologic when it starts to bleed, protrude, become inflamed or develops thrombosis.[8]

The literature is lacking enough research about the condition of the anal sphincters in patients with hemorrhoids. Endoanal ultrasonography (EAUS) is a well-tolerated and straight forward technique. It is commonly used now for the evaluation of the internal anal sphincter (IAS) and the external anal sphincter (EAS) in cases with fecal incontinence.[9]

The aim of this study is to utilize the availability of 3-dimensional EAUS for assessment of the thickness of internal and EASs in patients with hemorrhoids in comparison to healthy individuals.

MATERIALS AND METHODS

This cross-sectional non-randomized comparative study included 75 patients with hemorrhoids (3rd and 4th degree) and 75 healthy individuals as a control group. Patients and controls were recruited from the Outpatient Clinic of the Colorectal Unit in Kasr-El-Aini Hospital from May 2018 to October 2019. The study was approved by the scientific and Ethical Committee of the General Surgery Department. The procedure and was thoroughly explained to all individuals participating in the study and all of them consented to participate in the study. Patients were included if they had internal hemorrhoids of 3rd and 4th degree with no history of previous anal surgery nor concomitant anal disease (e.g., anal fissure). The control groups included individuals with no history of previous anal surgery nor or concomitant anal disease. After full history taking and thorough clinical examination, all participants had an enema half an hour before examination. With the participant in the left lateral position, digital rectal examination and proctoscopy were done to assess the degree of piles and to detect any concomitant disease which may exclude the case from the study. This was followed by endoanal ultrasound examination (Flex Focus 400 - type 1202 - endoanal ultrasound device) B & K from Denmark. The whole anal canal is depicted using 2D and 3D images. The thickness of the internal and EASs was measured (in mm) at the mid part of the anal canal at 3, 6, 9, 12 o'clock positions. Then, the mean was calculated (Figure 1, 2).

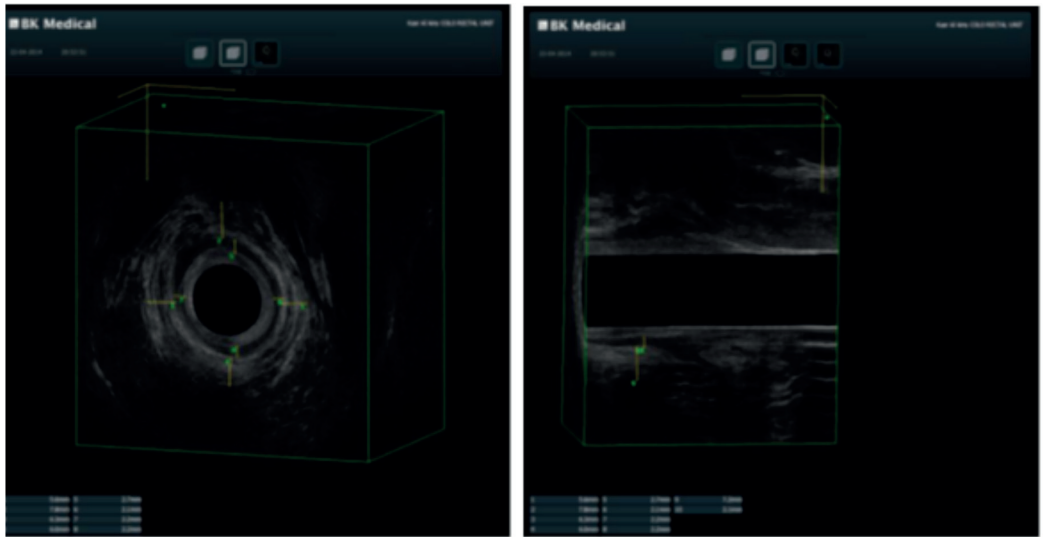

Figure 1. Internal and external anal sphincter thickness in control patient (during rest)

