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CLINICAL AND MORPHOLOGICAL FEATURES OF DESMOID TYPE FIBROMATOSIS

In order to study the clinical and morphological features of fibromatosis of the desmoid type, a literature review was conducted. It was found that fibromatosis of the desmoid type is a locally aggressive myofibroblastic tumor that occurs in muscular-aponeurotic structures. The clinical picture is affected by the localization of the tumor and its invasive growth into the surrounding tissues.

The complexity of diagnosis is due to the rarity and morphological similarity to soft tissue sarcomas. Immunohistochemical research is used to exclude malignancy. It was found that the features of fibromatosis of the desmoid type are characterized by pronounced diffuse nuclear expression of beta-catenin, reactions to DOG1, CD117, S-100 and SMA are negative.

Keywords: fibromatosis, desmoid tumor, immunohistochemistry, morphological features

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Introduction. Desmoid tumors (synonyms: desmoid fibromatosis, aggressive fibromatosis, desmoid fibroma) are rare connective tissue neoplasms [6, 24] with an intermediate degree of malignancy [5], arising in deep soft tissues [10] and characterized by locally aggressive growth [5, 6]. They can reach 5-20 cm in diameter [16] and lead to compression or invasion of adjacent structures [2, 6]. The growth pattern of this tumor is variable: it can gradually increase, remain stable, or in some cases spontaneously regress [10].

Desmoid tumors (DT) have a low incidence: from 2-4 [10, 29] to 5-6 cases per year per million people [11, 27], which is

approximately 0.03% of all tumors [9, 10, 16, 29] and less than 3% of soft tissue tumors [15, 17]. It should be noted that from 3.5% to 32% of cases are associated with familial adenomatous polyposis (FAP) [5, 10, 11, 27].

These tumors do not metastasize. About 20% arise in the abdominal cavity (the mesentery of the small intestine is most often affected) [13, 28], 16% on the abdominal wall (more often in women during or after pregnancy) [13] and 64% are extraperitoneal localizations (extremities, pelvic girdle, chest, head and neck) [13, 27]. DT can also affect the urinary system [18], mammary [12] and pancreas [28].

DT of the abdominal cavity usually present in two forms: large, dense formations that are palpable or sheet-like white plaques found in the mesentery [16]. DT is 2-3 times more common in women than in men [8, 27]. Most patients are diagnosed between the ages of 15 and 60 years [20], with the peak incidence occurring between the ages of 30 and 40 years [5, 20, 27]. Thus, the population of patients with DT includes a significant number of women of reproductive age (70%) [5, 23, 30, 31]. In turn, low incidence is observed in children, with its peak occurring between the ages of 5 and 8 years [2].

The low prevalence of this pathology limits awareness of it [8]. In turn, diagnosis is complex due to morphological heterogeneity and variability of clinical manifestations [8, 18].

Treatment of DT is a challenge for healthcare due to their diverse clinical presentation and unpredictable disease

course [6] with cycles of tumor progression, stabilization [26], and spontaneous regression [17] observed in 20-30% of patients within 2-3 years [31]. All this affects the quality of life, as well as daily activities in some patients [6].

The aim of our study is to analyze publications devoted to the study of clinical and morphological features of desmoid fibromatosis.

Materials and methods. In this review, we examined the literature on desmoid fibromatosis. We searched PubMed, Web of Science, and Google Scholar for the period 2020–2025. Search terms included fibromatosis, desmoid tumor, immunohistochemistry and morphological features.

A microscopic method conducted at the Belgorod Pathological Anatomy Bureau was also used during our own research. The material was fixed in 10% neutral buffered formalin for 24 hours, then it underwent histological processing in a Thermo Scientific Excelsior AS closed-loop histoprocessor (sequential dehydration, degreasing, and tissue impregnation with paraffin). Sections of 4 µm thickness were cut from the prepared paraffin blocks using a Thermo Scientific HM340e semiautomated rotary microtome. The sections were stained with hematoxylin and eosin, and antibodies manufactured by Cell Marque (USA) were used for immunohistochemical examination: beta-catenin (clone E247), CD117 (clone YR145), DOG1 (clone SP31), S-100 (clone 4C4.9), SMA (clone 1A4).

