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Eye Symptoms in Parathyroid Disorders: Clinical Presentation and Research Perspectives

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Abstract

Introduction and purpose: Endocrine disorders often affect multiple organs, frequently presenting with characteristic ophthalmic symptoms. Ocular abnormalities occur in both hyperparathyroidism and hypoparathyroidism, potentially affecting nearly all parts of the eyeball and orbital structures. This study aims to summarize available literature and recent findings on ophthalmic manifestations linked to parathyroid disorders.

Material and methods: A comprehensive literature review was conducted using keywords in the PubMed database, the Via Medica journal database, the Polish Society of Endocrinology guidelines, and the Polish Ophthalmological Society guidelines.

State of knowledge: Ophthalmic manifestations are common in parathyroid disorders. Hypoparathyroidism often causes cataracts, periorbital tetany, blepharospasm, and optic nerve issues, while congenital cases may involve microphthalmia and corneal opacities. Hyperparathyroidism leads to calcium deposition, affecting retinal arterioles, sclera, eyelids, and cornea, with reports of scleritis and orbital brown tumors.

Summary: Recognizing ocular signs of parathyroid diseases aids in diagnosing endocrine disorders and highlights the need for ophthalmological consultation to improve early detection and treatment.

Keywords: "ocular", “parathyroid", "hyperparathyroidism", "hypoparathyroidism",

Introduction

Endocrine disorders affect multiple organs and present with a wide range of symptoms. Pathological changes involving ocular and orbital tissues are also frequently observed in various endocrine diseases [1]. In parathyroid disorders, numerous ophthalmic manifestations have been reported, resulting from functional and structural abnormalities of the eye. The characteristic ocular abnormalities differ between hyperparathyroidism and hypoparathyroidism [2] [3].

The eye is a sensory organ consisting of the eyeball and accessory structures, including the extraocular muscles, conjunctiva, lacrimal apparatus, eyelids, and eyebrows. It is situated within the orbit, a bony cavity that protects its delicate structures [4]. The eyeball consists of three primary layers. The outermost layer, known as the fibrous tunic, comprises the transparent cornea and the opaque sclera. The middle layer known as the vascular tunic, includes the choroid (posterior part), ciliary body (intermediate part), and iris (anterior part). The innermost layer is formed by the retina, which lines the interior of the eyeball. The eye contains three chambers: the anterior chamber, posterior chamber, and vitreous chamber. The anterior chamber is bordered anteriorly by the cornea and posteriorly by the iris and lens. The posterior chamber is a space surrounding the lens, bounded anteriorly by the iris, laterally by the ciliary body, and posteriorly by the vitreous body. Both chambers are filled with a transparent aqueous humor. The vitreous chamber, located between the posterior surface of the lens and the retina, is filled with the vitreous body. The optic nerve is responsible for transmitting visual impulses from the retina to the cerebral cortex, enabling visual perception [4] [5]. In the clinical division of the eyeball, two sections are distinguished: the anterior segment, which includes the cornea, anterior chamber, iris, sclera, ciliary body and lens; and the posterior segment, which includes the vitreous body, retina, ocular part of the optic nerve and choroid.

Purpose

This article reviews the current knowledge of ocular manifestations that may indicate parathyroid disease. The objective of the present study is to summarise the data available in the literature, as well as recent findings and research on ocular symptoms in both hyperparathyroidism and hypoparathyroidism. It also aims to highlight the links between parathyroid disease and the visual system.

