

## **A Comprehensive Review of Keratinous Cysts: Etiology, Diagnosis, and Management**

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**Abstract.** Keratinous cysts, predominantly referred to as epidermoid cysts (ECs) or epidermal inclusion cysts (EICs), represent the most prevalent subtype of cutaneous cysts. These benign, slow-growing lesions are defined by a pathological cavity situated within the dermis or subcutis, lined by stratified squamous epithelium and filled with macerated keratin and lipid-rich debris. While often asymptomatic, their clinical significance arises from a high frequency of complications, including inflammation, infection, and rupture, which prompt medical consultation. Furthermore, their presentation can mimic a wide range of other soft tissue tumors, necessitating accurate diagnosis. This paper provides a exhaustive, systematic review of keratinous cysts, delving into their historical context, precise nomenclature, and epidemiological patterns. It elaborates on the multifactorial etiologies and molecular pathways underpinning their pathogenesis. The clinical and histopathological features are described in detail, alongside advanced diagnostic imaging characteristics. A critical analysis of the differential diagnosis is presented, followed by a comprehensive discussion on management strategies, from conservative approaches to sophisticated surgical techniques aimed at minimizing recurrence and optimizing cosmetic outcomes. The paper also explores rare associations and future research directions, consolidating current evidence to serve as a definitive reference for dermatologists, surgeons, and primary care physicians. condition.

### Highlights:

1. Keratinous cysts, including epidermoid and epidermal inclusion cysts, are the most common cutaneous cysts with significant clinical impact when complicated.
2. Accurate diagnosis requires distinguishing them from other cystic and tumor-like lesions using clinical, histological, and imaging features.

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3. Management ranges from observation and intralesional corticosteroids to complete surgical excision, with prognosis generally excellent after definitive treatment.

**Keywords:** Epidermoid Cyst, Epidermal Inclusion Cyst, Keratinous Cyst, Pilar Cyst, Surgical Excision, Ruptured Cyst, Minimal Excision Technique.

## Introduction

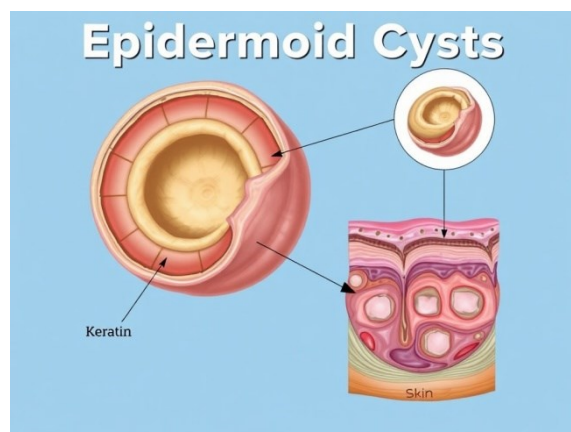
Cutaneous cysts are encapsulated, epithelium-lined cavities that commonly present in outpatient dermatology and general surgery clinics. Among these, lesions filled with keratinaceous material—collectively termed **keratinous cysts**—are overwhelmingly the most frequent. The terminology applied to these cysts has evolved and often been a source of confusion in both clinical and academic settings. The term **epidermal inclusion cyst (EIC)** is deeply entrenched in medical literature and accurately reflects a common pathogenic mechanism: the traumatic implantation of surface epidermis into the dermis [1]. The more general term **epidermoid cyst (EC)** denotes a cyst lined by epidermis-like squamous epithelium that possesses a granular layer and produces keratin [2]. It is crucial to distinguish these from **pilar cysts** (trichilemmal cysts), which are derived from the outer root sheath of the hair follicle, lack a granular layer, and are typically filled with a denser, more homogeneous keratin [3].

