Fox-Fordyce Disease after Laser Hair Removal: A Hidden Consequence of Cosmetic Procedures

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ABSTRACT

Background: Fox-Fordyce disease (FFD) is a chronic, pruritic inflammatory disorder of apocrine gland–bearing skin, most often affecting women of reproductive age. Traditionally linked to keratin plugging and ductal obstruction, it remains underdiagnosed. Recent case reports suggest laser hair removal (LHR) may act as an iatrogenic trigger, adding a novel dimension to its pathogenesis. **Objective:** To synthesize current evidence on the relationship between LHR and FFD, highlighting clinical patterns, possible mechanisms, diagnostic challenges, and management considerations. **Methods:** A narrative review was conducted using PubMed and Embase, supplemented by dermatology conference proceedings. Search terms included "Fox-Fordyce disease," "apocrine miliaria," "laser hair removal," and "cutaneous complications." 34 PubMed-indexed sources, case reports, and abstracts were included. Findings were integrated thematically due to the rarity of the condition and variability of study designs. **Results:** Evidence from case reports and small series indicates that FFD can develop after LHR, with latency periods ranging from weeks to years. Proposed mechanisms include laser-induced follicular injury, altered keratinization, and apocrine duct obstruction, potentially influenced by hormonal factors. Histology parallels idiopathic FFD but may lack mast cell infiltration. Diagnosis is often delayed, particularly in darker skin types where lesions present with variable pigmentation. Management remains inconsistent, spanning topical, systemic, and procedural options, with pulsed dye laser showing

emerging therapeutic promise. **Conclusion:** LHR may be an underrecognized precipitating factor for FFD. Greater clinician awareness, inclusive dermatologic training across skin types, and targeted research into pathogenesis, prevention, and treatment are essential for improving diagnosis and patient outcomes.

Keywords: Fox-Fordyce Disease, Apocrine Glands, Laser Hair Removal, Iatrogenic Dermatology, Follicular Disorders, Cosmetic Procedure Complications

INTRODUCTION

Fox-Fordyce disease (FFD), or apocrine miliaria, is a rare, chronic, pruritic, inflammatory dermatologic condition involving areas of the skin with apocrine glands such as the axillae, anogenital area, and periareolar skin. FFD predominantly affects women between puberty and menopause, with a female-to-male ratio of approximately 9:1 [1]. On exam, physicians will observe 2 to 3 mm dome-shaped follicular or perifollicular papules with color variation from skin-colored to yellow, reddish, or violaceous [2]. Despite these defining features on physical exam, the disease remains poorly understood and frequently misdiagnosed. There is significant clinical overlap with other disorders of the follicle, such as folliculitis or lichen planopilaris [1]. FFD remains largely overlooked within the field of dermatology, both in education and research. Limited clinical awareness about FFD is due to the low prevalence of the disease and the absence of robust, large-scale studies. Patients often experience delayed diagnosis and delayed care. Most dermatology references mention FFD only briefly; no standardized diagnostic or therapeutic guidelines currently exist, and etiology is not thoroughly studied.

Obstruction of the apocrine gland duct due to keratin accumulation in the follicular infundibulum is thought to play a role in this disease; however, the exact etiology remains unknown [1]. Several hypotheses have been suggested. One hypothesis suggests hormonal influences are involved in FFD. The symptoms of FFD rarely appear before puberty, can worsen around menses, change either better or worse during pregnancy, and can go into remission during menopause [2]. Androgens have been known to disrupt keratinocyte adhesion and modify sweat composition [3]. This results in retained secretions, rupture of apocrine glands, and subsequent dermal inflammation. The leakage of glandular contents into surrounding tissue is believed to trigger pruritus, which

often precedes the visible emergence of papules in FFD. Stress, sweating from exercise, tight clothing, and humid environments also trigger the characteristic pruritus in FFD.

Emerging evidence suggests that laser hair removal (LHR) may contribute to the pathogenesis of FFD. Aesthetic hair reduction involves lasers that use photothermolysis to destroy follicular structures [4]. The thermal energy from the laser results in follicular infundibulum injury, dysmaturation, and follicular obstruction [5]. This promotes keratin plugging and can disrupt apocrine gland outflow, a suggested mechanism of FFD. Chronic low-grade inflammation from repeated laser sessions could prime the folliculo-apocrine unit for dysfunction. Most patients will tolerate LHR without complication; however, there have been only few cases reported of FFD after LHR [6,4,7]. No causative relationship has been investigated or proven. However, the parallel timing, anatomical distribution, and histologic overlap suggest possible involvement.