Material and methods

A review of the extant literature was conducted using the PubMed database, the Via Medica journal database, the guidelines of the Polish Society of Endocrinology and the guidelines of the Polish Society of Ophthalmology. The following keywords were used to retrieve articles: "eye," "ocular," "ophthalmopathy," "retinopathy," "cataract," "parathyroid," "hyperparathyroidism," "hypoparathyroidism," and "parathormone." The search provided 740 results, of which 31 articles were included in the study after excluding those that did not meet the authors' criteria**.**

State of knowledge

Hypoparathyroidism

Hypoparathyroidism is an endocrine disorder characterised by a chronic deficiency or low serum levels of parathyroid hormone (PTH). An atypical form of this condition is pseudohypoparathyroidism, in which PTH levels are elevated, but the symptoms of PTH deficiency arise due to target tissue resistance caused by a defect in the PTH receptor [6]. Parathyroid hormone (PTH) plays a crucial role in regulating calcium and phosphate homeostasis in the body. It exerts a direct influence on bone resorption and calcium and phosphate transport in the kidneys, and an indirect influence on calcium absorption in the gastrointestinal tract through enhanced production of the active form of vitamin D [1,25(OH)D] [2]. A deficiency or complete absence of PTH results in elevated serum phosphate levels and hypocalcemia, defined as low serum calcium levels corrected for albumin concentration or low ionized calcium levels [1]. The diagnosis of hypoparathyroidism requires at least two confirmed instances of hypocalcemia, accompanied by low or undetectable parathyroid hormone (PTH) levels and a normal serum magnesium concentration [7]. Hypoparathyroidism is a rare disease with an estimated prevalence ranging from 6.4 to 37 per 100,000 person-years, while its incidence varies between 0.8 and 2.3 cases per 100,000 person-years [6]. The most prevalent etiological factor for hypoparathyroidism is surgical intervention on the neck region, accounting for approximately 75% of cases observed in adults [6][8]. The remaining 25% of hypoparathyroidism cases are attributed to autoimmune disorders, such as autoimmune polyglandular syndrome type 1 (APS-1), genetic disorders (e.g., DiGeorge syndrome), exposure to ionising radiation, infiltrative diseases (e.g.,

amyloidosis), and other rarer causes [8]. Throughout a patient's life, hypoparathyroidism has the potential to result in a multitude of complications, with the capacity to affect virtually all organs within the body [6]. Hypocalcemia is responsible for the majority of symptoms associated with hypoparathyroidism. A decrease in calcium levels can lead to muscle cramps, paresthesia, and life-threatening complications such as cardiac arrhythmias, laryngeal and bronchial spasms, and seizures in cases of acute calcium deficiency. Chronic hypocalcemia and hyperphosphatemia may result in soft tissue calcifications, primarily affecting the brain and kidneys. Calcifications may also be present in the joints, skin, blood vessels, eyes, and other organs [6]. In bones, chronic PTH deficiency slows down bone remodeling processes, leading to impaired bone microarchitecture, reduced mineral density, and decreased skeletal strength [7].

Eye and hypoparathyroidism

In the course of hypoparathyroidism, abnormalities may affect both the anterior and posterior segments of the eyeball, as well as the accessory ocular structures. The observed ocular changes, categorized by eye segment, are presented in Table 2. [2] [9]. In severe cases of hypoparathyroidism, periorbital tetany, eyelid spasms, optic neuritis, and optic disc edema have been reported [3]. In addition, congenital hypoparathyroidism has been observed to be associated with microphthalmia, iris coloboma, and corneal clouding [5]. In a study analysing 118 cases of idiopathic hypoparathyroidism, cataracts were observed in 58% of patients, optic disc edema in 11%, corneal changes in 10%, photophobia and eyelid spasms in 8%, and eyebrow loss in 7% of patients [10].

Table 1. Ocular changes in hypoparathyroidism

Accessory organs	**Eyeball**	
	Anterior segment	**Posterior segment**
Blepharospasm Periorbital tetany Chronic keratocnjuncitivitis	Colobomata Corneal opacities Cataract formation	Optic neuritis Papilledema

