

FEATURES OF COMBINED ODONTOLOGICAL AND OPHTHALMOLOGICAL PATHOLOGIES

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Abstract

A wide range of complex pathologies affecting the dento-alveolar system and the visual apparatus are well documented in medical literature. These conditions include neuralgic pain that radiates to the eyes, vasomotor disturbances accompanied by conjunctival congestion, alterations in salivary and tear secretion patterns, and motor dysfunction affecting ocular movements. Additionally, there are general neurotrophic disorders affecting both the dentoalveolar region and the eyes. Dental conditions such as caries, periodontitis, periostitis, abscesses, gangrenous lesions, and periodontal diseases are particularly prone to causing ocular changes. In cases of dental granulomas and chronic periodontal inflammation, not only the trigeminal nerve endings but also sympathetic nerve fibers become irritated, leading to a pathological reflex response in the eyes and other organs. Furthermore, foci of pathological stimulation may arise following treatment involving depulped teeth or those sealed or crowned with metal restorations. Odontogenic accommodation palsy is also a known complication.

Keywords: the appearance of flickers, dark and light spots, stars, stripes or fog in front of the eyes, decreased vision of various duration in some cases can be explained by reflex vascular reactions originating from dental foci.

QO'SHMA ODONTOLOGIK VA OFTALMOLOGIK PATOLOGIYALARNING XUSUSIYATLARI

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Annotatsiya

Dentoalveolyar tizim va ko'zning ko'plab kombinatsiyalangan patologiyalari ma'lum: ko'zga tarqaladigan og'riqli nevralgiya, kon'yunktiva giperemiyasi bilan kechadigan vazomotor buzilishlar, tupurik va ko'z yoshi suyuqligining gipo- va gipersekretsiyasi hodisalari, ko'zning motor apparati va umumiy neyrotrofiyalar bilan bog'liq ko'z motor buzilishlari. Ko'zdagi o'zgarishlar kariyes, periodontit, periostit, xo'ppoz, gangrena, periodontal kasallik, granuloma kabi tish kasalliklarida ko'proq uchraydi. Tish granulomalari va surunkali periodontal yallig'lanish bilan nafaqat trigeminal, balki simpatik asabning terminal uchlari tirnash xususiyati keltirib chiqaradi, bu ko'z va boshqa organlarga patologik refleks ta'sir ko'rsatadi. Patologik tirnash xususiyati o'choqlari muhrlangan yoki metall tojlar bilan qoplangan depulpatsiya qilingan tishlarni davolashdan keyin paydo bo'lishi mumkin. Odontogen akkomodatsiya falajining ma'lum holatlari mavjud.

Kalit so'zlar: ko'z oldida miltillashlar, qorong'u va engil dog'lar, chiziqlar yoki tumanlarning paydo bo'lishi, ba'zi hollarda turli xil davomiylikdagi ko'rishning pasayishi tish o'choqlaridan kelib chiqadigan refleks tomir reaksiyalari bilan izohlanishi mumkin.

ОСОБЕННОСТИ СОВМЕЩЕННЫХ ОДОНТОЛОГИЧЕСКИХ И ОФТАЛЬМОЛОГИЧЕСКИХ ПАТОЛОГИЙ

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Аннотация

В медицинской литературе широко освещён спектр сложных патологий, поражающих зубочелюстно-альвеолярную систему и органы зрения. К таким состояниям относятся невралгические боли, иррадиирующие в глаза, вазомоторные нарушения, сопровождающиеся гиперемией конъюнктивы, изменения в секреции слюны и слёзной жидкости, а также двигательные дисфункции, влияющие на движения глаз. Стоматологические заболевания, такие как кариес, пародонтит, периостит, абсцессы, гангренозные

поражения и заболевания пародонта, часто вызывают изменения в глазах. При гранулемах и хроническом воспалении пародонта раздражаются не только окончания тройничного нерва, но и симпатические нервные волокна, что приводит к патологической рефлекторной реакции в глазах и других органах. Кроме того, очаги патологической стимуляции могут возникать после проведения стоматологического лечения зубов с удалённой пульпой, а также зубов, подвергшихся пломбированию или покрытию металлическими реставрациями. Известно также такое осложнение, как одонтогенный паралич аккомодации.

