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GASTROINTESTINAL PATHOLOGY IN SYSTEMIC SCLERODERMA

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Abstract
Gastrointestinal tract (GIT) involvement holds an important place among the visceral manifestations of systemic scleroderma (SSc). Pathologies of the esophagus, stomach, small and large intestines can range from mild functional disorders to the development of pronounced chronic inflammation with mucosal metaplasia and dysplasia, formation of multiple erosions, hemorrhages, and deep ulcers. This review discusses the main clinical manifestations, diagnostic possibilities, and treatment of gastrointestinal involvement in systemic scleroderma.

Keywords: Systemic scleroderma, gastrointestinal manifestations, diagnosis.

Introduction

Systemic scleroderma (SSc) is a systemic rheumatic disease that arises due to excessive fibroblast proliferation, overproduction of collagen, and systemic involvement of small vessels (arterioles) [1]. Although the "image" of this disease is primarily associated with skin thickening and vascular disorders (Raynaud's syndrome, trophic changes), SSc is also characterized by various visceral manifestations, among which gastrointestinal tract pathology occupies a leading position [2, 15–17]. SSc is the only systemic disease in which a characteristic esophageal lesion—scleroderma esophagitis—occurs. The causes of this pathology involve the involvement of smooth muscles and fibrosis of the submucosal layer in the lower third of the esophagus and the cardiac part of the stomach. A decrease in the tone of the cardiac sphincter, leading to its gaping (chalasia), and persistent gastroesophageal reflux (GER) result in the development of severe reflux esophagitis, accompanied by pronounced heartburn, odynophagia, dysphagia, retrosternal pain, and the appearance of erosions and ulcers in the lower third of the esophagus. Scarring of ulcerative defects can cause the formation of strictures in the area of the cardiac sphincter. Prolonged inflammation of the esophageal mucosa induces the development of gastric, and subsequently intestinal metaplasia—Barrett's esophagus—a potentially precancerous condition that is detected in 10–30% of patients with systemic sclerosis (SSc) [2, 18]. Overall, esophageal involvement is noted in 70–90% of SSc patients and is quite characteristic of the limited form of this disease (CREST syndrome), which is associated with the presence of anticentromere antibodies [2, 15–17].

Besides esophageal pathology, SSc can cause distinctive changes in the mucosa of the antral part of the stomach, with linear thickening of the folds, dilation of small blood vessels, and multiple hemorrhages—gastric antral vascular ectasia (GAVE), or “watermelon stomach.” This pathology can lead to recurrent bleeding and the development of iron-deficiency anemia [2, 16, 17].

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In 10–50% of SSc patients, involvement of the small intestine may develop. The pathology involves smooth muscle damage and fibrosis of its wall, leading to impaired peristalsis and decreased elasticity, while vascular changes cause increased permeability and reduced reparative potential of the mucosa. Disruption of the microbiota and bacterial overgrowth syndrome play an important role in the formation of intestinal lesions. The outcome is chronic enteritis with pseudo-obstruction, intestinal dilation, persistent constipation or, conversely, diarrhea and malabsorption.

Less commonly in SSc, the colon is affected, usually manifesting as persistent constipation. Intestinal manifestations of SSc carry serious complications: perforation, sepsis, and cachexia associated with impaired absorption of essential nutrients. It should be noted that intestinal involvement is characteristic of a more aggressive diffuse variant of SSc, associated with antibodies to topoisomerase (Scl-70) [2, 16, 17].

Systemic sclerosis (SSc) is a systemic rheumatic disease resulting from excessive proliferation of fibroblasts, collagen overproduction, and systemic involvement of small vessels (arterioles) [1]. Although the “appearance” of this disease is primarily associated with skin thickening and vascular disorders (Raynaud’s phenomenon, trophic changes), SSc is also characterized by various visceral manifestations, among which gastrointestinal pathology occupies a leading position [2, 15–17]. Thus, SSc is the only systemic disease in which characteristic esophageal involvement “scleroderma esophagitis” occurs. The causes of this pathology include involvement of the smooth muscles and fibrosis of the submucosal layer in the lower third of the esophagus and the cardiac region of the stomach. A decrease in the tone of the cardiac sphincter, leading to its opening (achalasia), and persistent gastroesophageal reflux (GER) result in the development of severe reflux esophagitis, accompanied by pronounced heartburn, odynophagia, dysphagia, retrosternal pain, and the appearance of erosions and ulcers in the lower third of the esophagus. Scarring of ulcerative defects may cause the formation of strictures in the area of the cardiac sphincter. Prolonged inflammation of the esophageal mucosa induces the development of gastric and then intestinal metaplasia—Barrett’s esophagus—a potentially precancerous condition detected in 10–30% of patients with systemic sclerosis (SSc) [2, 18]. Overall, esophageal involvement is observed in 70–90% of SSc patients and is quite characteristic of the limited form of the disease (CREST syndrome), which is associated with the presence of anticentromere antibodies [2, 15–17].

In addition to esophageal pathology, SSc can cause distinctive changes in the mucosa of the antral part of the stomach, including linear thickening of folds, dilation of small blood vessels, and multiple hemorrhages—gastric antral vascular ectasia (GAVE), also known as “watermelon stomach.” This pathology may lead to recurrent bleeding and the development of iron-deficiency anemia [2, 16, 17].