Most LHR side effects, such as erythema, burns, and dyspigmentation, are transient and widely known [8]. Less known is the possibility of delayed-onset follicular dermatoses like FFD, highlighting the need for further study and awareness by dermatologists. Given the widespread use of lasers and the underdiagnosis of FFD, this association deserves structured investigation. The purpose of this review is to reposition FFD within the scope of cosmetic dermatology and bring attention to laser hair removal as a potential contributor to its pathogenesis. This information could inform new protocols for post-laser monitoring, and we hope to stimulate both diagnostic vigilance and research momentum.

Pathophysiology of FFD

Fox-Fordyce disease (FFD) results from keratin plugging at the follicular infundibulum, which obstructs apocrine ducts and leads to rupture with localized inflammation in apocrine-rich areas. The condition produces intensely pruritic papules, predominantly in postpubertal females. Histopathological analysis, as described by Salloum et al. [1], consistently reveals follicular hyperkeratosis, ductal dilation, and lymphocytic infiltration. Chronic cases may additionally show perifollicular xanthomatosis [9]. Dermoscopy, as noted by Singal et al. [10], frequently demonstrates hyperpigmented folliculocentric lesions and loss of dermatoglyphics. Despite these patterns, variable clinical morphology and absence of definitive laboratory markers contribute to underdiagnosis, necessitating

close clinicopathologic correlation [2], necessitating a strong histological-clinical association for correct diagnosis.

Recent literature suggests that laser hair removal may act as a novel external trigger capable of initiating the same obstructive and inflammatory cascade. In a detailed case study, Tetzlaff et al. [11] described FFD lesions that appeared shortly after axillary laser hair treatment, with biopsy findings mirroring those of classical FFD. Similarly, Sepaskhah et al. [7] described a case involving both the axilla and the pubic area after Alexandrite/Diode laser irradiation, which revealed follicular dilation and periductal inflammation. Sammour et al. [5] elaborated on these findings by documenting many laser-induced cases and observing that their patients' histologic characteristics, such as dyskeratosis and infundibular widening, were identical to spontaneous occurrences. A subsequent study by Zargari and Azimi [12] confirmed this pattern, presenting a 26-year-old lady with bilateral axillary lesions following diode laser treatment, bolstering the notion of a direct causal relationship.

Despite the similarity in histology, some major distinctions between idiopathic and laser-induced FFD cases may indicate differences in immune activity or disease stage. For example, Sammour et al. [5] reported that none of the laser-related cases had mast cell infiltration, which is a variable feature in typical FFD. This may imply that laser-induced instances are an acute phase or a unique immune response. Helou et al. [13] postulated that laser-induced follicular damage changes keratinocyte maturation, resulting in ductal blockage and inflammation. Additionally, hormonal predisposition appears to amplify susceptibility to such damage, especially in women of reproductive age [14]. Taken together, these findings support a multifactorial model of FFD pathogenesis in which structural injury, hormonal influence, and localized immune dysfunction interact to produce disease. These findings collectively suggest that laser hair removal may mimic the natural pathophysiologic sequence of idiopathic FFD, albeit with subtle immunological differences.

Cosmetic Dermatology & Laser Hair Removal

Laser hair removal (LHR) within the field of cosmetic dermatology has exploded in popularity within the past few decades owing to its safety, tolerability, and enduring effects on hair loss following treatment [15,16]. Its appeal over conventional hair loss modalities such as shaving, waxing, and hair removal creams can be attributed to its greater aesthetic

appeal and the durability of its effects [15,17]. Apart from cosmetic uses, laser hair removal is also a mainstay for the treatment of conditions such as hirsutism, pseudofolliculitis barbae, hidradenitis suppurativa, and hypertrichosis [18,19,16]. The mechanism of LHR relies on the concept of selective photothermolysis, in which absorption of light in the range of 300-1200nm by melanin in hair is employed for hair destruction [16]. Within the field, numerous laser types exist and the optimal choice typically varies according to the Fitzpatrick skin types [20,16]. The most common laser types include the Alexandrite (755nm) and Diode (810 nm) lasers which target skin types I-III and I-IV, respectively [20,16]. The Nd:YAG (1064 nm) is preferred for Fitzpatrick skin types V-VI [20,16].