Ключевые слова: появление мельканий, темных и светлых пятен или тумана перед глазами, снижение зрения различной продолжительности в некоторых случаях можно объяснить рефлекторными сосудистыми реакциями, исходящими из зубных очагов.

Introduction. Odontogenic infection may give rise to thrombophlebitis and phlebitis of the facial venous system, frequently on the side affected by dental caries. In some cases, the infection extends to the adjacent paranasal sinuses, potentially affecting the eye [5, 6].

The etiology of inflammatory conditions such as orbital cellulitis, osteomyelitis, and subperiosteal abscesses may also be attributed to erysipelas, facial and scalp abscesses, as well as various inflammatory conditions of the dentoalveolar complex — acute and chronic periapical periodontitis, jaw root-near granulomas, cellulitis, and maxillofacial and cervical abscesses [1, 4].

Materials and methods. In infants, orbital cellulitis most commonly arises from inflammation of the maxilla. Following hematogenous osteomyelitis of the maxilla, defects in the lower orbital margin of the orbit typically develop, accompanied by cicatricial eversion of the eyelids. It is crucial to differentiate acute inflammation of the orbit from hematogenous osteomyelitis of the maxilla, and chronic osteomyelitis of the latter from dacryocystitis. Furthermore, suborbital abscesses must be distinguished from cellulitis affecting the lacrimal sac.

Malignant neoplasms of the paranasal sinuses, whether primary or secondary in origin, which have sprouted from the nasopharyngeal region, as well as tumors affecting the upper jaw, hard palate, and soft palate, can invade the orbit. Simultaneously, an ophthalmologist may detect the initial manifestations: as a consequence of compression on the optic nerve and vascular structures, stagnation phenomena occur, resulting in exophthalmos and double vision. The earliest symptom to manifest is edema of the eyelids, particularly in the morning hours.

In cases where the tumors originate from the primary sinus, persistent headaches, prominent exophthalmos along the axis, a decline in visual acuity, and congested optic discs are observed. Complex orbital and maxillofacial neoplasms may arise, with hemangiomas, lymphangiomas, and dermoids being the most prevalent. These neoplasms can manifest beneath the muscle layers of the oral floor, beneath the tongue, within the tongue itself, and in the vicinity of the nasal root.

Cleft lip and palate anomalies, along with enamel hypoplasia, may be accompanied by congenital cataracts. In the context of tetany, there is a tendency for layered cataracts to develop, accompanied by a layered distribution of enamel within the dental structure.

Results. Bilateral, often markedly pronounced exophthalmos may be caused by hypoplasia of the maxilla and orbital cavity. Craniofacial exostosis may lead to a mongoloid facial appearance, astigmatism, nystagmus, subluxation of the lens, skull deformities, open bite, paralysis of cranial nerves, and other conditions.

Behcet's disease, described by the Turkish dermatologist Behçet N., combines recurrent hypopyon-iridocyclitis, recurrent aphthous stomatitis, genital ulcers, and skin lesions into a single syndrome. The disease manifests itself suddenly in the form of attacks lasting from several weeks to several months, followed by recurrent episodes over an extended period of time, up to 25 years. Typically, there are four to five exacerbations per year, affecting both eyes, sometimes

simultaneously, but more often at different times.

The presence of all symptoms is observed in approximately one third of patients, with individual symptoms often manifesting at intervals spanning several months or even years, making early diagnosis challenging.

The precise duration of the incubation period remains undetermined. The onset of the illness is rarely marked by ocular symptoms, often beginning with vague malaise accompanied by fever, followed by the emergence of aphthous lesions on the mucosal surfaces of the mouth and tongue. Subsequently, genital ulceration occurs. Aphthous lesions are characterized by their painful nature and the appearance of small, whitish-to-yellowish patches surrounded by a variable-sized red border. These lesions frequently occur in clusters, with single instances being rare. Aphthous ulcers tend to resolve without leaving any visible trace.

Aphous stomatitis frequently coincides with exacerbations of hypopyon-iritocyclitis, with each recurrence becoming increasingly severe. Patients report a decline in visual acuity, accompanied by cloudiness in the anterior chamber, the appearance of hypopyon, hyperemia of the iris, and the formation of opacities with varying intensities in the vitreous humor. Repeated episodes lead to the development of posterior synechiae, an increase in intraocular pressure, and potentially, the progression to complicated cataracts and persistent vitreous opacity. In most cases, this process culminates in blindness.