The historical understanding of these cysts dates back to the 19th century, with detailed pathological descriptions emerging as microscopy advanced. Their ubiquitous nature makes them a universal clinical encounter. While predominantly benign and managed with straightforward procedures, their propensity to become inflamed, infected, or rupture transforms them from an incidental finding into a painful condition requiring urgent intervention. Moreover, the presentation of multiple cysts can be a cutaneous marker of underlying genetic syndromes, adding a layer of diagnostic importance [4]. This paper aims to synthesize a comprehensive body of knowledge on keratinous cysts, providing a resource that spans from fundamental pathology to advanced therapeutic options, thereby enhancing clinical decision-making and patient care.

## **Epidemiology**

Keratinous cysts are a global phenomenon with a high incidence, though exact prevalence is difficult to ascertain as many asymptomatic cysts go unreported. They can occur at any age but are exceptionally rare in infants and show a rising incidence after puberty, peaking in the third and fourth decades of life [5]. A slight male predominance has been observed in several large-scale studies, with a male-to-female ratio of approximately 1.5:1 to 2:1 [6]. The reasons for this disparity are not fully understood but may be linked to higher rates of traumatic exposure in certain male-dominated occupations and differences in androgen-mediated sebaceous activity.

Anatomically, these cysts demonstrate a predilection for regions rich in pilosebaceous units. The most common sites include the face (particularly the periorbital area and cheeks), neck, trunk (back and chest), and scrotum [6]. On the scalp, what appears clinically as an epidermoid cyst is often, upon histological examination, a pilar cyst, as the latter constitutes the majority of cysts in this location [3]. The occurrence of multiple cysts is relatively uncommon in the general population. However, the presence of numerous cysts, especially at a young age or in unusual locations, should raise clinical suspicion for syndromic associations. The most well-established link is with **Gardner's syndrome**, a variant of Familial Adenomatous Polyposis (FAP) caused by mutations in the APC gene. Patients with this syndrome develop multiple epidermoid cysts, often on the face and scalp, which can precede the diagnosis of intestinal polyposis by years [7]. Other associated conditions include basal cell nevus syndrome (Gorlin-Goltz syndrome) and the rare CYLD cutaneous syndrome (Brooke-Spiegler syndrome), which is associated with multiple familial trichoepitheliomas, spiradenomas, and cylindromas [8].



**Figure 1:** Keratinous cysts

## **Etiology and Pathogenesis**

The formation of a keratinous cyst is a process of epithelial sequestration and subsequent proliferation. The pathogenesis can be broadly categorized into two primary mechanisms: spontaneous and acquired.

**Spontaneous Development:** The vast majority of cysts develop spontaneously from the **infundibulum**, the uppermost portion of the hair follicle. This process is thought to begin with the obstruction of the follicular orifice. The continuous production and shedding of keratinocytes (a process known as keratinization) within the plugged follicle leads to the accumulation of keratin and lipids. This expanding mass gradually causes the follicular wall to dilate, eventually forming a true cystic structure lined by epithelium that is histologically identical to the infundibulum, complete with a granular layer [2]. This explains the high density of cysts in areas with numerous hair follicles.

**Acquired Implantation:** The second mechanism involves the traumatic implantation of epidermal fragments into the dermis. This can occur through various insults, including blunt trauma, laceration, surgical procedures (e.g., grafting, tattooing, vaccination), or even chronic friction and micro-abrasions [9]. The implanted epidermal cells retain their proliferative capacity

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and begin to produce keratin in an enclosed space. As the keratin accumulates, the lesion expands, forming a true cyst. This etiology is common in areas prone to injury, such as the palms and soles, where pilosebaceous units are sparse, making spontaneous origin less likely [1].

**Molecular and Genetic Factors:** Beyond gross anatomy, molecular pathways are implicated. Mutations in genes regulating epithelial cell growth and differentiation may play a role. In Gardner's syndrome, the dysfunctional APC protein leads to constitutive activation of the Wnt/ $\beta$ -catenin signaling pathway, which promotes epithelial proliferation and is hypothesized to contribute to cystogenesis [7]. Furthermore, studies have shown upregulation of certain cytokeratins and inflammatory cytokines within the cyst wall and contents, which may influence the cyst's growth rate and its tendency to provoke an inflammatory response upon rupture [10].