Results and discussion. Desmoid fibromatosis (DF) is a locally aggressive

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myofibroblastic tumor [18] that arises in musculoaponeurotic structures [21, 22]. The World Health Organization (WHO) defines desmoid as a clonal fibroblastic proliferation that arises in deep soft tissues, characterized by infiltrative growth and a tendency to local recurrence without the ability to metastasize [11, 22, 23].

The term "desmoid tumor" was introduced in 1838 by Mueller and comes from the Greek term *desmos*, indicating its consistency and resemblance to tendons [4, 11]. However, the first description of this disease was made in 1832 by MacFarlane [11].

The etiology of DF is not fully understood [11, 23] and genetic, endocrine and physical factors play a decisive role in the development and growth of the tumor [9]. Depending on the etiology, DF can be divided into two groups: sporadic and familial [16, 30].

Sporadic DF occurs in 85-90% of cases, predominantly in extraperitoneal locations [11, 21] and is associated with various factors. Thus, most patients have a history of surgery or trauma [10], including breast [14], pregnancy [9], cesarean section, hormonal fluctuations [10, 14], and use of oral contraceptives [11, 21]. However, the exact role of hormonal influences has not been fully studied [19, 23].

In contrast, the familial form of DF is an autosomal dominant inherited form

associated with familial adenomatous polyposis (FAP) [16]. These tumors are located predominantly in the abdominal wall or abdominal cavity, mesentery, and visceral organs [6, 11, 21, 29].

The pathogenesis of DF also remains unclear, but studies have shown that activation of the Wnt/ β -catenin signaling pathway is crucial in the development of DF [9, 10, 33]. The Wnt protein plays an important role in the proliferation of mesenchymal cells. Mutations in this signaling pathway lead to the absence of the Wnt protein and disruption of the regulation of fibroblast proliferation, resulting in mesenchymal stem cells remaining undifferentiated (in a proliferative state) [16]. β -catenin is a molecule with multiple functions that are regulated by the adenomatous polyposis coli (*APC*) gene and the Wnt signaling pathway [11, 20, 30].

Most sporadic tumors are associated with a somatic [31] mutation in exon 3 of the β -catenin 1 gene (*CTNNB1*) [6, 9, 20-22, 29], most commonly at mutation sites *T41A* (50-55%), *S45F* (25-35%) and *S45P* (9-10%) [10, 11, 31].

The *CTNNB1* mutation interferes with the phosphorylation of β -catenin protein, blocking its proteasomal degradation in the cytoplasm. This leads to the accumulation of β -catenin, which translocates to the nucleus [4, 10, 31], activates transcription factors [9, 20] and forms a complex with T-cell factor/lymphoid enhancer

factor (TCF/LEF), increasing the expression of target genes [10, 20], which promotes cell proliferation, differentiation, migration, and apoptosis [9].

In DF associated with FAP, a mutation in the *APC* gene is usually observed [10]. Most mutations are located on chromosome 5q21 [16], which interferes with the phosphorylation of β -catenin, leads to an increase in the amount of cytoplasmic β -catenin and increased cell proliferation [10], which ultimately leads to tumor development [9, 11, 18, 21].

The second pathway involved in the development of desmoid tumors is the Notch pathway, which is activated in response to dysregulation of the Wnt pathway [8, 14]. Overactivation of the Notch pathway in desmoid tumors can be regulated by γ -secretase inhibitors, as they block Notch signaling through selective inhibition of γ -secretase-mediated cleavage of Notch receptors [8, 9, 17].

Although DF do not metastasize, they can exhibit a wide range of clinical manifestations, including spontaneous regression, indolent progression, or potentially life-threatening organ damage [8, 32].