The disease may commence with exudative chorioretinitis or neuroretinitis. Subsequently, the alterations escalate, manifesting as periarteritis accompanied by hemorrhages in the retina and the vitreous humor, retrobulbar neuritis, papillitis, and optic nerve atrophy. In some instances, the condition may be accompanied by conjunctivitis and superficial keratitis, characterized by recurrent corneal ulcers or the presence of stromal infiltrates. Other symptoms include skin lesions, tracheobronchitis, thrombophlebitis, and venous thrombosis in the lower extremities. Additionally, joint conditions such as rheumatoid arthritis, tonsillitis, or orchitis may arise, along with meningoencephalitis affecting the nervous system.

Diagnosis is based on a comprehensive evaluation of clinical manifestations.

Behçet's disease stands out from sarcoidosis, encephalitis, Crohn's disease, Reiter's syndrome, and Stevens-Johnson syndrome, among other similar clinical conditions, in terms of its distinct characteristics. There is currently no effective treatment available, nor is there a universally accepted treatment regimen.

The treatments employed, such as broad-spectrum antibiotics, sulfonamides, calcium chloride, blood transfusions, plasma infusions, immunoglobulins, corticosteroids administered subconjunctivally, retrobulbally, and internally, atropine instillations, autohemotherapy, ultrasonic therapy, and dehydration treatment, provide only temporary relief. Currently, treatment is being supplemented with the use of immunomodulatory agents. The clinical manifestations of the disease include damage to the lacrimal and salivary glands, the upper respiratory tract, and the gastrointestinal system, which occur alongside deforming polyarthritis.

The disease progresses gradually, manifesting itself in a chronic course with periods of exacerbation and remission. Patients' complaints are primarily focused on pain, dryness and burning sensations in the eyes, as well as difficulty opening their eyes in the morning and photophobia. Additionally, they report blurred vision, reduced tear production during crying, and reduced visual acuity. Upon objective examination, mild hyperemia and laxity of the eyelid conjunctiva may be observed, along with viscous discharge from the conjunctival sac in the form of fine, grayish, elastic threads. The cornea appears dull and thinned, with grayish filaments on its

surface, which may leave behind erosions after removal. Subsequently, opacity develops, accompanied by ingrown blood vessels, culminating in xerosis. However, the sensitivity of the cornea remains unchanged.

Using Schirmer's Test No. 1, a reduction in lacrimal gland function is detected, ranging from 3–5 millimeters to zero. Following the onset of ocular pathology, alterations in the oral mucous membranes and tongue become evident. Saliva assumes a viscous consistency, rendering speech, chewing, and deglutition processes challenging. Food must be accompanied by copious amounts of liquid to facilitate its passage.

Subsequently, xerostomia ensues due to a dearth of salivary secretion (sialoadenitis). Subsequently, the parotid glands enlarge, sometimes erroneously attributed to mumps. The oral mucosa and vermillion border of the lips exhibit signs of desiccation, with erosions and fissures appearing on the tongue's surface. The dryness extends to encompass the mucous membranes of the pharynx, oesophagus, stomach, larynx, trachea, and other regions, as the nasopharyngeal, buccal, gastric, tracheobronchial, and other glands become implicated in the process.

Gastritis, colitis, hoarseness, and occasionally, a dry cough manifest. Occasionally, the mucosal linings of the urethra, rectum, and anus are also affected. As a consequence of a reduction in the excretion of sweat and sebum by the skin's glands, xerosis, hyperkeratosis, and hyperpigmentation may develop, along with hair loss. Not all manifestations occur simultaneously, but xerosis of the conjunctiva, cornea, oral and nasal mucous membranes are constant features of this condition. The course is chronic, and both eyes are invariably affected. This disease falls within the category of collagenosis, which encompasses a range of autoimmune disorders.

To aid in diagnosis, a series of procedures is proposed for evaluating salivary gland functionality, including cytological analysis of secretions, sialography, salivary radiometry, scanning, and contrast X-rays of the glands, as well as comprehensive ophthalmological examination, encompassing assessment of lacrimal gland function. Differential diagnosis must be made with rheumatoid arthritis and systemic lupus erythematosus.