In 10–50% of SSc patients, involvement of the small intestine may develop. Smooth muscle pathology and fibrosis of the intestinal wall lead to impaired peristalsis and decreased elasticity, while vascular changes cause increased permeability and reduced reparative potential of the mucosa. Disruption of the microbiota and bacterial overgrowth syndrome play an important role in the development of intestinal lesions. The result is chronic enteritis with pseudo-obstruction, intestinal dilation, persistent constipation, or, conversely, diarrhea and malabsorption.

Less commonly, colonic involvement occurs in SSc, usually manifesting as persistent constipation. Intestinal manifestations of SSc are fraught with serious complications such as perforation, sepsis,

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and cachexia related to impaired absorption of essential nutrients. It should be noted that intestinal involvement is characteristic of the more aggressive diffuse variant of systemic sclerosis (SSc), which is associated with antibodies to topoisomerase I (Scl-70) [2, 16, 17]. Systemic sclerosis (SSc) is a systemic rheumatic disease that arises due to excessive proliferation of fibroblasts, overproduction of collagen, and systemic involvement of small vessels (arterioles) [1]. Although the "appearance" of this disease is primarily associated with skin thickening and vascular disorders (Raynaud's phenomenon, trophic changes), SSc is also characterized by various visceral manifestations, among which gastrointestinal pathology occupies one of the leading positions [2, 15–17].

Thus, SSc is the only systemic disease in which characteristic esophageal involvement "scleroderma esophagitis" occurs. The causes of this pathology are the involvement of smooth muscles and fibrosis of the submucosal layer in the lower third of the esophagus and the cardiac region of the stomach. Decreased tone of the cardiac sphincter, leading to its opening (achalasia), and persistent gastroesophageal reflux (GER) result in the development of severe reflux esophagitis, accompanied by pronounced heartburn, odynophagia, dysphagia, retrosternal pain, and the appearance of erosions and ulcers in the lower third of the esophagus. Scarring of ulcerative defects can lead to the formation of strictures in the cardiac sphincter region.

Prolonged inflammation of the esophageal mucosa induces the development of gastric, and subsequently intestinal, metaplasia—Barrett's esophagus—a potentially precancerous condition detected in 10–30% of SSc patients [2, 18]. Overall, esophageal involvement is observed in 70–90% of SSc patients and is quite characteristic of the limited form of this disease (CREST syndrome), which is associated with the presence of anticentromere antibodies [2, 15–17].

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In 10–50% of SSc patients, involvement of the small intestine may develop. Pathology of the smooth muscles and fibrosis of the intestinal wall leads to impaired peristalsis and decreased elasticity, while vascular changes cause increased permeability and reduced reparative potential of the mucosa. An important role in the development of intestinal lesions is played by disruption of the microbiota and bacterial overgrowth syndrome. The result is chronic enteritis with pseudo-obstruction, intestinal dilation, persistent constipation, or, conversely, diarrhea and malabsorption. Less commonly, colonic involvement occurs in systemic sclerosis (SSc), usually manifesting as persistent constipation. Intestinal manifestations of SSc are fraught with serious complications such as perforation, sepsis, and cachexia associated with impaired absorption of essential nutrients.

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Conclusion

Gastrointestinal involvement is one of the significant visceral manifestations of systemic sclerosis, substantially affecting patient health and, in some cases, leading to life-threatening complications. Scleroderma esophagitis, enteritis, and gastritis are pathologies that require careful attention and effective medical management. Differential diagnosis of these conditions and the choice of treatment strategy present certain challenges and require specialized knowledge; therefore, the management of patients with systemic sclerosis and gastrointestinal involvement should be coordinated with gastroenterology specialists.

References

1. Rheumatology. Clinical Guidelines. Edited by E.L. Nasonova. Moscow: GEOTAR-Media, 2010; 752 p. (in Russian).
2. Ebert E. Esophageal disease in progressive systemic sclerosis. Curr Treat Options Gastroenterol. 2008;11(1):64–69.
3. Ntoumazios S., Voulgari P., Potsis K., et al. Esophageal involvement in scleroderma: gastroesophageal reflux, the common problem. Semin Arthritis Rheum. 2006;36(3):173–181.
4. Kowal-Bielecka O., Landewe R., Avouac J., et al. EULAR recommendations for the treatment of systemic sclerosis: a report from the EULAR Scleroderma Trials and Research group (EUROSTAR). Ann Rheum Dis. 2009;68(5):620–629.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

5. Wipff J., Coriat R., Masciocchi M., et al. Outcomes of Barrett's oesophagus related to systemic sclerosis: a 3-year EULAR Scleroderma Trials and Research prospective follow-up study. Rheumatology (Oxford). 2011 Mar 16 [Epub ahead of print].
6. Rabeneck L., Cook K., Wristers K., et al. SODA (severity of dyspepsia assessment): a new effective outcome measure for dyspepsia-related health. J Clin Epidemiol. 2001;54(8):755–765.
7. Roberts D., Miner P. Safety aspects and rational use of a naproxen + esomeprazole combination in the treatment of rheumatoid disease. Drug Healthc Patient Saf. 2011;3:1–8.
8. Lazebnik L.B., Drozdov V.N., Kim V.A. Effectiveness of famotidine in the prevention of NSAID-induced gastropathy. Results of the Russian multicenter study ZASLON-1 (protection of the gastric mucosa from non-steroidal anti-inflammatory drugs). Expert Clin Gastroenterol. 2009;2:3–9. (in Russian)
9. Kalinin A.V. Gastroesophageal reflux disease: diagnosis, therapy, and prevention. Pharmateka. 2003;7:23–29. (in Russian)
10. Paull A., Trier J., Dalton M., et al. The histologic spectrum of Barrett's esophagus. N Engl J Med. 1976;295:476–480.

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