Cataracts and hypoparathyroidism

Cataract is the most frequently documented ocular complication of hypoparathyroidism. A recently published cross-sectional study analysing patients with chronic hypoparathyroidism found cataracts in 62% of the participants [11]. In earlier research, Goswami et al. reported the presence of cataracts in 51% of patients with non-surgical hypoparathyroidism, with its occurrence correlating with the duration of symptoms and the presence of basal ganglia

calcifications [12]. In a Scottish cohort study involving 280 cases of chronic hypoparathyroidism, it was demonstrated that all patients, regardless of disease etiology, had an increased risk of developing cataracts (HR 2.10 (1.30–3.39)) [13]. In Danish studies conducted by Underbjerg et al., no increased risk of cataracts was observed in patients with postoperative hypoparathyroidism (HR 1.17, 95% CI 0.66–2.09). Additionally, the study revealed no statistically significant difference in the age of onset of vision impairment due to cataracts between the study group (64.3 ± 2.3 years) and the control group (67.2 ± 1.7 years). Among patients with non-surgical hypoparathyroidism, the risk of developing cataracts was found to be four times higher compared to healthy controls (HR 4.21; 95% CI, 2.13–8.34). Additionally, cataracts were diagnosed at a younger age, with a mean age of 53 years (95% CI, 45–61) in the study group, compared to 60 years (95% CI, 49–73) in the control group [14][15]. Similar findings were reported by Saha et al., who demonstrated that patients with idiopathic hypoparathyroidism underwent cataract surgery at a younger age compared to endocrinologically unaffected individuals (34 ± 16 vs. 58 ± 11 years; $P < 0.001$). In addition, a higher incidence of posterior capsule opacification requiring subsequent laser capsulotomy was observed in the study group [16].

The mechanisms leading to the development of cataracts in the course of hypoparathyroidism are not well understood [1, 2, 13]. Risk factors for lens opacification include idiopathic hypoparathyroidism and prolonged disease duration [6].

Hyperparathyroidism

Hyperparathyroidism is an endocrine disorder characterised by excessive secretion of parathyroid hormone (PTH) by the parathyroid glands. It is classified into three types: primary, secondary, and tertiary.

In primary hyperparathyroidism (PHPT), excessive PTH production is the consequence of the absence or reduced sensitivity of parathyroid cells to the inhibitory effect of hypercalcemia. The causes include a single adenoma (approximately 85% of cases), multiple adenomas or parathyroid hyperplasia (around 15%), and parathyroid carcinoma (around 1%). In approximately 5% of cases, PHPT has a genetic basis and is associated with multiple endocrine neoplasia (MEN) syndromes, such as MEN type 1 (MEN1) or type 2A (MEN2A) [17]. Primary hyperparathyroidism is a condition that typically manifests in adults and is observed to be 3 to 4 times more prevalent in women than in men [17][18]. The prevalence of this disorder varies across different regions of the world and depends on multiple factors, with

the availability of serum calcium and parathyroid hormone (PTH) measurement in screening programs playing a key role. In the United States, the morbidity rate of primary hyperparathyroidism is 233 per 100,000 in women and 85 per 100,000 in men [19]. The course of the disease may be asymptomatic or symptomatic and depends on the duration of excessive parathyroid hormone (PTH) secretion and the severity of hypercalcemia [17]. The symptoms of primary hyperparathyroidism include bone and joint pain, fractures secondary to osteoporosis or osteitis fibrosa cystica, and gastrointestinal disturbances (constipation, nausea, peptic ulcers, pancreatitis, cholelithiasis). Central nervous system manifestations include depression, cognitive and memory impairment, and seizures. Neuromuscular symptoms may present as weakness and hypotonia, while renal complications include nephrolithiasis, polyuria, and secondary polydipsia [18]. Secondary hyperparathyroidism is characterised by excessive parathyroid hormone (PTH) secretion in response to chronic hypocalcemia [18]. It is most commonly associated with chronic kidney disease. Other causes include acute kidney injury, conditions leading to chronic hypocalcemia, such as malabsorption syndromes, and vitamin D deficiency. Additionally, it has been observed to be a complication of bariatric surgery [17]. The clinical manifestations of secondary hyperparathyroidism are primarily related to chronic kidney failure. Bone changes and other effects of excessive PTH secretion are generally less severe than in primary hyperparathyroidism. A characteristic feature of secondary hyperparathyroidism is the process of calciphylaxis, in which vascular wall calcification occurs. These changes are most commonly found in the dermis and subcutaneous adipose tissue but may also affect visceral organs and skeletal muscles, leading to ischemic damage [20].