The treatment is symptomatic and includes vitamin therapy, administration of immunosuppressive agents, subcutaneous injections of a 0.5% solution of galantamine, local instillations of artificial tears, lysozyme, peach oil, sea buckthorn oil, and laser stimulation of the parotid salivary glands and lacrimal apparatus. This is a manifestation of Stevens-Johnson Syndrome. The characteristic features of this condition include erosive inflammation of the mucosa of most natural orifices, polymorphic skin eruptions, and a febrile state. A prominent symptom of this syndrome is damage to the mucosal tissues of the eyes, oral cavity, nasopharynx, and genitalia.

The disease, which is more prevalent among children and adolescents but can affect individuals of any age, exhibits a tendency to recur in the spring and autumn seasons. It is associated with localized infections, drug allergies, and autoimmune processes. On the mucous surfaces of the cheeks, gums, tonsils, pharynx, palate, tongue, and lips, there is a marked hyperemia and swelling, accompanied by the formation of blisters that rapidly rupture. These blisters, when merged, give rise to continuous erosions that bleed profusely. The tongue becomes enlarged and coated with a mucopurulent substance, causing severe pain, excessive salivation, difficulty in speaking and eating. Lymph nodes, particularly those located in the cervical region, become enlarged. The manifestation of conjunctival damage takes the form of catarrhine, purulent, or membranous conjunctivitis. The symptoms include swelling, hyperemia, and adhesion of the eyelids due to copious purulently bloody exudate. Catarrhal conjunctivitis resolves without

complications, leaving no long-term consequences. Purulent conjunctivitis, however, may lead to deeper complications. Due to the secondary infection, the cornea becomes involved in the inflammatory process, resulting in superficial or deep ulcers. These ulcers may lead to scarring and, in severe cases, perforation of the cornea, potentially causing partial or complete vision loss. Membranous conjunctivitis involves conjunctival necrosis, leading to scarring. This condition can result in trichiasis (abnormal hair growth on the eyelid), inversion of the eyelids, opacity of the cornea (simblepharon), and even ankyloblepharon (fusion of the eyelids). It is important to note that ocular manifestations of this condition are not limited solely to conjunctivitis. Irritation, episcleritis (inflammation of the outer layer of the sclera), dacryocystitis (inflammation of the lacrimal sac), and even panophthalmitis (inflammation affecting all structures of the eye) have also been reported in some cases.

The treatment regimen involves a combination of broad-spectrum antibiotics, sulfonamide medications, vitamins, corticosteroids, antihistamines, blood transfusions, and immune serum. For local application, corticosteroid eye drops and subconjunctival injections are employed, along with vitamin eye drops for cases of dryness, which may require the use of artificial tears. Surgical intervention is recommended in cases of trichiasis, eyelid inversion, and xerosis.

Nasociliary syndrome, also known as Charlin's syndrome, is a complex set of symptoms resulting from neuralgia of the nasociliary nerve, which is the largest branch of the ophthalmic division of the trigeminal cranial nerve. The long ciliary nerves emanating from this branch extend to the eyeball. When the nasociliary nerve becomes irritated within the area it innervates, there occurs a change in sensory perception (pain), disturbance in secretion (excessive lacrimation or increased secretory activity of the nasal mucous membrane), and trophic disturbances (in the skin and cornea).

Pterygopalatine ganglion syndrome, also known as Slyder's syndrome, is a constellation of symptoms associated with pterygopalatine neuralgia. The pterygopalatine ganglion is a structure of the parasympathetic nervous system that comprises multipolar cells with three roots: sensory, parasympathetic, and sympathetic. Its branches innervate various structures, including the lacrimal gland, mucous membranes of the palate and nasal cavity, posterior cells of the ethmoidal bone, and sinuses of the maxilla and main bone. At the onset of the illness, the patient experiences acute, piercing pains in the region of the nasal base, surrounding and behind the eyes, in the eyes themselves, in the upper and lower jaws, and in the teeth. The pain radiates to the temples, ears, neck, shoulders, forearms, and hands. The most severe pain is felt in the orbital area, nasal root, and mastoid process.

