Lacrimal Gland Pathologies from an Anatomical Perspective

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Abstract

Most of the patients in our daily practice have one or more ocular surface disorders including conjunctivitis, keratitis, dry eye disease, meibomian gland dysfunction, contact lens related symptoms, refractive errors, computer vision syndrome. Lacrimal gland has an important role in all above mentioned pathologies due to its major secretory product. An anatomical and physiological knowledge about lacrimal gland is a must in understanding basic and common ophthalmological cases. In this paper it is aimed to explain the lacrimal gland diseases from an anatomical perspective.

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Introduction

Lacrimal gland is pinkish-gray, lobulated serous gland. The aqueous component of tear film is mainly provided by lacrimal gland (1). In the first trimester of pregnancy and at 19-21 mm stage of embryologic development, it appears as epithelial buddings from superolateral conjunctival fornix ectoderm. The mesenchymal condensations around these clusters than turn in to secretory components. These early epithelial buds with secretory components form the orbital lobe of lacrimal gland. At the 40-60 mm stage of fetal development another group of epithelial stalks appears which form the palpebral lobe of the lacrimal gland later. Development of lacrimal gland continues for 3-4 years after birth (2, 3). Congenital alacrima is characterized by deficient lacrimation from birth as a result of embryological development disorder and lacrimal gland hypoplasia (4). Congenital alacrima can also be seen as a component of some syndromes such as triple A syndrome in which achalasia and addison disease accompanies alacrima. Besides dry eye, mental retardation, autonomic dysfunction, deafness and hyperkeratosis on palms of hands and soles of feet are additional symptoms of this syndrome (5).

Lacrimal gland is situated in the superotemporal orbit. It measures about 20 mm long, by 12 mm wide and by 5 mm thick (6). In normal human population, by increasing age periductal fibrosis, paraductal blood vessel loss, aciner cell atrophy and interacinar fibrosis causes age related primary lacrimal gland deficiency (7). It is anatomically divided in to two portions as orbital and palpebral lobe by the lateral horn of levator aponeurosis. But this separation is incomplete and around lateral end of aponeurosis, lobes show continuity (Figure 1, Figure 2). The larger orbital
lobe is about the 60-70 % of total mass of lacrimal gland and situated anterior to levator aponeurosis, posterior to orbital septum and preaponeurotic fat pad. The smaller palpebral lobe is about the 30-40 % of total mass of lacrimal gland, located posterior to levator aponeurosis and anterior to palpebral conjunctiva (6-9). In upper eyelid surgical procedures lacrimal gland must be differentiated from preaponeurotic fat pads, otherwise surgical removal of lacrimal gland causes severe dry eye symptoms. This differentiation can be easily made by color difference; fat pads appear yellowish while lacrimal tissue appears pinkish (10).

Two to six secretory ductules of orbital lobe pass through the palpebral lobe at where ductules of palpebral lobe are joined and finally a total number of 6-12 tubules empty in to superolateral conjunctival fornix approximately 4-5 mm above the tarsus (11) (Figure 1,2). Therefore any damage to palpebral lobe may cause similar results as any damage to whole lacrimal gland. Upper conjunctival fornix damage and obstruction of lacrimal gland ducts by trachoma, cicatricial pemphigoid, mucous membrane pemphygoid, erythema multiforme, trauma, chemical burns and thermal burns lead to secondary lacrimal gland deficiency and aqueous deficient dry eye syndrome (12-16).

In addition to supporting function of relation between lacrimal gland pseudocapsule and periorbita of frontal bone, Whitnall's ligament and lateral horn of levator aponeurosis are other major supporters stabilizing the gland in shallow lacrimal fossa. Lacrimal gland prolapsus is not an uncommon result of upper eyelid surgical procedures when Whitnall's ligament disrupted and it should be kept in mind that refixation of lacrimal gland should be needed to avoid postoperative cosmetic problems and dry eye (17).

Lacrimal gland is composed of lobules separated from each other by loose fibrovascular connective tissue (Figure 3). This interlobular septa is the extension of lacrimal gland pseudocapsule, which is connected to periorbita as mentioned earlier. Of lobules 80% are secretory acini which are composed of an inner layer of columnar or pyramidal shaped epithelium around a central lumen and surrounded by a basal layer of myoepithelial cells (18, 19). Contraction of myoepithelial cells aid in secretion. Columnar epithelial cells of secretory acini have well developed endoplasmic reticulum, golgi complex, moderate numbers of mitochondria, free ribosomes, lipid droplets and vacuoles. Of lobules lacrimal ducts forms the 10-12 % and composed of one or two layers of cuboidal cells. The other cellular units of lacrimal gland are lymphocytes, plasma cells, mast cells and macrophages (18, 19).

The most common space occupying lesions of lacrimal gland are cysts (dacryops) which are derived from cystic dilatation of ductal epithelial cells and dermoid cysyts (20). The most common benign primary lacrimal gland tumor of lacrimal gland is pleomorphic adenoma (benign mixed cell tumor). This benign tumor is consisted of myoepithelial and cellular components as well as ductal epithelium. Very rarely myoepithelioma may appear as another benign tumor mimicking pleomorphic adenoma (21, 22). The most common epithelial origin malignant tumor is adenoid cystic carcinoma. Malign mixed cell tumor, adenocarcinoma, squamous cell carcinoma are other types of lacrimal gland malignant tumors (21, 22).