Clinically, the tumor appears as a dense, well-defined or poorly defined mass [4]. It should be noted that the clinical picture of DF is influenced by the location of the tumor [18] and its invasive growth into surrounding tissues. This can affect vital structures or disrupt body

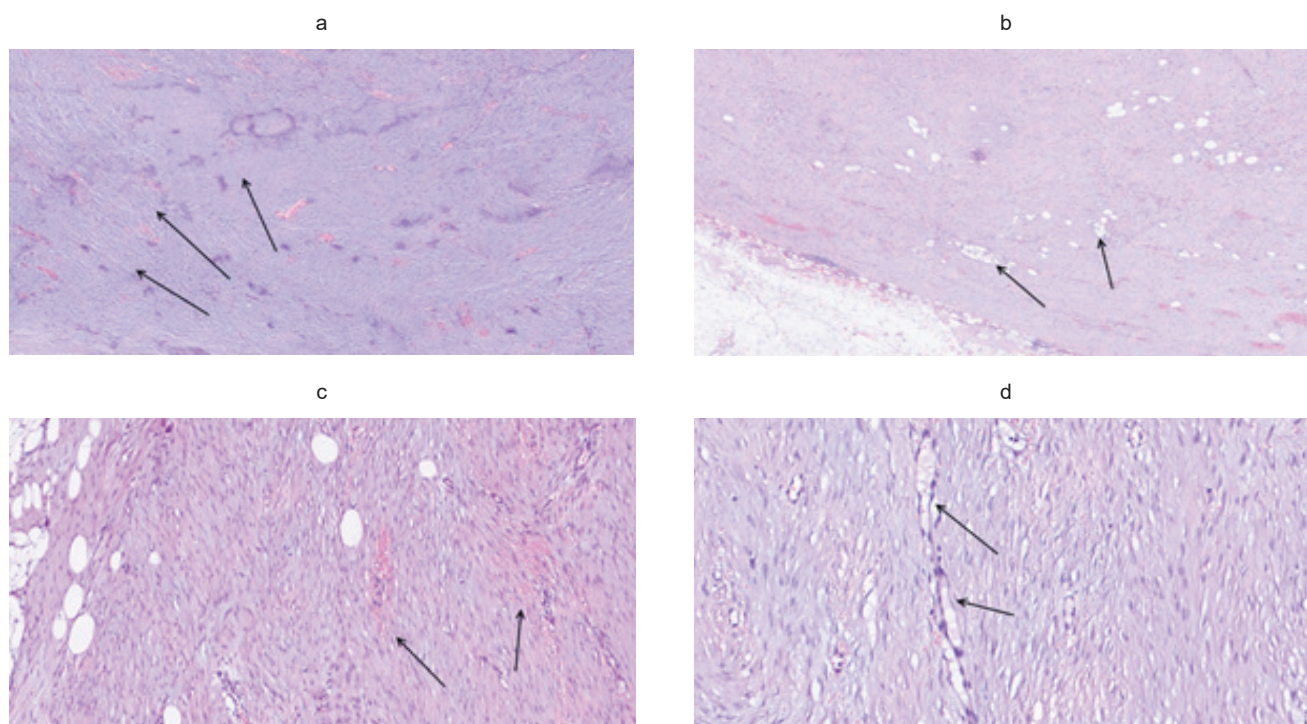


Fig. 1. Patient, born in 1955. Desmoid fibromatosis in the stomach wall. a – the tumor is represented by multidirectional bundles of spindle-shaped cells; b – infiltrative tumor growth in the fatty tissue of the omentum; c – extravascular accumulations of erythrocytes (extravasates); d – elongated capillary blood vessels (magnification a, b x50, c x100, d x200, staining: hematoxylin and eosin).