The duration of the pain ranges from a few hours or days to several weeks. During an attack, the patient may also experience a burning sensation in the nose, bouts of sneezing, runny nose, excessive tearing, drooling, dizziness, nausea. In some cases, asthma-like episodes and a distortion of taste occur.

From the side of the eyes, there is marked photophobia, spasm of the eyelids, tearing, swelling of the upper eyelids, conjunctival congestion, dilation or constriction of the pupil, and sometimes a transient increase in intraocular pressure.

This condition can persist for an extended period, sometimes lasting for months or even years. During the intercostal interval, there is often a dull ache in the maxillary region, the nasopalatine region, and the orbital region. There may also be a swelling of the afflicted half of the face.

Pterygopalatine neuralgia is a condition associated with paranasal sinus and oral cavity

perifocal infection, as well as with various head infections, such as purulent otitis and cerebral arachnoiditis. The condition may also be caused by jaw-related tumors. Unlike nasociliary neuralgia, pterygopalatine neuralgia does not result in anatomical changes in the anterior segment of the eyeball. Instead, increased sensitivity of the nasal mucosa occurs in the posterior portion of the nasal cavity.

It is important to differentiate this condition from other types of facial neuralgia. Treatment typically involves administration of pain relievers and decongestants, as well as corticosteroids and anti-infective medications. Ganglioblockers and anticholinergics may also be prescribed, along with physiotherapy procedures and biogenic stimulants.

Ciliary ganglion syndrome, also known as Hageman-Pochtman syndrome, is a condition characterized by inflammation of the ciliary ganglion, a small structure located behind the eyeball, between the beginning of the external rectus muscle and the optic nerve. This ganglion is connected to the eye by four to six short ciliary nerves. Symptoms of the disease typically manifest as sudden pain in the head, particularly in the depth of the orbit. The pain can radiate to the jaw and teeth, and may intensify with movement of the eyeball or pressure on it. The pain often extends to the corresponding side of the head, and its duration can range from a few days to several weeks. Unilateral mydriasis, characterized by the symmetrical and round shape of the pupils, is accompanied by a lack of pupillary responses to light and accommodation, as well as a decrease in corneal sensitivity. Additionally, there is an edema of the corneal epithelium and a temporary increase in intraocular pressure. In rare cases, optic neuritis may develop. This condition typically affects one side and resolves within 2–3 days, although in some instances it may persist for up to a week or longer. The paralysis or paresis of accommodation can persist for an extended period of time, frequently serving as the sole indication of the underlying process. Recurrences are also possible. The causative factors contributing to the development of this condition can include inflammatory processes affecting the paranasal sinuses and teeth, as well as infectious diseases, traumatic injuries, or orbital contusions.

Treatment strategies may involve rehabilitation of the paranasal sinus region, oral cavity, application of ganglion blockades, the use of corticosteroids and vitamin therapy, administration of diphenhydramine and novocaine retrobulbars, as well as local pain relievers and vitamin eye drops. A peculiar feature of this condition is the occurrence of paradoxical eyelid movements, characterized by unilateral ptosis, which disappears when the patient opens their mouth and moves their jaw in a direction opposite to the direction of the ptosis. As the patient continues to open their mouth, the eye may appear wider open. Conversely, during chewing movements, the ptosis tends to decrease. This syndrome can manifest either congenitally or acquire later in life, and its severity may diminish with advancing age. In the context of acquired diseases, paradoxical movements of the eyelid may arise following an injury, dental extraction, damage to the facial nerve, concussion, encephalitis or psychological trauma. It is hypothesized that there exists an aberrant connection between the trigeminal and oculomotor nerves, or the nuclei associated with these nerves.

Occasionally, this syndrome is attributed to a disruption in corticobulbar pathways. Concomitantly, alterations in the visual system and the body at large can be observed, such as paralytic strabismus on the side of ptosis, epileptic seizures with rare convulsions, and other manifestations. The color of the lesions in childhood is initially pink, subsequently evolving to bluish-red hues. Angioma-tosis on the face often extends to the skin surrounding the eyes, conjunctiva, and sclera. In some cases, angiomas may develop within the choroid, potentially

leading to retinal detachment in alignment with the location of the vascular lesion. Typically, this condition is accompanied by the development of glaucoma. The symptoms of brain damage may include intellectual disability, clonic convulsions, epileptic-like seizures, recurrent hemiplegia or hemiparesis. There may also be the presence of hemangiomas in internal organs. The treatment options include X-ray therapy, sclerotherapy, surgical removal of cutaneous angiomas and management of glaucoma.