Besides aqueous secretion, lacrimal gland also secretes protein and electrolytes. The mechanism of this secretions are under neural and hormonal control. Acetylcholine and vasoactive intestinal peptide are parasympathetic and norepinephryne is the sympathetic neurotransmitter of lacrimal gland (23). Many hormones take role in regulation of lacrimal gland functions. By the stimulation of parasympathetic nerves, acetylcholine released which activate M3 muscarinic receptors located at acinar basal and lateral membranes result in increase in intracellular concentration of calcium in activation of several protein kinase C. Protein secretion is stimulated by these biochemical

![Figure 3. Ultrastructural anatomy of a lacrimal lobule.](image)
pathways. Norepinephrine causes an increase in protein secretion by the ability of binding both alpha and β-adrenergic receptors (24-27). Other chemical mediators that regulate secretion of lacrimal gland include alpha melanosome stimulating hormone, adrenocorticotropic hormone, epidermal growth factor, substance P, calcitonin gene and androgens. Ductal epithelial cells modify the secretory product of acini before reaching to conjunctival fornix and ocular surface of final composition (28-30).

The final electrolyte composition of lacrimal gland secretion is similar to plasma except for lower level of Na and higher levels of K and Cl. It contains many kind of proteins including lysozyme, lactoferrin, secretory IgA, epidermal growth factors, a lacrimal gland specific protein lacrytin, surfactan proteins A-D (31-33).

Lacrimal gland receives arterial supply from lacrimal artery, a branch of the ophthalmic artery, with contributions from the infraorbital artery and sometimes from the recurrent menengial artery. Lacrimal artery enters its posterior border pass through the gland and reaches to conjunctiva where it takes the name lateral palpebral artery which anostomoses with medial palpebral artery over the eyelids. Venous drainage of lacrimal gland is in to the superior ophthalmic vein then to cavernous sinus. The lymphatic drainage of lacrimal gland joins with conjunctival lymphatics to drain in to superficial parotid lymph nodes.

Lacrimal gland has both sensorial and autonomic innervations (Figure 4). Lacrimal nerve is the smallest branch of ophthalmic nerve. Like as lacrimal artery lacrimal nerve also pass through the gland reaches to adjacent conjunctiva and eyelid carrying sensorial innervation. Preganglionic parasympathetic secretomotor fibers originate in the lacrimal nucleus of brainstem, exit from the ventrolateral portion as the nervus intermedius in company with motor division of facial nerve and enters the auditory canal to reach geniculate ganglion (Figure 4). The greater superficial petrosal nerve arises from the preganglionic parasympathetic fibers of geniculate ganglion. Greater superficial Petrosal nerve unites with the deep petrosal nerve carrying postganglionic sympathetic fibers and together they form the nerve of pterygoid canal (vidian nerve). Vidian nerve reaches to pterygopalatine ganglion (34, 35) (Figure 4).

Pterygopalatine ganglion is the largest parasympathetic ganglion located in pterygopalatine fossa, suspended by nerve roots from the maxillary nerve. Here parasympathetic nerve fibers synapse and continues as postganglionic parasympathetic nerve whereas sympathetic nerve fibers do not. Postganglionic parasympathetic nerve fibers leave the pterygopalatine ganglion through the branches of zygomatic nerve and finally reach to lacrimal nerve and lacrimal gland with communicating branches (34, 35).

The postganglionic sympathetic nerve fibers arising from superior cervical ganglion do not synapse in pterygopalatine and uses the same way as postganglionic parasympathetic nerve fibers to reach lacrimal gland (Figure 4). Disruption in any part of autonomic system of lacrimal gland results in deterioration of lacrimal gland fluid and electrolyte secretion. Familial dysautonomia is a rare inherited autonomic and sensorial nervous system malfunction characterized by insensitivity to pain, reduced lacrimal gland secretion, labile blood pressure, emotional instability, pupillary function abnormality and many other symptoms varying person to person (36).
A decrease in corneal sensitivity (hypoesthesia, hypesthesia) causes less stimulation of lacrimal gland and reduced reflex tear production. There are many factors leading to hypesthesia. Corneal sensitivity decreases steadily with age like all other neural transmissions. Topically used proparacaine, non steroidal anti inflammatory agent’s diclofenac, ketorolac, timolol maleate, betaxolol are some commonly prescribed agents by physicians which may cause reduced corneal sensitivity (37-44).

Contact lens usage is another common mechanism of lacrimal gland hyposecretion due to reduced corneal sensitivity. Diabetes mellitus may cause lacrimal gland hyposecretion by two ways; reduced corneal sensitivity and microvasculature damage of lacrimal gland. Herpes simplex keratitis, herpes zoster ophthalmicus, refractive surgery, extracapsular cataract surgery, keratoplasty are other common causes of corneal hypoesthesia and diminished reflex tears secretion (45-50).

A cerebellopontin angle tumor or any other space occupying lesion pressing on trigeminal nerve root may cause unilateral corneal hypoesthesia as a first sign. The afferent pathway of reflex lacrimation pathway may be affected as the complication of trigeminal neuralgia surgery (51-55).

Bilateral corneal hypoesthesia is commonly associated with diabetes, amyloidosis, and vitamin A deficiency.

Nervus intermedius may be damaged in a neurosurgery procedure usually for brainstem pathology such as vestibular schwannoma (56-60).

Secondary lacrimal gland deficiency is the result of destructive infiltration of lacrimal gland by several diseases including lymphoma, sarcoidosis, AIDS, graft versus host disease, hemochromatosis, neurofibroma, tumors and Sjögren syndrome (61-64).

Patients admitting with the symptoms of aqueous deficient dry eye disease make one of the largest groups of our daily practices. Every ophthalmology expert should know the major, microstructural and neurophysiological anatomy of lacrimal gland to handle with the problems of different etiological factors. This paper summarizes the anatomy of lacrimal gland for the purpose of best systematic clinical approach.

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