Steatocystoma Multiplex

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Introduction

Steatocystoma multiplex (SM, also known as steatocystomatosis, sebocystomatosis, or epidermal polycystic disease) is a rare benign intradermal true sebaceous cyst of various sizes.[1,2] It is considered a nevoid or hamartomatous malformation of the pilosebaceous junction.[3] The classification of SM includes localized, generalized, facial, flexural, acral, syndromal, and suppurative types.[4] This review intends to draw attention to this unique dermatological condition.

History

The term SM translates to "a bag of fat." In 1873, SM was first described by Jamieson in a case with numerous cysts distributed throughout the body. The term 'steatocystoma multiplex' was coined in 1899 by Pringle.[4] In 1982, Brownstein described steatocystoma simplex as a distinct entity.[5]

Epidemiology

The exact prevalence of SM is not known in the general population, with no gender or ethnic predilection.[6] In acral subcutaneous SM, a female preponderance is reported.[7] The occurrence of SM is common in the second and third decades of life. However, the skin lesions can occur at any age, as evidenced by their occurrence at birth or as old as 78 years of age.[8] SM in four successive generations in the same family have been reported.[9]

Aetiopathogenesis

The exact pathogenesis of SM is not known. According to the retention cyst theory, an initial sebaceous duct blockage with a keratinous plug results in cyst formation. However, it fell out of favor, as on numerous occasions, researchers were not able to show the blockade histopathologically.[10] Kligman and Kirchbaum hypothesized that pluripotent ectodermal cells retain the embryonic capacity to form appendages or naevi rather than inclusion or retention cysts.[1]

The keratin 17 gene (KRT17) encodes for a type 1 intermediate filament (K17) that is predominantly expressed in the hair follicles and sebaceous glands. The missense mutation of the K17 gene is associated with nevoid cyst formation in SM.[10,11] Most of the cases of SM are sporadic and are rarely inherited in an autosomal dominant trait that is linked to a mutation in exon 1 of keratin 17.[12] Other mutations rarely associated with SM include N92S, R94C, and R94H.[6] The different mutations can result in the same clinical phenotypes, whereas the same mutations can cause different clinical phenotypes.[13] Factors such as infections, trauma, or immunological episodes might be responsible as a trigger factor in SM.[3]

Clinical features

Clinically, SM presents as asymptomatic, numerous, round, smooth, firm, mobile, cystic papules, and nodules.[1,10] The lesions are uniform, with a size of a few millimeters to centimeters along the long axis.[14] The superficial lesions are yellowish, and deeper lesions tend to be skin-colored.[1] The fluid in SM is odorless, oily, clear or opaque, milky or yellow.[15] The overlying epidermal skin is often normal, with no central punctum.[1] SM can occur anywhere in the body but is more frequently seen in areas rich in pilosebaceous units such as the trunk (especially the presternal region), neck, scalp, axilla, proximal extremities, and inguinal region [Figures 1 and 2].[10]

SM can transform into steatocystoma multiplex suppurativum (SMS, also known as steatocystoma multiplex conglobatum) at any point in time during its natural

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course [Figure 3a].[16] It can have associated pain, pruritus, and pyrexia.[17] Cysts often become inflamed, rupture, and drain, resulting in scarring, thus sharing overlapping features with hidradenitis suppurativa. The secondary bacterial infection leads to malodourous discharge and abscess.[2,16]

The sporadic solitary tumor counterpart to SM is steatocystoma simplex [Figure 3b]. The atypical clinical presentations include giant SM, linear SM, palatal SS, and extensive calcification of SM.[1,12,18,19] Vulvar SM affecting the sexual activity of women is known.[20] SM of the scalp with concurrently acquired alopecia secondary to trichotillomania has been reported.[21] The occurrence of multiple SM is reported in a psoriasis patient on ustekinumab treatment.[11]

Associations

SM has been associated with hidradenitis suppurativa (HS), pachyonychia congenita, ichthyosis, koilonychia, acrokeratosis verruciformis of Hopf, hypotrichosis, hypohidrosis, hypothyroidism, hypertrophic lichen planus, multiple keratoacanthomas, rheumatoid arthritis, preauricular sinus, pili torti, pili canaliculi, neurofibromatosis 1, and polycystic kidney disease. Patients should be screened for any other ectodermal abnormalities.[14,15,22-24]