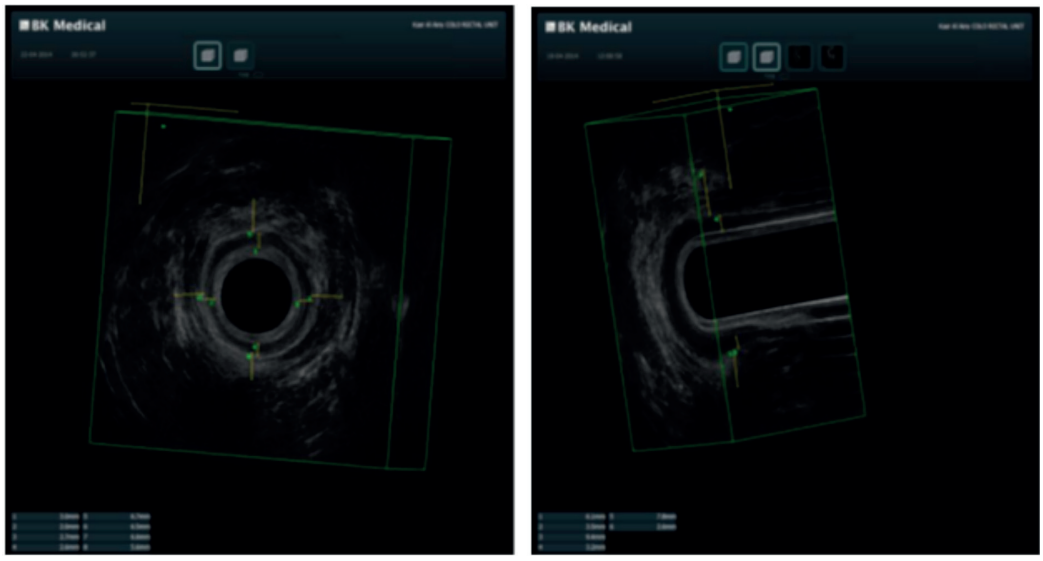

Figure 2. Internal and external anal sphincter thickness in patient with hemerrhoids (during rest)

The patient is asked to push "strain" and then the mean thickness of the internal and EAS during push was measured as previously described (Figure 3, 4). The mean thicknesses of internal and external sphincters in both groups were compared.

Statistical Analysis

All statistical calculations were done using computer programs SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 15 for Microsoft Windows. Data were described as a mean and standard deviation. Comparison of numerical variables between the study groups was made using Mann-Whitney U test. A p-value <0.05 was considered significant.

RESULTS

The two groups were comparable regarding gender. The mean age of the patients group was 39.2 years (range: 22-54 years) and 37.9 years (range: 21-53 years) for the control group ($p=0.76$). The diagnosis of control group individuals was a hernia in 60% (45 cases), chronic calcular cholecystitis in 20% (15 cases), simple nodular goiter in 20% (15 cases) of them. Sixty percent of the patients had third-degree hemorrhoids, while 40% had fourth-degree disease. The thickness of the IAS during rest was apparently higher in the hemorrhoids group compared to the control group, but the difference was not statistically significant ($p=0.071$). During push, the thickness of the IAS was comparable in the two groups (Table 1). There was no significant change of

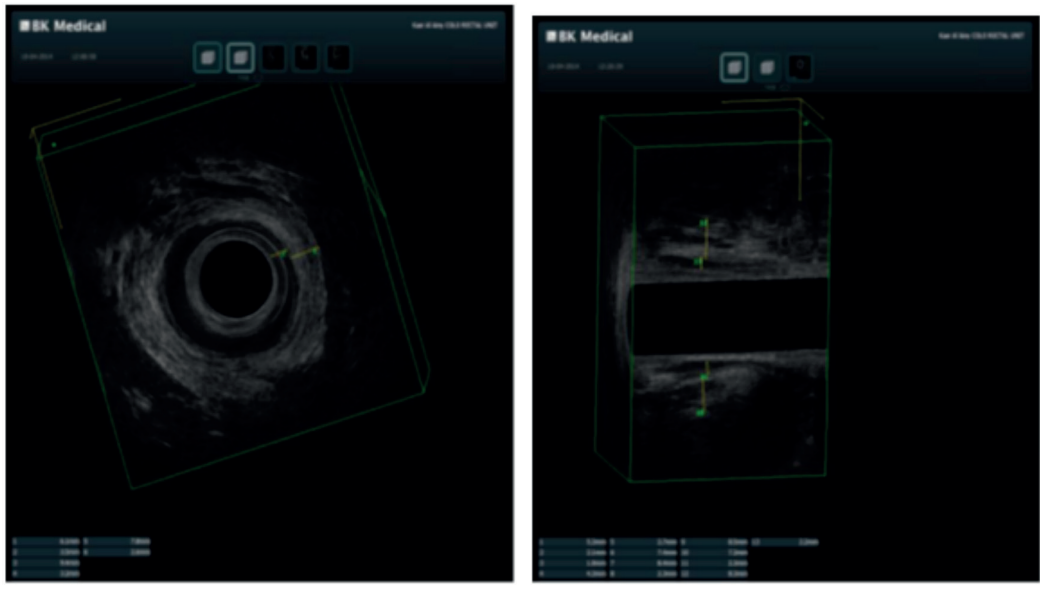

Figure 3. Internal and external anal sphincter thickness in control patient (during push)