## Clinical Presentation and Classification

The classic presentation of a simple, uncomplicated keratinous cyst is a slow-growing, firm-to-fluctuant, round, mobile, subcutaneous nodule. The overlying skin is typically normal, though it may appear slightly translucent or have a yellowish hue. The most pathognomonic feature is a **central punctum**—a small, often darkened opening that represents the connection to the surface epidermis or the occluded follicular ostium. Pressure on the cyst may express a thick, foul-smelling, cheesy material consisting of keratin and lipid breakdown products; this substance is sometimes semi-solid and has been descriptively compared to cottage cheese [3].

Keratinous cysts can be classified based on their clinical behavior:

- **Simple Cyst:** Asymptomatic, mobile nodule with no signs of inflammation.
- **Inflamed Cyst:** Presents with pain, tenderness, erythema, and swelling, often due to rupture of the cyst wall and subsequent leakage of keratin into the dermis, triggering a foreign-body granulomatous reaction. This can occur without bacterial infection.

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- **Infected Cyst:** Shows signs of inflammation (erythema, warmth, pain, swelling) accompanied by purulent drainage, fever, or other systemic signs of infection. Often follows manipulation or spontaneous rupture.
- **Ruptured Cyst:** May present as a sudden, painful, indurated mass. The initial cystic architecture can be lost, making diagnosis challenging.
- **Calcified Cyst:** A long-standing cyst may undergo dystrophic calcification, presenting as a very hard, rock-like nodule.

**Table 1:** Clinical Features of Common Cutaneous Cysts

Cyst Type	Origin	Common Location	Common Location	Content	Punctum	Histological Key Feature
Epidermoid	Infundibulum of hair follicle	Face, Neck, Trunk	Face, Neck, Trunk	Foul-smelling, cheesy keratin	Often present	Stratified squamous epithelium with granular layer
Pilar (Trichilemmal)	Outer root sheath of hair follicle	Scalp (>90%)	Scalp (>90%)	Dense, homogeneous, odorless keratin	Usually absent	No granular layer; palisaded outer layer
Dermoid	Entrapped ectoderm along embryonic lines	Periorbital, midline	Periorbital, midline	Keratin, hair follicles, sebaceous glands	Sometimes present	Contains adnexal structures (e.g., sebaceous glands)

## Histopathology

Histological examination provides the definitive diagnosis and is essential for ruling out malignancy, especially in atypical cases. The hallmark features of an epidermoid cyst are consistent across most specimens [2, 11]:

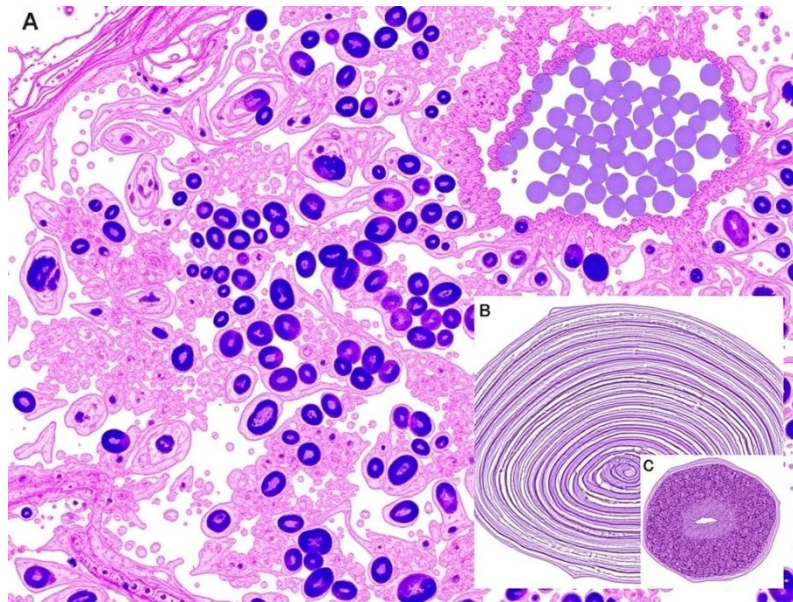
- **Cyst Wall:** Composed of several layers of stratified squamous epithelium that is morphologically identical to the surface epidermis or the infundibulum of a hair follicle. A prominent **stratum granulosum** is a critical diagnostic feature, containing keratohyalin granules.
- **Cyst Contents:** The lumen is packed with eosinophilic, **laminated orthokeratotic keratin**, arranged in concentric layers.

