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Rectocele: Whether Systemic Disorders of the Motor Function of the Gastrointestinal Tract are Possible

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Rectocele is a SAC - like bulge of the rectum wall towards the vagina, accompanied by a disorder of the act of defecation. There is an anterior rectocele, which makes up the majority of cases of the disease, when the rectum wall through thinning or defects in the rectovaginal fascia protrudes in the direction of the vagina [1]. A rarer form of the disease is known – the posterior rectocele, in which the posterior wall of the rectum protrudes towards the anal-coccygeal ligament, that is, back towards the coccyx. According to G.I. Vorobyov [2], rectocele occurs in 15-43% of women; according to foreign authors, rectocele is detected in 15-80% of middle-aged and elderly women examined (Tjandra Van Laarheven (1999), Chen H/H (2001)). Anterior rectocele was observed in 48% of patients and posterior rectocele in 52% of patients with complaints of difficult rectal emptying [3]. Rectocele is considered as an isolated special case of pelvic floor prolapse or pelvic prolapse syndrome. Pelvic prolapse, in addition to rectocele. It includes prolapse of the vaginal walls, cystocele, rectocele, prolapse and prolapse of the uterus, enterocele, perineal prolapse, United by common causes and mechanisms of development [4].

This disease is polyetiological and widespread pathology – it accounts for 2.5 % of the total number of female genitalia, each pregnancy increases the risk of developing rectocele by 31%. The pathogenesis of rectocele is based on changes in the connective tissue structures of the pelvic floor. The production of collagen and elastin, proteoglycans decreases, and the spatial structure of the protein-carbohydrate complex of the connective tissue matrix is disrupted. As a result, the muscular ligamentous apparatus of the pelvic floor and, in particular, the rectovaginal fascia, ensures the normal location of organs (in women who gave birth by caesarean section, the risk of rectocele is relatively lower).

Two factors are key in the development of rectocele:

- failure of the musculoskeletal system of the pelvis;
- Chronic (long – term and frequent) increase in intra-abdominal pressure-an increase at the time of straining (lifting weights or frequent constipation), when coughing (chronic lung diseases, such as chronic obstructive pulmonary disease or bronchial asthma).

The pathogenesis of rectocele consists in the fact that part of the rectum stretches towards the vagina and gradually prolapses its wall, forming a SAC-like pocket. The main role in the development of rectocele is assigned to the divergence of the anterior portions of the muscles that raise the anus, thinning and overstretching of the rectovaginal septum with the formation of a diverticular protrusion of the anterior wall of the rectum. The birth of a large fetus may be a trigger factor in the development of the pathology under consideration [5-7].

Taking into account the symptoms, the following degrees of rectocele are distinguished:

- Grade 1: there are no complaints, the act of defecation is not violated, rectal examination palpates the protrusion of the anterior wall of the rectum;
- Grade 2: patients complain of difficulties during defecation and a feeling of incomplete bowel movement. Rectal examination reveals a SAC-like pocket that reaches the border of the vestibule of the vagina;
- Grade 3: patients complain of severe difficulty defecating. The anterior wall of the rectum and the posterior wall of the vagina protrude beyond the genital cleft.
- The clinical picture develops gradually. At the beginning, defecation becomes less regular, there is a tendency to constipation. There is a feeling of incomplete bowel emptying or a foreign body in the rectum. As symptoms worsen, patients with rectocele take laxatives or use enemas. Artificial stimulation of the act of defecation contributes to the aggravation of the existing pathology. Rectocele is progressing. However, despite numerous studies of rectal motility, a systematic study of the motor function of the gastrointestinal tract (GI) and biliary tract (VD) in rectocele has not been conducted.

Objective

To identify the role of disorders of the motor function of the gastrointestinal tract and VD in the development of rectocele.

Materials and methods

9 patients with rectocele of women aged 58±6.5 years of 1-2 degrees were under observation. Rectocele was combined in 66.7% of cases with adhesions in the small pelvis (a history of cesarean section, inflammatory diseases of the female genital organs , pathology of the ileocecal angle and colon), in 33.4% - with abnormal mobility of the pelvic floor muscles, with pain in the left colon and sigmoid colon in every third patient. In 33.4% of cases, there were chronic cholecystitis, chronic pancreatitis, and bacterial overgrowth syndrome (SIBR). The disease debuts with difficulty emptying the rectum, there is a feeling of incomplete evacuation of the rectum. The progression of the disease is accompanied by the need for manual assistance. A dense fecal lump in the blind rectovaginal pocket can cause the urge to defecate, and with prolonged presence – inflammatory changes. The 2nd degree of the disease is characterized by a feeling of incomplete emptying, difficult defecation. Patients resort to manual assistance. In the third degree, a finger examination of the rectum determines the protrusion of the anterior wall in the vagina beyond the external sphincter of the anus of the rectum. To the feeling of incomplete emptying is added a mandatory manual allowance, frequent urge to defecate, sphincter insufficiency, noticeable when trying to set a cleansing enema.