Discussion. The prognosis for the patient's life may be grim due to severe neurological complications. Craniofacial dysostosis, also known as Crouzon's disease, is characterized by bilateral exophthalmos, which is caused by the underdevelopment of the maxilla and orbit. This condition is accompanied by divergent strabismus, expansion of the root area of the nose, and an increased distance between the eyes. On the fundus, there is stagnation of the discs of the optic nerves, leading to secondary atrophy due to either a narrowing of the bony canal or an increase in intracranial pressure resulting from the synostosis of the majority of cranial sutures. Crouzon disease is a rare congenital disorder characterized by a distinctive facial appearance, including Mongoloid-type eye slits, astigmatism, nystagmus, and congenital subluxation of the lens. Other symptoms may include hydrophthalmos and cataracts.

The disease is associated with cranial abnormalities, such as a beak-like nose and a short upper lip, as well as impaired sense of smell, hearing, and moderate mental retardation. In some cases, Crouzon syndrome is accompanied by open bite and syndactyly of hands and feet. Crouzon disease is considered a familial hereditary anomaly of the cranial region. Treatment typically involves surgical intervention, such as bone grafting. Mandibulofacial dysostosis, also known as Franceschetti's disease, is a syndromic condition characterized by various maxillofacial abnormalities that occur in various combinations and are inherited in a familial manner. The ocular manifestations of this condition include oblique «anti-Mongoloid» ocular slits, bilateral omission of the external corner of the eye, eyelid colobomas, epibulbar dermoids, paresis of the oculomotor muscles, and rarely, microphthalmos. Additionally, congenital cataracts and colobomas of the vascular tract may be present, along with optic nerve abnormalities.

In terms of the maxillofacial system, hypoplasia of facial bones is evident, resulting in significant facial asymmetry and severe underdevelopment of teeth. Frequent features include ear aplasia, hyperplasia of the frontal sinus, facial bone splitting, and skeletal deformities. Occasionally, there may be an enlargement of the tongue, absence of the parotid gland, hydrocephalus, and heart abnormalities.

Alongside the typical presentations of the syndrome, atypical forms exist, characterized by the presence of only a subset of symptoms. Congenital cataracts, colobomas of the iris and choroid, strabismus, dermoid cysts in the vicinity of the limb, as well as severe refractive errors, are possible manifestations. The common features include a broad nasal bridge, hypoplasia of the maxilla, cleft palate, oligodontia, a conical shape to the anterior teeth, dysplasia of dental enamel, hydrocephalus, and malformations in the heart, kidneys, and spine. In some cases, short stature is attributed to growth hormone deficiency. The type of inheritance is autosomal dominant.

Bloch–Sulzberger Syndrome. The condition is characterized by a constellation of ocular symptoms, including strabismus, nystagmus, ptosis, microphthalmos, cataracts, and various forms of keratitis. Additionally, there is atrophy of the optic nerve and retrolental fibroplasia, often accompanied by retinal detachment.

Neurological manifestations include delayed eruption of deciduous and permanent dentition, hypoplasia of jaws and alveolar processes, and cleft lip and palate. In severe cases, there

may be an incomplete number of teeth or their absence.

Central nervous system involvement manifests as micro- and hydrocephalus, epileptic seizures, tetraplegia or paraplegia, delay in mental development, and mental retardation. There are also potential anomalies such as talipes equinovarus, syndactyly, dysplasia of the hip joint, malformations of the auricles and nails, cardiopathy of congenital origin, and delayed speech acquisition. This syndrome is categorized as hereditary disorders of the ectoderm. There is currently no effective treatment available.

Conclusions. Torg–Winchester syndrome is a rare congenital disorder characterized by a severe pathology of the facial bones and joints, including the gross pathology of the skull and facial skeleton. It is also associated with thinning and opacification of the cornea, as well as dwarfism and the «dissolution» of short carpal and tarsal bones. These changes are caused by mutations in the MMP2 gene, and surgical treatment of corneal pathology may be indicated.

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