Co-occurrence of SM with hidradenitis suppurativa, eruptive vellus hair cysts, trichofolliculomas, trichoepitheliomas, and trichoblastomas have been reported.[15] Syndromal associations include X-linked recessive Lowe syndrome, Gardner syndrome, Noonan syndrome, Alagille-Watson syndrome, and Favre-Racouchot syndrome.[4]

Diagnosis

Differential diagnosis

SM is often confused with eruptive vellus hair cyst (EVHC), whose differences are mentioned in Table 1.[25,26] The other differential diagnosis of SM includes multiple epidermoid cysts, neurofibromatosis, xanthomatosis, lipomatosis, milia, follicular infundibular tumors, severe nodulocystic acne, pseudofolliculitis. The salient clinical and histopathological features of the diseases that can mimic SM and its variants are shown in Table 2. When the lesions are inflamed, folliculitis, acne conglobata, and hidradenitis suppurativa should be considered.[3,4,17,25,27]

Diagnostic methods

A diagnosis of SM should always be confirmed with histopathology. Histologically, the cysts are well encapsulated, the walls have several layers of epithelial cells that intricately fold and flattened sebaceous gland globules within or close to the cyst wall. A thick, homogenous, eosinophilic cuticle without an intervening granular layer lines the inside wall of the cyst and protrudes irregularly into the lumen [Figure 4a]. The lumen often contains oil, hair, and keratin.[1,10]

In immunohistochemistry, the inner epithelial lining of the cysts shows positive staining with calretinin, the upper layer cells in the upper cyst wall express keratin 17, and basal and suprabasal layers express keratin 14.[20,28] The fine needle aspiration cytology of the cyst fluid shows predominantly acellular, granular debris, crystalline structures, rare anucleated squamous cells, and cholesterol crystals.[29]