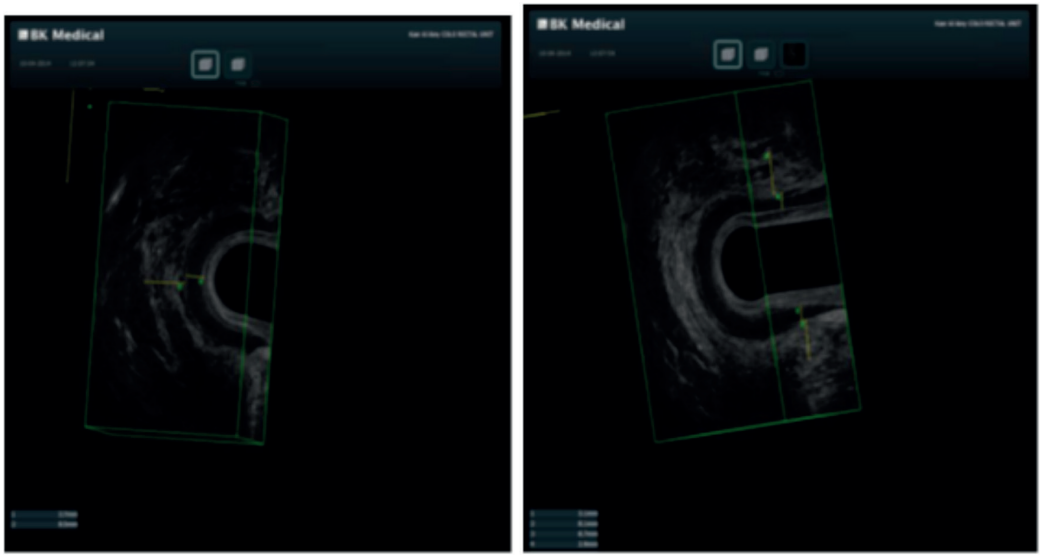

Figure 4. Internal and external anal sphincter thickness in a patient with hemerrhoids (during push)

Table 1. Thickness of the internal and external anal sphincters measured with endoanal ultrasonography in the hemorrhoids and control groups

	Hemorrhoids group (n=75)	Control group (n=75)	*p*-value*
Internal sphincter thickness (mm)			
During rest	2.4±0.5	2.1±0.4	0.071
During push	2.4±0.5	2.1±0.4	0.175
p-value§	0.243	0.310	-
External sphincter thickness (mm)			
During rest	6.6±1.1	5.7±0.9	0.025
During push	6.9±0.9	6.2±0.7	0.022
p-value§	0.132	0.004	-

*Comparison of the two group; §comparing measurements during rest and push

the thickness of the IAS with push in the two groups. Conversely, the hemorrhoids group had significantly thicker EAS compared to the control group during rest and on push (Table 1). Pushing resulted in a significant increase in the thickness of EAS in the control group (p=0.004), but not in the hemorrhoids group (p=0.132).

DISCUSSION

Anatomy and physiology of the anal canal are quite complex. EAUS is a valuable technique for evaluation of the anal sphincters and pelvic floor in patients with anorectal diseases.[10] The introduction of three-dimensional endosonography added new potentials in the diagnosis of anorectal diseases. Volume reconstruction of axial images can precisely present the anatomy of the anal canal and anal sphincters. Several articles described the use of EAUS in cases of anal sphincter defects,[11] anal fistulas,[12] perianal abscesses,[13] and anal tumors.[14] However, we found only a single study in the literature that studied anal sphincter anatomy in patients with hemorrhoids. The current study was designed to address the anatomy of anal sphincters in patients with hemorrhoidal disease in comparison to healthy subjects using three-dimensional endosonography. The study demonstrated that EAS thickness is significantly increased during straining in healthy subjects, but not in patients with hemorrhoids. Also, patients with hemorrhoids had significantly thicker EAS compared to normal subjects.

The flow equation and hybrid law[15] can explain the findings of the current study. According to the equation and the results of the present study, the anal canal wall thickness has to increase proportionately to protect the anal canal against the increasing wall tension. Hemorrhoids can be viewed as a trial to protect the bowel wall against increased tension by increasing the size of the anal cushions, as in these patients, the significantly thickened external sphincter in patients with hemorrhoids during rest, while it was not significantly thicker during push. This may suggest that hemorrhoid patient had already exhausted their

reserve by increasing the thickness of the wall and then develop hemorrhoids to compensate for increasing tension during defecation.