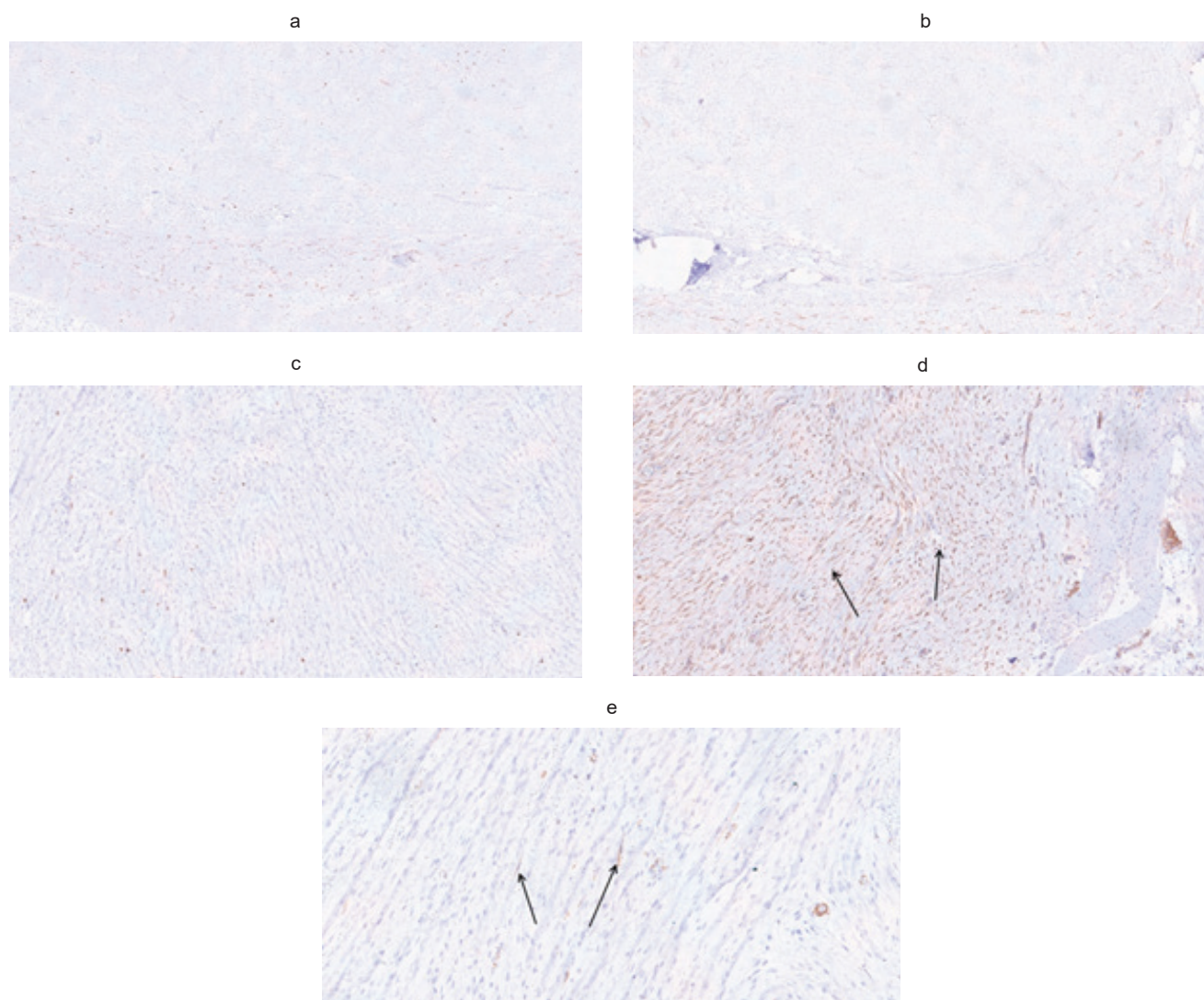


Fig. 2. Patient, born in 1955. Desmoid fibromatosis in the stomach wall. a - tumor cells lack CD117 expression (stain: antibody to CD117); b - tumor cells lack DOG1 expression (stain: antibody to DOG1); c - tumor cells lack S-100 expression (stain: antibody to S-100); d - tumor cells show diffuse nuclear expression of beta-catenin (marked with arrows, stain: antibody to beta-catenin); e - tumor myofibroblasts show weak cytoplasmic expression of smooth muscle actin (marked with arrows, stain: antibody to SMA) (magnification a, b x50, c, d x100, e x200).

functions [26], compress blood vessels and nerves [20, 31], cause paresthesia, severe pain or polyneuropathy [4, 11, 14, 25], and also destroy bones and affect muscles [31].