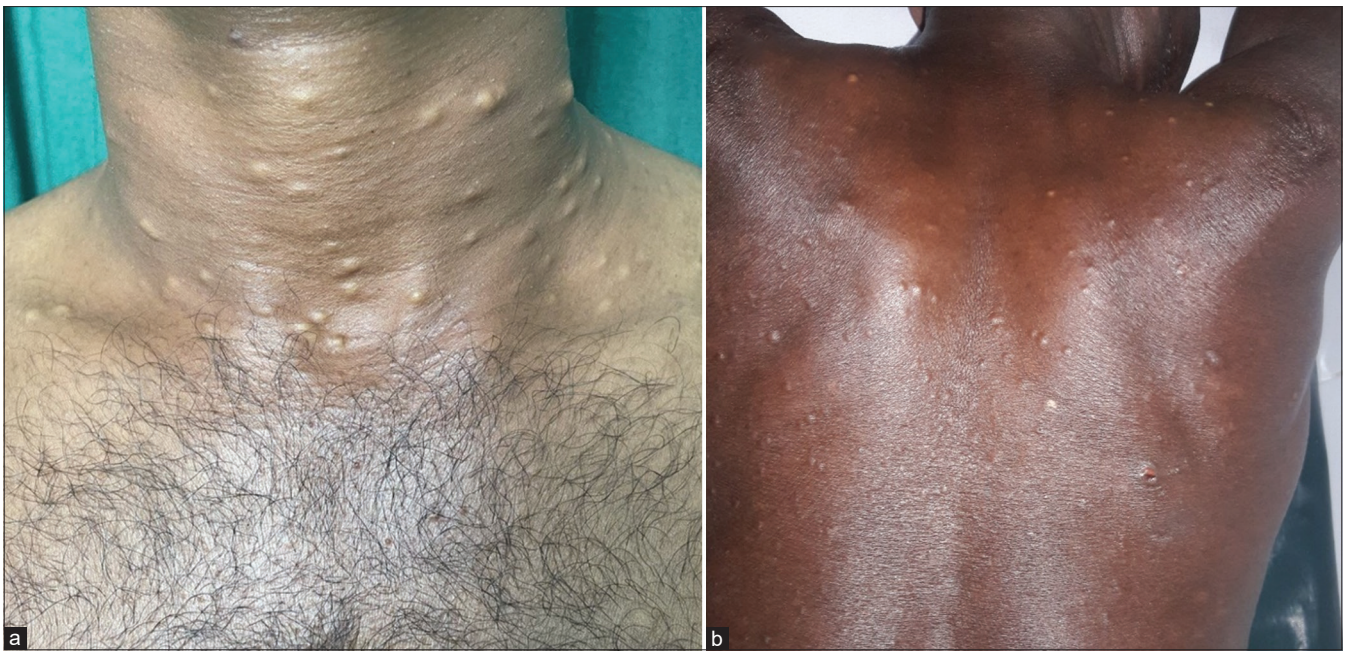

Figure 1: (a) Steatocystoma multiplex over the neck. Note the yellowish nature of the lesions (b) Steatocystoma multiplex over the posterior trunk

Table 1: Differences between steatocystoma multiplex and eruptive vellus hair cyst

Features	Steatocystoma multiplex	Eruptive vellus hair cyst
Synonym	Steatocystomatosis, sebocystomatosis, epidermal polycystic disease	Vellus hair cyst
Age	Second and third decade	Common in the second decade
Gender	No gender predilection	No gender predilection
Pathogenesis	Initial sebaceous duct blockage with a keratinous plus result in cyst formation	Developmental abnormality of vellus hair follicles that predisposes them to occlusion at the infundibulum level
	Pluripotent ectodermal cells retain the embryonic capacity to form appendages	Retention of hairs, cystic dilation of the proximal part of the follicle, and secondary atrophy of the hair bulbs
Cytokeratin expression	Express both cytokeratin 10 and 17	Express cytokeratin 10
Clinical features	Asymptomatic, multiple, round, firm, skin-colored to yellowish, mobile cystic papules and nodules of size 2 mm to 20 mm	Asymptomatic, small follicular red or brown papules of 1 to 2 mm diameter
Surface changes	Often normal with no surface changes	Some can have central puncta, umbilicated, or a hyperkeratotic crust
	Can suppurate	
Site	Common in the trunk, neck, scalp, axilla, proximal extremities, inguinal region	Chest and axilla
Course	No spontaneous resolution	Spontaneous resolution in 25% of lesions
Dermoscopy	Yellowish structureless areas with diffuse margins	Erythematous maroon halo with occasional irregular radiating capillaries at the periphery
		Vellus hairs open into the dermis
Histopathology	Well-encapsulated cysts whose walls contain several layers of intricately folded epithelial cells and flattened sebaceous gland globules within or in close proximity to cyst wall	Squamous epithelium-lined cysts in the mid-dermis containing vellus hair and keratin debris
	A thick eosinophilic cuticle protrudes into the lumen	

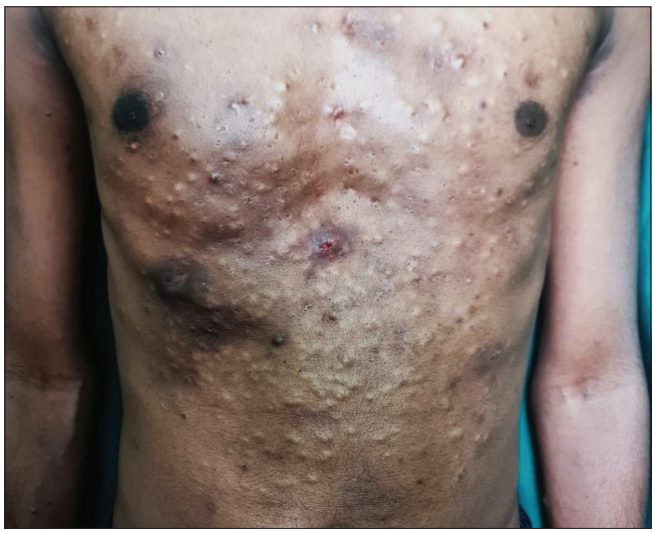

Figure 2: Extensive steatocystoma multiplex over the anterior trunk

Dermoscopy of SM shows yellowish structureless areas (corresponds to sebum contained in the cyst cavity) with diffuse margins [Figure 4b].[4] Mammographs typically reveal multiple oil cysts that have a central fat density. Ultrasound imaging shows multiple anechoic cysts with posterior acoustic enhancement.[6] Ultrasonography with color Doppler is a non-invasive tool that can differentiate SM and HS.[16] Magnetic resonance imaging shows hyperintense lesions.[30]