- **Surrounding Dermis:** Typically unremarkable in a simple cyst.

The histological picture changes dramatically if the cyst ruptures. The leakage of keratin into the dermis acts as a potent irritant, inciting a significant **foreign-body granulomatous inflammatory reaction**. This is characterized by an influx of neutrophils, lymphocytes, histiocytes, and multinucleated **giant cells** that attempt to phagocytose the keratin fragments (See Figure 2). The surrounding tissue often shows fibrosis and chronic inflammation, which can make complete surgical excision more challenging.

The differential diagnosis histologically includes:

- **Pilar Cyst:** Lacks a granular layer. The epithelium stains more basophilically and shows palisading of the outer layer. The keratin contents are more homogeneous and not laminated.
- **Dermoid Cyst:** Contains adnexal structures such as sebaceous glands and hair follicles within its wall.
- **Steatocystoma:** The cyst wall is characteristically thin and lined by stratified squamous epithelium with **corrugated eosinophilic cuticle** and contains **sebaceous lobules** within the wall.



**Figure 2:** Histopathology of a Ruptured Epidermoid Cyst

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\*This hematoxylin and eosin (H&E) stained section (100x magnification) demonstrates the classic features of cyst rupture. Fragments of the eosinophilic, laminated keratin (K) are visible in the dermis. The surrounding tissue shows a dense inflammatory infiltrate with numerous multinucleated foreign-body giant cells (arrows), which are congregating around the keratin debris. Remnants of the cyst wall (CW) are seen on the right.\*

*(Source: Adapted from Weedon's Skin Pathology, 2019)*

## Diagnostic Imaging

While clinical examination is usually sufficient, imaging plays a crucial role in diagnosing deep, recurrent, or complicated cysts, and in excluding other soft tissue masses.

- **Ultrasonography (USG):** This is the first-line imaging modality due to its accessibility, low cost, and real-time capabilities. A typical simple epidermoid cyst appears as [12]:
  - A well-circumscribed, oval or round, hypoechoic mass.
  - **Posterior acoustic enhancement** (increased echogenicity behind the cyst) due to its fluid-filled nature.
  - A **pseudotestis appearance** due to its homogeneous internal echotexture.
  - Internal echogenic foci representing keratin debris or cholesterol crystals.
  - A tract leading to the skin surface (the punctum) may sometimes be visualized.
  - In infected or inflamed cysts, the wall becomes thickened and hyperemic on color Doppler, showing increased vascular flow.
- **Magnetic Resonance Imaging (MRI):** MRI is superior for evaluating deep-seated lesions, planning surgery for complex cases, and clarifying ambiguous ultrasound findings. The characteristic MRI findings include [13]:
  - **T1-weighted images:** Typically show **isointensity** or slight **hyperintensity** compared to muscle, due to the lipid-rich content of the keratin.
  - **T2-weighted images:** Usually show **marked hyperintensity**.

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- **Diffusion-Weighted Imaging (DWI):** A key diagnostic feature is **restricted diffusion**, appearing hyperintense on DWI with corresponding low signal on Apparent Diffusion Coefficient (ADC) maps. This helps distinguish them from other purely fluid-filled cysts.
- **Post-contrast images:** The thin cyst wall may show smooth, rim enhancement. In ruptured cysts, there is pronounced peripheral enhancement of the inflamed capsule and surrounding tissue.
- **Computed Tomography (CT):** CT is less specific but may be used incidentally. It shows a well-defined, fluid-density mass. Dystrophic calcification within the wall may be evident.