Thus, tumors located in the abdominal cavity grow asymptotically until they reach a large size. They cause intestinal obstruction, ischemia and, in rare cases, perforation, bleeding [11, 24, 31], abscess [23], fistula [9], as well as flatulence, constipation [3, 9, 25], weight loss, cachexia and malaise [9, 31], ureteral obstruction and hydronephrosis [3, 15, 16]. In case of invasion of the bladder, clinical manifestations of DF are a sensation of volume with frequent urination or hematuria [18].

Extraperitoneal DF are most often localized in the head and neck region, where they affect the airways or large

vessels, which can manifest as dysphonia and shortness of breath. In patients, tumors in the extremities manifest as palpable masses [23]. Limited joint mobility [5, 8, 15, 31], muscle contractures [23], problems with arm or hand movement, lameness, or other movement disorders may also be observed [17].

In addition, specific symptoms of DF may negatively impact (everyday life): study, work, psychosocial functioning [8, 15, 25], cognitive and emotional spheres [31]. Fatigue, insomnia, anxiety and depression may be observed [5]. In rare cases, a fatal outcome is possible [26], which is due to the local aggressiveness of the tumor, especially with intraperitoneal location [16].

FAP is associated with adenomatous polyps of the colon (tubular, villous, tubulovillous), adenomatous polyps of the

stomach and small intestine, and a risk of developing periampullary carcinomas. In addition, osteomas and various types of skin lesions (fibromas, neurofibromas, pigmented skin lesions) are observed in 50% of cases, occurring in almost two-thirds of patients [16].

Tumors associated with FAP usually progress more aggressively and present 10 years earlier as larger multifocal tumors than in the sporadic variant [11].

Accurate differential diagnosis is a serious challenge due to its rarity and numerous histological and clinical similarities with other soft tissue sarcomas. According to some authors, the misdiagnosis rate during initial examination can reach 30-40% [5, 24].

Macroscopically, DF appear as dense, uneven, elastic [16] formations of gray or white color, elongated in the direction

of muscle fibers and resembling fibrous scar tissue [11, 16, 31]. A cross-section reveals a shiny white surface with a rough trabecular structure [16].

The histological hallmark of DF is a heterogeneous, poorly defined [11] and uniform proliferation of elongated spindle-shaped cells with rare mitotic figures [29] that resemble myofibroblasts embedded in a collagen-rich stroma and unencapsulated vasculature. Atypia, necrosis or mitoses are typically absent. The nuclei may contain euchromatin or heterochromatin [11, 18, 24, 31]. In turn, the proliferation of cells without clear boundaries with uneven collagen deposition may mimic the proliferative phase of wound healing [14, 19]. These tumors are unencapsulated and infiltrate surrounding tissues, often merging with adjacent normal structures [2] and demonstrating intratumoral heterogeneity [14].

Our study is consistent with the authors' opinion, since DF consists of multidirectional bundles of spindle-shaped cells, elongated capillary-type blood vessels, and extravascular accumulations of erythrocytes (fig. 1 a-d).

Immunohistochemical testing for specific cellular markers of various origins is an effective method for differentiating DF from other tumors. DF has characteristic immunohistochemical features, primarily nuclear staining of β -catenin, to be considered positive [14, 19, 28], and can also be positive for vimentin and smooth muscle actin, indicating their fibroblast origin. Their mitotic activity is usually low and they do not have marked nuclear atypia or necrosis, which distinguishes them from malignant sarcomas [2, 18], cyclooxygenase-2 (COX-2) and often β -estrogen receptors [31], androgens [11, 16] and negative expression of CD34, CD117, S-100 and SMA, which excludes gastrointestinal stromal tumor, schwannoma, leiomyoma and leiomyosarcoma, respectively [16, 19, 23, 28].

The results of our immunohistochemical study are consistent with the results of other authors; thus, diffuse nuclear expression of beta-catenin is detected in tumor cells; reactions to DOG1, CD117, S-100 and SMA are negative (Fig. 2 a-e).