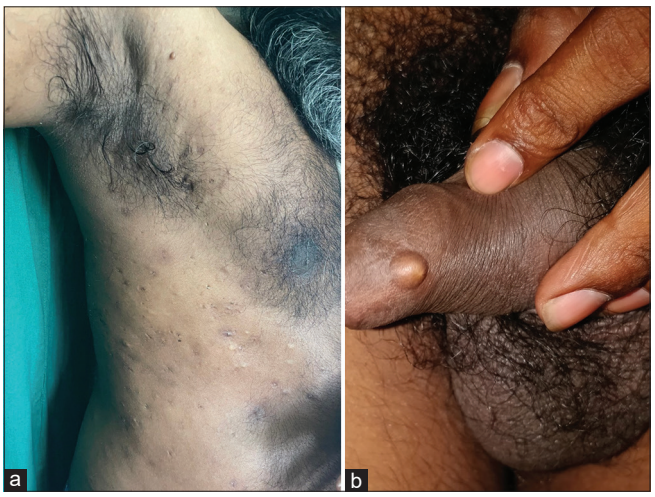

Figure 3: (a) A case of steatocystoma multiplex suppurativum. Note the simultaneous presence of active steatocystoma multiplex lesions along with scars clinically mimicking acne conglobata (b) Steatocystoma simplex in the shaft of the penis

Treatment

The goals of SM management include a substantial reduction of cyst size, good cosmesis, and good patient satisfaction. Most of the time, patients seek medical advice for cosmetic reasons.[8] There is no specific gold standard treatment option, and it should be geared toward the patient's clinical presentation.[6] A wide variety of treatment modalities

Table 2: Characteristics of the differential diagnosis of variants of steatocystoma