The comparison group consisted of 11 patients suffering from GERD

Motor function of the gastrointestinal tract and VD was recorded electromyographically by applying bipolar silver electrodes to the area of projection of the organ on the anterior abdominal wall. The amplitude-frequency characteristics of slow waves and spikes, the power of phase and tonic contractions, and propulsive activity were analyzed using the Conan-M hardware and software package with a bandwidth of 1-10 mV. Statistical analysis was performed using a nonparametric Mann-Whitney system (at $p < 0.05$).

Results

The frequency of slow esophageal waves was 18.0 ± 0.9 \ min (an increase of 28.5%, $p < 0.05$), the frequency of 0.12 ± 0.003 mV (an increase of 20%, $p < 0.05$), the power of tonic contractions was 2.16 ± 0.113 (an increase of 54.3%, $p < 0.05$). The rate of spikes was 4.0 ±0.1 (increase 299.8%, $p < 0.001$), amplitude 0.05 ± 0.002 mV (a decrease of 49.9%, $p < 0.05$), the power phase of the cuts – 0.2 ± 0.03 (increase 199,8% , $p <0.001$), the propulsive activity of 10.8 ± 1.3 (decline of 20.3%, $p < 0.05$).That is, with rectocele, the propulsive activity of the esophagus decreased. Electromyographically, the frequency of slow stomach waves is 9.7 ± 0.5 \min (increase by 78.2%, $p < 0.05$), the amplitude is 0.11 ± 0.004 mV (increase by 10%, $p < 0.05$), the power of tonic contractions is 1.067 ± 0.035 (increase by 53.5%, $p < 0.05$). The frequency of spikes was 4.8 ± 0.9 (an increase of 380%, $p < 0.001$), the amplitude was 0.03 ± 0.002 mV (a decrease of 69.9 %, $p < 0.05$), the power of phase contractions was 0.144 ± 0.012 (an increase of 44%, $p < 0.05$), and the propulsive activity was 7.41 ± 0.5 (a decrease of 11.8%, $p < 0.05$). That is, with rectocele, there is a decrease in the propulsive activity of the stomach.

The frequency of slow duodenal waves was 15.8 ± 0.7 \ min (a decrease of 28.2%, $p < 0.05$), the amplitude was 0.14 ± 0.003 mV (an increase of 39.9%, $p < 0.05$), the power of tonic contractions was 2.212 ± 0.136 (an increase of 0.5%, $p > 0.1$). The rate of spikes was 4.0 ± 0.21 (increase 299.9 %, $p < 0.001$), amplitude – 0.03 ± 0.002 mV (a decrease by 69.9%, $p < 0.05$), the power phase of reductions 0.12 ± 0,014 (an increase of 20%, $p < 0.05$), the propulsive activity of 18.43 ± 1.33 (decrease of 16.2%, $p < 0.05$). That is, rectacel showed a decrease in the motor function of the duodenum, apparently due to the development of SIBR. Electromyographically, the frequency of slow waves of the jejunum was 15.9 ± 0.7 \min (decrease by 20.5%, $p < 0.05$), the amplitude was 0.09 ± 0.002 mV (decrease by 10%, $p < 0.05$), the power of tonic contractions was 1.431 ± 0.115 (decrease by 28.5%, $p < 0.05$). The frequency of spikes was 3.3 ± 0.003 (an increase of 230%, $p < 0.001$), the amplitude was 0.015 ± 0.0004 mV (a decrease of 85%, $p < 0.05$), the power of phase contractions was 0.0495 ± 0.0031 (a decrease of 50.5%, $p < 0.05$), and the propulsive activity was 28.9 ± 1.4 (an increase of 44.5 %, $p < 0.05$). That is, there were phase changes in the propulsive activity of the small intestine – a decrease in the duodenum, due to SIBR, and the restoration of the jejunum, due, apparently, to the restoration of the gut microbiota.

The frequency of slow waves of the right colon was 11.2 ± 0.3 (an increase of 1.9 %, $p > 0.1$), the amplitude was 0.08 ± 0.002 mV (a decrease of 20%, $p < 0.05$), the power of tonic contractions was 0.896 ± 0.043 (a decrease of 18.5%, $p < 0.05$). The frequency of spikes was 4.0 ± 0.3 (increase by 300.1%, $p < 0.001$), the amplitude – 0.02 ± 0012mv (decrease by 80.1%, $p < 0.05$), the power of phase contractions – 0.08 ± 0.003 (decrease by 20%, $p < 0.05$), propulsive activity – 11.2 ± 0.61 (increase by 1.9 %, $p > 0.1$). That is, the propulsive activity of the right colon is within the reference values. Electromyographically, the frequency of slow waves of the left colon (proximal part) was 8.5 ± 0.4 \min (increase by 41.7%, $p < 0.05$), the amplitude – 0.15 ± 0.003 mV (increase by 49.9%, $p <0.05$), the power of tonic contractions - 1.275 ± 0.13 (increase by 112.5%, $p < 0.001$). The frequency of spikes was 4.5 ± 0.5 (increase by 350%, $p < 0.001$), the amplitude was 0.02 ± 0.001 m (decrease by 79.9%, $p < 0.05$), the power of phase contractions was 0.90 ± 0.003 (decrease by 10%, $p < 0.05$), and the propulsive activity was 14.1 ± 1.2 (increase by 135%, $p < 0.001$).