Due to the unpredictable course of DF, it resolves on its own in 20-28% of cases, remains stable in 32-60% of cases, and progresses in 20-40% of cases [6]. Due to possible spontaneous tumor regression, dynamic observation of asymptomatic patients is recommended [20]. Until the 2010 [7], the main method of treating this pathology was surgery [1]. It should be noted that resection of locally aggressive tumors without clear borders [13]

and invasive growth patterns [28] leads to local recurrences in more than 40% of cases [7], and also negatively affects local soft tissue structures, including muscles, nerves, or blood vessels, which can lead to more serious complications [13]. Risk factors for recurrence include patient age under 30-37 years, tumor size greater than 5 cm in diameter, and a history of trauma in the area of the primary tumor [13].

In recent decades, active surveillance in combination with pain relief [18, 31, 33] has been recommended as an alternative to surgical treatment [9]. The term active surveillance was introduced in the 1990. Initially, active surveillance was offered only to patients with recurrent tumors, but after 2005, it was also offered to patients with primary tumors [1]. This approach to the management of patients with this pathology is justified, since spontaneous tumor regression is possible [1, 33] without treatment [31].

Conclusion. The analysis revealed that desmoid fibromatosis is a locally aggressive myofibroblastic tumor arising in musculoaponeurotic structures. The clinical presentation is influenced by the tumor's location and its invasive growth into surrounding tissue.

The diagnosis is challenging due to its rarity and morphological similarity to soft tissue sarcomas. Immunohistochemistry is used to exclude malignancy. Desmoid fibromatosis is characterized by pronounced diffuse nuclear expression of beta-catenin, and negative reactions to DOG1, CD117, S-100, and SMA, which we confirmed in our study.

The authors declare no conflict of interest.

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THE NEUROPROTECTIVE POTENTIAL OF BILINGUALISM IN THE PREVENTION OF COGNITIVE IMPAIRMENT

This literature review analyses current scientific research that focuses on the impact of bilingualism and multilingualism on human cognitive functions. The focus is on the impact of two or more language experiences on executive functions such as inhibitory control, cognitive flexibility and working memory. The article examines the concept of «bilingual advantage» and the factors that modulate its manifestation, as well as the role of bilingualism in the formation of cognitive reserve.

Keywords: bilingualism, multilingualism, cognitive functions, neuroplasticity, cognitive reserve, neuropsychology.

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The phenomenon of bilingualism has stimulated scientific interest in studying how the experience of managing multiple language systems affects cognitive processes and brain structure.

In the context of globalization and growing cross-cultural communication, the phenomenon of bilingualism and multilingualism is attracting increasing attention from researchers in psychology, neuropsychology and neurology.

A central focus in research on the cognitive effects of bilingualism is the study

of its influence on executive functions – a set of higher-order cognitive processes responsible for planning, attention control, ignoring distractions, and multitasking.

The key components of executive functions are inhibitory control, cognitive flexibility, and working memory. The "bilingual advantage" hypothesis suggests that the constant need for bilinguals to control and manage two simultaneously activated language systems, by inhibiting the irrelevant language, acts as continuous training for executive functions [32].

Numerous studies confirm the existence of this advantage. For example, the work of A.N. Veraksa and co-authors (2020) demonstrated that bilingual children exhibit higher performance in tasks requiring attentional switching and the suppression of impulsive responses compared to their monolingual peers [18].

The constant practice of switching between languages seems to develop a general ability for cognitive flexibility, that is the ability to switch between different tasks or mental settings.

Studies using non-verbal tests, such as the Stroop task or the task-switching paradigm, often show that bilinguals perform better on them, with faster reaction times and fewer errors [16]. All of the above is related to the fact that the mechanism used to inhibit one language and activate another is domain-general and extends its influence to other cognitive tasks requiring control.

However, simply being bilingual or multilingual is not enough to protect against cognitive decline. It is important to note that only certain types of so-called "active" bilingualism have the greatest impact on brain health, namely those where individuals are sufficiently exposed to their languages, use them regularly, and are actively engaged in situations that require switching between languages [14].

Bilinguals constantly face an additional cognitive load: unlike monolinguals, they have to control their languages. Research shows that all languages of bilinguals/multilinguals are constantly activated, regardless of contextual necessity or conscious intention. Lan-

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