Condition	Age and gender	Clinical features	Site	Investigations
Steatocystoma simplex				
Solitary apocrine hidrocystoma	Common in adults aged 30–70 years. No gender predilection	Solitary, intradermal, firm, dome-shaped, translucent, bluish, cystic nodule of size ranges from 3 to 15 mm.	Head, face, and neck	Histopathological examination (HPE): Inner cyst wall lined by columnar, eosinophilic cells that show prominent luminal blebbing
Solitary eccrine hidrocystoma (Smith type)	Common in adults without any gender predilection	Clinically, they are solitary, dome-shaped, have an amber, brown, or bluish tint, and range from 1 to 6 mm in diameter. Cysts grow in size in summer	Around eyelid skin	HPE: Unilocular cyst in the dermis lined by a thin epithelial layer consisting of 1–2 layers. The cells are bilaminar and have slightly eosinophilic cytoplasm
Dermoid cyst	Common in children with no gender predilection	It presents as an asymptomatic, pale, flesh-colored, pearly, dome-shaped, firm, deep-seated, subcutaneous nodule	Head and neck region	HPE: Well-defined wall lined by stratified squamous epithelium and a lumen that may be filled with hair follicles and shafts, sebaceous and eccrine glands.
Steatocystoma multiplex				
Epidermoid or infundibular cyst	Young and middle-aged adults with no sex predilection	Slowly-growing, yellowish, white, or skin-colored, firm, smooth, dome-shaped cysts with a central keratin-filled punctum.	Scalp, face, neck, upper trunk	HPE: Early cysts lined by stratified squamous epithelium with a granular layer Cyst is filled with horny material arranged in laminated layers
Milia	Common in all age groups	Multiple, superficially located, firm, white, globoid lesions of 1–2 mm in diameter	Face, cheeks, and eyelids	HPE: Cyst with a stratified squamous epithelial lining with a granular layer and contains lamellated keratin located in the superficial dermis
Trichilemmal or pilar cyst	More common in middle-aged women	Smooth, mobile, firm, and rounded nodules without punctum Inflamed cysts become tender and rupture following an infection.	Scalp	HPE: Cysts lined by stratified squamous epithelium that show a distinct peripheral palisading. No granular layer Cyst contents are homogenous and eosinophilic and contain cholesterol clefts
Neurofibromatosis	Onset in childhood	Soft, lilac-pink tumors, sessile and dome-shaped ranging from a few millimeters to several centimeters	Common in trunk and limbs	HPE: Circumscribed tumors of reticular dermis composed of thin spindle cells with elongated nuclei spaced regularly among thin, wavy, collagenous strands. Hypercellular and enlarged small nerves
Familial multiple lipomatosis	Common in the third decade without any gender predilection	Multiple, discrete, round-to-oval, rubbery, encapsulated lipomas with variable tenderness on the trunk and extremities	Trunk and extremities	HPE: Proliferation of mature adipocytes with paucicellular fibrous septa
Pseudofolliculitis	After puberty in males	Multiple, small papules and pustules with a chronic relapsing and remitting course Papules may scar, form keloid and leaves hyperpigmentation	Beard area, particularly over the jaw and neck	HPE: Acute inflammation, microabscesses, and foreign body giant cell granuloma formation in the follicle and perifollicular areas
Steatocystoma multiplex suppurativum				
Acne conglobata	Common in second and third decade	Multiple and extensive inflammatory papules, tender nodules, and abscesses coalesce to form malodorous draining sinus tracts that heal with hypertrophic and atrophic scars	Trunk and upper limbs extending to the buttocks	No consistent laboratorial abnormalities Bacterial culture from the discharge fluid
Severe nodulocystic acne	Seen in second decade Common in males	Severe form of acne characterized by multiple nodules, sinuses, and scarring	Face, chest, and back	Clinical diagnosis Hormonal evaluation

Contd...

Table 2: Contd...

Condition	Age and gender	Clinical features	Site	Investigations
Steatocystoma multiplex suppurativum				
Hidradenitis suppurativa	Onset in the second and third decades Common in females	Recurrent and chronic lesions characterized by painful subcutaneous nodules or abscesses, fistula, sinus tracts, and scarring	Axilla, inguinal, ano-genital, sub- and intermammary areas	Microbiology (swabs, purulent exudate and tissue) HPE: Follicular hyperkeratosis, follicular epithelial hyperplasia, perifolliculitis, and dense mixed inflammatory infiltrates in early lesions. Neutrophilic abscess that may connect with squamous epithelium-lined cysts and sinus tracts in advanced lesions Imaging (Ultrasound and magnetic resonance imaging) defines extension of the disease