In the current study, the EAS was thickened in patients with hemorrhoids which might indicate increased activity of this sphincter. This increase in EAS thickness was suggested by Farag[15], is due to work hypertrophy in patients with hemorrhoids due to premature straining before full relaxation-thickening of the EAS as is seen in normal controls is evident by our results which show significant difference in thickness of external sphincter between hemorrhoid and control group (p=0.022). In the hemorrhoid patient group the increase in the EAS thickness during push was not significant (p=0.132) which may suggest that hemorrhoid patient had already exhausted their reserve in increasing the thickness of the wall and then develop hemorrhoids to compensate for increasing tension during defecation.[15]

This high anal canal wall tension may results in reflex spasm of anal sphincters which may lead to hypertrophy of the external sphincter muscle. This hypertrophy may lead to hemorrhoids by impairment of venous drainage of the hemorrhoidal venous plexus in patients predisposed to hemorrhoids by lax anal mucosa and congenitally weak mesenchyme. Those hemorrhoids will further increase the anal canal wall thickness, which will protect the anal canal wall in face of increased anal canal wall tension during defecation.

CONCLUSION

We conclude that the hemorrhoids develop as a protective mechanism to protect the anal canal against excessive tension during straining especially if the patient starts to strain before full relaxation-thickening of the EAS during defecation. As a result of this increased wall tension, the IAS showed a slight but not significant thickening in patients with hemorrhoids, while the EAS is significantly thicker in these patients. Behavioral instructions and education should be an integral part of treatment of hemorrhoids, which should aim at normal urgency defecation, i.e., to reply to the call of nature rather than to obsessively strain to induce it.

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ETHICS

Ethics Committee Approval: The study was approved by the Research Ethics Committee of Cairo University on February 2018.

Informed Consent: Informed consent was obtained.

Peer-review: Internally and externally peer-reviewed.

Contributions

Surgical and Medical Practices: A.F., H.MS.M., AR.N.M., O.R., M.Y.E.; Concept: A.F., Design: A.F., H.MS.M., AR.N.M., O.R., M.Y.E.; Data Collection or Processing: A.F., H.MS.M., AR.N.M., O.R., M.Y.E.; Analysis or Interpretation: A.F., H.MS.M., AR.N.M., O.R., M.Y.E.; Literature Search: O.R., M.Y.E.; Writing: A.F., H.MS.M., AR.N.M., O.R., M.Y.E.

DISCLOSURES

Conflict of Interest: No conflict of interest was declared by the authors.

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REFERENCES

1. Lorenzo-Rivero S. Hemorrhoids: diagnosis and current management. Am Surg 2009; 75: 635-42.
2. Beck DE. The ASCRS textbook of colon and rectal surgery. New York: Springer; 2011.
3. Sun Z, Migaly J. Review of Hemorrhoid Disease: Presentation and Management. Clin Colon Rectal Surg 2016; 29: 22-9.
4. Riss S, Weiser FA, Schwameis K, et al. The prevalence of hemorrhoids in adults. Int J Colorectal Dis 2012; 27: 215-20.
5. Lohsiriwat V. Hemorrhoids: From basic pathophysiology to clinical management. World J Gastroenterol 2012; 18: 2009-17.
6. Sun WM, Peck RJ, Shorthouse AJ, Read NW. Haemorrhoids are associated not with hypertrophy of the internal anal sphincter, but with hypertension of the anal cushions. Br J Surg 1992; 79: 592-4.
7. Ho YH, Seow-Choen F, Goh HS. Haemorrhoidectomy and disordered rectal and anal physiology in patients with prolapsed haemorrhoids. Br J Surg 1995; 82: 596-8.
8. Farag A. Use of the Hagen-Poiseuille law: a new mathematical approach for the integration and evaluation of anorectal physiological testing in patients with faecal incontinence and pelvic dyschezia and in normal controls. Eur Surg Res 1998; 30: 279-89.
9. Carrington EV, Scott SM, Bharucha A, et al. Expert consensus document: Advances in the evaluation of anorectal function. Nat Rev Gastroenterol Hepatol 2018; 15: 309-23.
10. Mascagni D, Grimaldi G, Naldini G. Endoanal Ultrasound. In: Mongardini M, Giofrè M, editors. Manag Fecal Incontinence [Internet]. Cham: Springer International Publishing; 2016 [cited 2018 Jul 22]. p. 23– 32. Available from: http://link.springer.com/10.1007/978-3-319-32226- 1_3
11. Albuquerque A. Endoanal ultrasonography in fecal incontinence: Current and future perspectives. World J Gastrointest Endosc 2015; 7: 575-81.
12. Felt-Bersma RJF. Endoanal ultrasound in perianal fistulas and abscesses. Dig Liver Dis 2006; 38: 537-43.
13. Kim MJ. Transrectal ultrasonography of anorectal diseases: advantages and disadvantages. Ultrasonography 2014; 34: 19-31.
14. Jacopo M. Endoanal ultrasound for anal cancer staging. Int J Colorectal Dis 2011; 26: 385-6.
15. Farag A. The use of flow equation in functional coloproctology: a new theory in anorectal physiology. Pelviperineology 2009; 28: 17-23.