## Differential Diagnosis

The clinical presentation of a keratinous cyst can overlap with numerous other subcutaneous nodules. A systematic approach is necessary for accurate diagnosis.

- **Pilar Cyst (Trichilemmal Cyst):** Clinically very similar but overwhelmingly located on the scalp. The wall is more fragile and the contents are often denser and odorless. Definitive distinction is histological.
- **Lipoma:** A benign tumor of adipocytes. It is typically softer, more lobulated, and lacks a central punctum. Ultrasound readily differentiates a hyperechoic, striated lipoma from a hypoechoic cyst.
- **Abscess:** A collection of pus. An infected cyst is essentially an abscess surrounding a cyst capsule. A history of a pre-existing nodule suggests an infected cyst, while a rapidly developing painful swelling may suggest a primary abscess.
- **Pilomatrixoma:** A benign tumor of hair matrix cells. Common in children and young adults. Often presents as a hard, "rock-like" nodule with a reddish-blue discoloration of the overlying skin. The "tent sign" (multiple angles and facets) may be present.
- **Ganglion Cyst:** A mucin-filled cyst connected to a joint or tendon sheath. Common on the wrists and hands. It is transilluminable and contains viscous, clear fluid, not keratin.
- **Dermoid Cyst:** A congenital inclusion cyst that occurs along embryonic fusion lines. Contains mature skin appendages. Often diagnosed in childhood.

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- **Malignant Tumors:** Rarely, metastatic skin nodules, soft tissue sarcomas, or cystic basal cell carcinomas can mimic a cyst. Features suggestive of malignancy include rapid growth, fixation to deep structures, ulceration, and neurological symptoms. Any atypical feature warrants biopsy.

**Table 2:** Key Differentiators in the Clinical Diagnosis of Subcutaneous Nodules

Feature	Epidermoid Cyst	Lipoma	Abscess	Pilomatrixoma
Consistency	Firm, fluctuant	Soft, lobulated	Fluctuant, tender	Very firm, "rock-like"
Punctum	Often present	Absent	Absent	Absent
Content	Cheesy, keratinous	Fatty tissue	Purulent	Gritty, calcified material
Transillumination	Sometimes	No	No	No

## Complications

Complications are the primary reason patients seek treatment for otherwise benign cysts.

- **Inflammation and Rupture:** This is the most frequent complication. Rupture can be spontaneous or secondary to trauma. The ensuing foreign-body reaction causes significant pain, tenderness, erythema, and induration, often mimicking cellulitis or a furuncle. It can lead to scarring and fibrosis, making future excision more difficult [14].
- **Secondary Infection:** Bacteria (typically *Staphylococcus aureus* and other skin flora) can invade the cyst, usually through the punctum, leading to an infected abscess. This presents with increased pain, swelling, warmth, and purulent drainage. Systemic symptoms like fever may occur.
- **Calcification:** Long-standing cysts may undergo dystrophic calcification, presenting as a hard, calcified mass on palpation and imaging.
- **Fistula Formation:** A chronically draining sinus tract may develop from a recurrently infected or inflamed cyst.

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- **Malignant Transformation:** This is an exceedingly rare event, with an incidence estimated at less than 0.01% [15]. The most common malignancy arising within an epidermoid cyst is **squamous cell carcinoma (SCC)**, followed by basal cell carcinoma (BCC). Transformation should be suspected in cysts demonstrating rapid growth, ulceration, fixation, or recurrence after adequate excision. Diagnosis is confirmed histologically.

## Management and Treatment

Management is tailored to the cyst's presentation: asymptomatic, inflamed, infected, or requiring definitive cure.

**Conservative Management:** Small, asymptomatic cysts require no intervention. Patient education is key to discourage attempts at self-drainage, which often lead to infection and scarring.