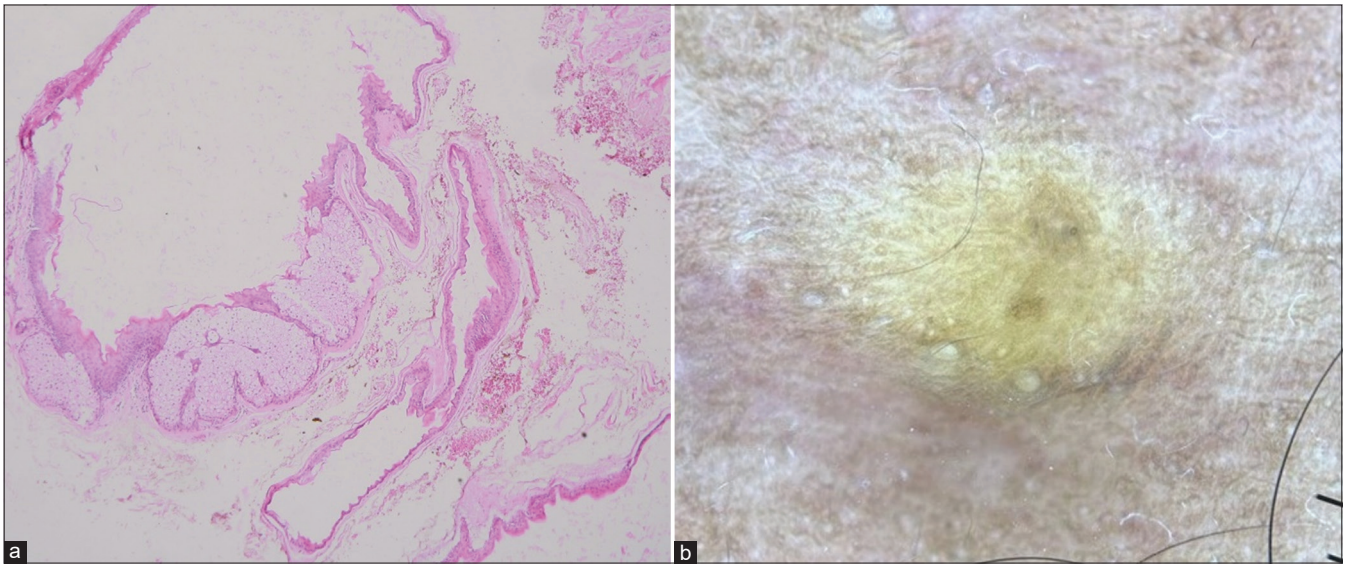

Figure 4: (a) Cyst lining in a wavy, homogenous, eosinophilic horny layer with several layers of epithelial cells collapsed around the cystic space. Note the embedded lobules of sebaceous glands among the epithelial cells (H&E, 20x) (b) Dermoscopy showing the yellowish structureless area (Dermlite D4, polarized, original magnification x10)

have been tried for SM.[8] The treatment response for this condition is generally not satisfactory.[17] The eventual recurrence of the lesions after treatment is the rule.[1]

Surgical management includes simple aspiration with an 18-gauge needle, incision and expression of the cyst contents, and radiofrequency probe-mediated extrusion of the contents [Figure 5].[1,8] The use of tissue adhesives for skin closure following the surgical removal of the cyst has good efficacy.[31] For localized variants of SM, local destructive methods such as cryotherapy have been reported to be efficacious.[32] The complete surgical excision of the cysts followed by skin grafting has been tried in the past.[33] Tricarboxylic acid treatment of the cyst walls can be administered after the aspiration of contents.[34]

For non-infectious inflammatory lesions, a short course of oral tetracyclines, topical benzoyl peroxide wash, or clindamycin might be considered. Systemic isotretinoin, due to its anti-inflammatory effect, decreases the size of pre-existing cysts and prevents the formation of new lesions.[35] However, some authors have reported worsening or exacerbating the condition in patients with SM and severe inflammation. Few have observed that systemic isotretinoin had no effect on non-inflamed lesions of SM. A combination of oral rifampicin and clindamycin has shown efficacy in the case of SMS.[36] The treatment with adalimumab in patients with coexistent SMS and HS has shown good efficacy.[2]

Lasers are advantageous because they are minimally invasive. Ablative laser therapies, such as erbium: yttrium-garnet laser, fractionated and non-fractionated Co2 laser, have been tried.[37] A combination of two non-ablative lasers with complementary mechanisms,

such as the 1550-nm fractionated Erbium-doped fiber laser that targets dermal cysts and the 1450-nm diode laser that targets abnormal sebaceous glands have shown dramatic improvement in a patient with recalcitrant SM.[38] The 1927-nm fiber-optic diode laser has demonstrated excellent clinical outcomes with minimal adverse effects in a patient with facial SM.[39] A summary of the treatment options for SM is mentioned in Table 3.[31,35,37-42]

Conclusion

SM should be considered as one of the differential diagnoses in patients with multiple asymptomatic intradermal cysts. Early disease recognition and appropriate counseling might help alleviate the psychological implications associated with the disease. It is a rare clinical entity with poor treatment outcomes.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Table 3: Summary of various treatment modalities in steatocystoma multiplex