The frequency of slow waves of the recto sigmoid Department was 7.0 ± 0.5 \min (an increase of 40%, $p < 0.05$), the amplitude was 0.11 ± 0.003 mV (an increase of 10%, $p < 0.05$), the power of tonic contractions was 0.792 ± 0.04 (an increase of 58.4%, $p < 0.05$). The frequency of spikes was 4.3 ± 0.06 (an increase of 330%, $p < 0.001$), the amplitude was 0.02 ± 0.001 mV (a decrease of 80%, $p < 0.05$), the power of phase contractions was 0.086 ± 0.0035 (a decrease of 14%, $p < 0.05$), and the propulsive activity was 9.2 ± 0.7 (an increase of 81%, $p < 0.05$). That is, with rectocele, there is a decrease in the power of phase contractions of the circular muscles with a slight increase in tonic ones. The frequency of smooth muscles of the choledochus was 10.5 ± 1.2 \min (an increase of 16.7%, $p < 0.05$), the amplitude was 0.14 ± 0.002 mV (an increase of 39.9%, $p < 0.05$), the power of tonic contractions was 1.47 ± 0.08 (an increase of 63.3%, $p < 0.05$). The rate of spikes was 3.5 ± 0.2 (increase of 250%, $p < 0.001$), amplitude of 0.03 ± 0.0011 mV (a decrease by 69.9%, $p < 0.05$), the power phase of reductions 0,105 ± 0,003 (an increase of 5%, $p < 0.05$), the propulsive activity of 14.0 ± 1.3 (increased by 55.5 %, $p < 0.05$).

Electromyographically, the frequency of slow gallbladder waves was 9.4 ± 0.3 \min (an increase of 18.9%, $p < 0.05$), the amplitude was 0.13 ± 0.002 mV (an increase of 30%, $p < 0.05$), and the power of tonic contractions was 1.222 ± 0.11 (an increase of 52.8%, $p < 0.05$). The frequency of spikes was 4.0 ± 0.05 (an increase of 300.1%, $p < 0.001$), the amplitude was 0.03 ± 0.002 mV (a decrease of 70%, $p < 0.05$), the power of phase contractions was 0.12 ± 0.014 (an increase of 20%, $p < 0.05$), and the propulsive activity was 10.18 ± 1.10 (an increase of 27.3%, $p < 0.05$). That is, with rectocele, there is a moderate increase in the propulsive activity of the biliary system, possibly having a compensating (constipation) character. Conclusion. The main role in the pathogenesis of rectocele is assigned to the divergence of the anterior portions of the muscles that raise the anus, thinning and overstretching of the rectovaginal septum with the formation of a diverticular protrusion of the anterior wall of the rectum. In 66.7% of cases, rectocele develops in women who have a history of severe multiple births, as well as childbirth with a large fetus, which can be considered as a trigger factor for the pathology under consideration [5,6]. The most common cause of rectocele is perineal ruptures of various degrees – frequent complications of labor and occur in 4.2 – 39% of births [4,5]. Ruptures contribute to postpartum insufficiency of the pelvic floor and anal sphincter muscles. According to Zhukov B. N.(2009), the combination of rectocele with another anorectal pathology occurs in 32.8% of cases (hemorrhoids, anal fissure, polyps).

Analysis of changes in the propulsive activity of various parts of the digestive tract in rectocele showed that the propulsive activity of the esophagus. The stomach and duodenum is characterized by a decrease in propulsive activity, varying from 11.8 to 20.3 %. The motor activity of the jejunum exceeds the motility of the duodenum by 60.7%, which indicates the restoration of the intestinal microbiota or the presence of additional independent pacemakers in the jejunum. An increase in motor activity can compensate for spastic activity of the circular muscle layer of the colon, which aggravates the insufficiency of the pelvic floor musculoskeletal system.

Conclusions

- Electromyography can be used to assess the functional state of the muscles of the anorectal region, rectum and colon.
- Significant spike activity of the circular muscle layer of the colon was revealed, which worsens the violation of intestinal transit.
- rectocele revealed hypermotor dyskinesia of the biliary system with an increase in bile clearance, which makes it possible to compensate for violations of intestinal transit through the colon

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