Tertiary hyperparathyroidism is caused by autonomous excessive secretion of parathyroid hormone (PTH) in individuals with long-standing secondary hyperparathyroidism [18]. The principal consequence of this hormonal disorder is the development of hypercalcemia. It typically occurs in patients with chronic kidney disease undergoing dialysis, with the primary cause being ineffective treatment of secondary hyperparathyroidism [17].

Eye and hyperparathyroidism

Hyperparathyroidism, like hypoparathyroidism, can lead to alterations affecting nearly every part of the eyeball. The ophthalmic changes reported to date are summarised in Table 3. [2][3]. Chronic hypercalcemia leads to excessive calcium deposition in the eyeball, resulting in retinal arteriole calcification, choroidal calcification, calcifications along the eyelid margins,

and band keratopathy of the cornea [4][21]. Furthermore, cases of recurrent conjunctivitis that are unresponsive to standard treatment have been documented [3]. Scleritis, manifesting as ocular redness, has also been described as a symptom associated with hyperparathyroidism [22]. A relatively recently identified ocular condition observed in the course of hyperparathyroidism is sclerochoroidal calcification [23]. In addition to the changes affecting the eyeball, cases of brown tumors of the craniofacial skeleton involving the orbit have been reported in the course of hyperparathyroidism [24][25].

Table 2. Ocular changes in hyperparathyroidism

Accessory organs	**Eyeball**	
	Anterior segment	**Posterior segment**
Recurrent conjunctivitis Calcification of eyelids	Band keratopathy Scleritis	Choroidal calcification Retinal calcification

Band keratopathy

Band keratopathy is a chronic degenerative disease characterised by the formation of whitish or greyish opacities on the surface of the cornea. The underlying mechanism of corneal opacity development involves the accumulation of calcium hydroxyapatite crystals within the superficial layers of the cornea [26][27]. Research has indicated a correlation between this process and chronic hypercalcemia [28][29]. The association between corneal calcium deposition and hyperparathyroidism has been observed in several studies [29][30]. A recently published retrospective case-control study found that patients with hyperparathyroidism have a higher risk of developing band keratopathy compared to the control group (adjusted OR, 11.287; 95% CI, 5.461–23.33; $P < 0.001$) [31]. In addition, there are case reports of patients in whom the presence of corneal calcium deposits led to the diagnosis of hyperparathyroidism [32] [33].

Sclerochoroidal calcification

Sclerochoroidal calcification (SCC) is a condition that is characterised by the deposition of calcium in the sclera, with secondary involvement of the choroid. These lesions exhibit a characteristic appearance, presenting as poorly demarcated, yellowish-white, elevated changes in the fundus of the eye [23]. These lesions are typically asymptomatic and are often detected incidentally during fundoscopic examination or orbital CT imaging [20].

However, a recently published case report described sclerochoroidal calcification in a patient with long-standing primary hyperparathyroidism. Progressive severe vision loss occurred in this patient due to dural calcification along the optic nerves [34]. Choroidal neovascularisation has also been reported in a patient with sclerochoroidal calcification secondary to hyperparathyroidism [35]. Shields et al. identified hyperparathyroidism in 27% of examined patients with sclerochoroidal calcification [36].

Conclusions

Parathyroid disorders can manifest with ocular symptoms and are frequently associated with ophthalmic complications. The recognition of the relationship between various ocular manifestations and parathyroid dysfunction provides valuable diagnostic insight for both ophthalmologists and primary care physicians, highlighting the need for further evaluation of potential hormonal imbalances. This knowledge is also essential for endocrinologists, enabling them to incorporate timely ophthalmologic consultations into the treatment plan. A well-directed diagnostic approach can facilitate earlier diagnosis and help reduce the risk of complications.

Disclosures

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