**Management of Inflamed Cysts:** For a cyst that is inflamed due to rupture but not infected, the treatment of choice is **intralesional corticosteroid injection** (e.g., triamcinolone acetonide 5-10 mg/mL). This potently suppresses the foreign-body granulomatous inflammation, leading to a rapid reduction in pain, swelling, and erythema within 48-72 hours [16]. The cyst may resolve completely or regress significantly.

**Management of Infected Cysts:** A cyst with clear signs of bacterial infection (purulence, significant erythema) requires management as an abscess. This involves:

- **Incision and Drainage (I&D):** A small incision is made to evacuate the pus and keratin debris. The cavity may be packed.
- **Systemic Antibiotics:** Prescribed if there is surrounding cellulitis or systemic symptoms. Antibiotics with coverage for *S. aureus* (e.g., cephalexin, dicloxacillin) are appropriate. It is critical to inform the patient that I&D is not a definitive treatment and that the cyst is very likely to recur once the acute infection resolves. Elective excision of the residual capsule should be planned 4-6 weeks later.

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**Definitive Surgical Treatment:** Complete surgical excision of the cyst and its entire wall is the only permanent cure. The goal is to remove the cyst **intact** to prevent spillage of inflammatory contents and recurrence. Two main techniques are employed:

1. **Conventional Elliptical Excision:** This is the gold standard, especially for larger or previously inflamed cysts. An elliptical incision is made, encompassing the punctum. The cyst is then meticulously dissected from the surrounding fibrous tissue using blunt and sharp dissection until it is freed and removed en bloc. This technique offers the lowest recurrence rate but results in a longer linear scar [17].
2. **Minimal Excision Technique (MET) / "Keyhole" Technique:** This technique is favored for smaller, mobile cysts due to its superior cosmetic outcome. A 2-4 mm linear incision is made over the cyst. The cyst contents are expressed by applying pressure. Then, a hemostat or fine curette is inserted through the incision to grasp the entire cyst wall and deliver it through the small opening. The tiny incision is closed with a single suture or sterile adhesive strips. The recurrence rate is slightly higher than with conventional excision and is highly dependent on the surgeon's skill in removing the entire capsule without fragmentation [5, 17].

The choice of technique depends on cyst size, location, history of inflammation, and surgeon and patient preference.

### Prognosis and Follow-up

The prognosis for keratinous cysts is excellent. After complete surgical excision, the recurrence rate is very low, estimated at 1-2% for conventional excision and 3-5% for the minimal excision technique [17]. Recurrence is almost always due to incomplete removal of the cyst wall, often because of prior inflammation that has fragmented the capsule or obscured the dissection plane. No long-term follow-up is necessary for simple, excised cysts. Patients should be advised to seek care for any new, growing, or changing subcutaneous nodules. For patients presenting with multiple cysts, especially with a family history of colorectal cancer or other skin tumors, appropriate genetic counseling and screening should be considered.

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## Conclusion

Keratinous cysts, encompassing epidermoid and epidermal inclusion cysts, are a ubiquitous clinical entity with a significant impact on patient quality of life when complicated. A thorough understanding of their etiology, which ranges from spontaneous follicular occlusion to traumatic implantation, is fundamental. The classic clinical presentation is often diagnostic, but clinicians must be adept at recognizing complications and considering a broad differential diagnosis that includes both benign and malignant mimics. Ultrasonography serves as a valuable bedside tool for confirmation, while histopathology remains the diagnostic gold standard. Management is nuanced: asymptomatic cysts can be observed, inflamed cysts respond well to intralesional steroids, infected cysts require drainage and antibiotics, and definitive cure is achieved only through complete surgical excision of the capsule. Mastery of both conventional and minimal excision techniques allows the clinician to tailor treatment to the individual patient, ensuring optimal outcomes in terms of both cure and cosmesis. Future research into the molecular pathways of cystogenesis may open doors to novel non-surgical therapeutic options.

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