Treatment modality	Advantages	Disadvantages
Medical management		
Isotretinoin – administered orally at 1 mg/kg/daily for six months	Decreases the size of pre-existing cysts	Recurrence
	Prevents the formation of new lesions	Results are limited only to inflamed lesions
		Can produce exacerbation of flare of disease initially
Tetracyclines – administered orally	Benefit for suppurative variant of SM	Poor efficacy
Surgical management		
Cryotherapy	Treatment of multiple lesions in a single setting	Extremely low efficacy cosmetic disfigurement
	Suitable for larger lesions, even calcified ones	Blister formation
		Scarring
		Hypopigmentation
Radiofrequency - Expression of the contents with mini-incisions (1–2 mm) made on the cyst	Produces bloodless field	Time consuming
	No sutures and scar formation	
	No recurrence	
Needle aspiration with extirpation of the contents	Inexpensive	Requires a skilled operator. Does not work well on very large (>15 mm) and small (3 mm) cysts
	Well-tolerated	
		High recurrence rate
Surgical excision - surgery with elliptical excisions, flaps, or grafts	Followed in older days	Time consuming
		Scarring
		Not practical for widespread lesions
Surgical techniques – Fine incision followed by cyst wall extraction with forceps, curette, or vein hooks	Shows excellent cosmetic results	Time consuming and invasive
	Cost effective	Transient post-inflammatory hyperpigmentation
Lasers		
Fractional Co2 laser - For cyst opening, drainage, and vaporization of the cyst wall and remnants	Can treat multiple lesions in a single session with good cosmetic outcomes	Not suitable for larger cysts
	Minimal scarring	
	Low recurrence	
Erbium-YAG (Yttrium Aluminium Garnet) laser	Good toleration	Not cost effective
	No scarring	Not easily accessible
	Improved quality of life	

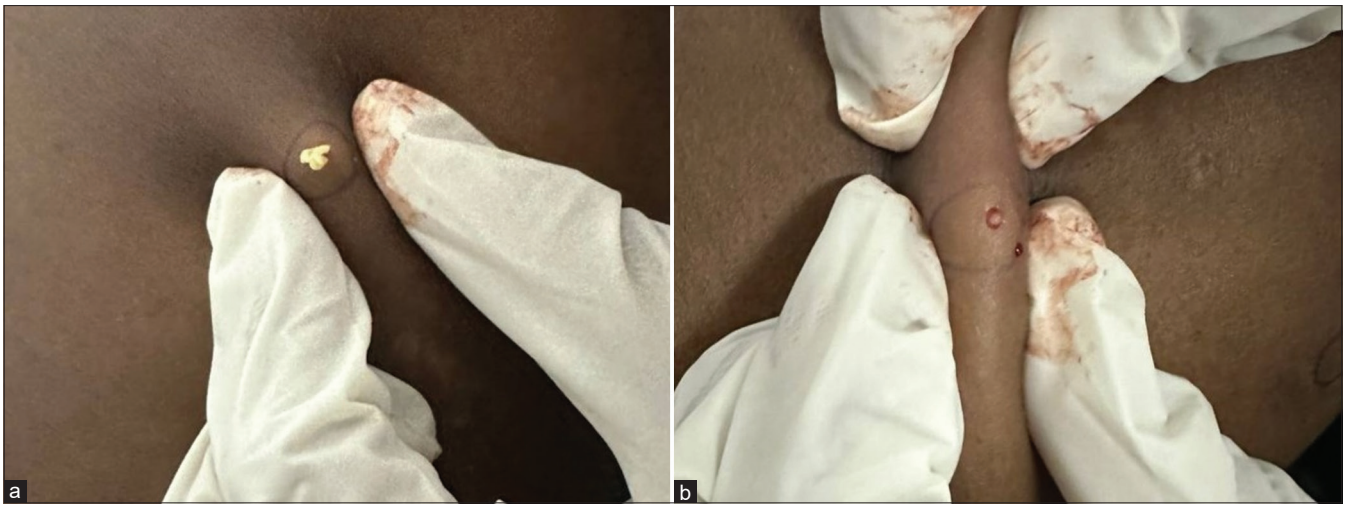

Figure 5: (a) Extrusion of cheesy, oily material after a small nick in a lesion (b) Sac protruding out of the steatocystoma multiplex

Conflicts of interest

There are no conflicts of interest.

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