The particular aim of LHR is to ablate hair follicle stem cells that are located in the hair shaft bulb and bulge [20,16]. As there is greater melanin in the hair shaft compared with the hair follicle, the heat from the laser that is absorbed by the shaft is dissipated to the hair follicle in which the stem cells reside [20,16]. This is referred to as the extended theory of selective photolysis that underpins how LHR works in practice [16]. Notwithstanding the allure of LHR in the cosmetic industry for its aesthetic and long-lasting outcomes, laser therapy for hair removal brings with it the potential for iatrogenic harm. Specifically, melanin within the dermoepidermal junction may absorb the light energy, leading to scars, burns, and post-inflammatory dyspigmentation [20]. Similarly, it is possible that LHR may serve as a critical source of persisting follicular trauma [21,22,23]. This notion is supported by literature that demonstrates that laser therapy oftentimes does not lead to complete destruction of hair follicles [21], but may instead inflict injury in the form of functional alterations in follicle stem cells from laser heat damage that is insufficient to cause cell death [22]. Histologic signs of injury to the hair follicle epithelium following laser therapy have been reported, encompassing nucleus elongation, marked eosinophilia, and follicular rupture [23]. Damage to the outer and inner root sheath of the follicle epithelium and the hair bulge containing stem cells following incomplete laser hair removal is one possible contributing factor to the functional derangement in hair follicles post treatment [22,23]. Taken together, these working hypotheses contribute to our current-day understanding of the pathogenesis of complications following LHR.

At the surface, perifollicular edema and erythema are common post-treatment sequelae with diode lasers [22], one

of the most widely used LHR devices in cosmetic dermatology practices today [20]. While several foundational studies from the mid-2000s remain widely cited for their mechanistic insights into follicular trauma and stem cell disruption [21-23], ongoing investigation is needed to confirm these effects in large, modern cohorts. At present, these findings among the literature illustrate the silent pathophysiological costs of laser hair removal and how much more we have yet to learn about their etiologies and pathogenesis [21-23].

Emerging Evidence: Case Reports

Current evidence from recent case reports suggests a relationship between laser hair removal and FFD. In a 2024 case report by Sepaskhah et al., a 23-year-old female presented with FFD in axillary and pubic areas after 5 sessions of laser hair removal. The lesions were described as bilateral pruritic skin-colored perifollicular papules. It was noted that these lesions were absent on other areas, such as the face and lower extremities, despite also undergoing the same laser hair removal treatment as the axillary and pubic areas. A skin punch biopsy revealed the following notable histopathological features: dilated follicular infundibulum filled with keratin material, dilated apocrine sweat glands, perifollicular fibrosis, periductal lymphohistiocytic infiltration, foamy cytoplasm of perifollicular histiocytes, and severely spongiotic follicular infundibulum with dyskeratotic acantholytic and exocytosis of lymphocytes [7]. This patient was managed with topical mometasone furoate 0.1% cream applied twice daily for 4 weeks. There was reported evidence of improvement of the lesions and pruritus, so to avoid adverse effects, they replaced the mometasone cream with tacrolimus 0.1% cream [7]. However, the patient's pruritus returned, and the initial treatment was restarted. This case depicts a "classic" presentation of FFD, which is a female of child-bearing age who develops FFD in the axillary and pubic areas after sessions of laser hair removal. Further, this case illustrates the current variety in treatment options often attempted for cases of FFD and the "trial-and-error" approach often taken to reach resolution.

In another case from 2020, Zagari & Azimi reported a 26-year-old female who had laser hair removal treatment to her extremities, face, axillae, pubic, and periumbilical areas. After 2 sessions of this treatment, the patient developed FFD of the axillae with pruritus. Their FFD was managed with topical tacrolimus. Beyond topicals, other treatment modalities have been emerging such as systemic and laser treatments [6]. A 2023 case involved a 28-year-old woman who presented with FFD in the axillae bilaterally for 4 years. The FFD was managed with 3 sessions of pulsed dye laser (PDL) on the right axilla and a fractional CO_2 laser on the left axilla [6]. After three treatment sessions, the papules appeared to have shrunken bilaterally but the PDL showed more improvement and better results overall. The patient did not experience recurrence post-treatment. This highlights recent therapeutic innovations for FFD and further underscores the variety of treatment modalities that exist for this clinical entity.

Across five cases in a 2016 case series by Sammour et al., female patients ranging from 24-42 years old showed FFD in the axillae when that area was treated except for one case where FFD was only found in the inguinal region, despite both areas being treated. Additionally, only half of the patients reported pruritus. The timeline for symptom onset varied greatly among these reported cases; from 2 months to 4 years after the last laser hair removal session. Across these reported cases of FFD, many treatment options are considered in management, including topical and oral treatments which have shown high rates of recurrence [6]. Further research is needed in the management of FFD as there is currently no definitive treatment for all cases, and long term results are not well documented due to limited patient follow-up. Due to the clinical variability in existing cases of FFD, future studies should prioritize longitudinal follow up on patients to bridge this current gap in the literature.

Hypothesized Mechanisms

The specific molecular mechanisms by which laser hair removal causes Fox-Fordyce disease (FFD) are yet unknown, although repeating histological and clinical patterns are helping to shed light on the subject. One potential mechanism is laser-induced thermal damage to the follicular infundibulum, which disturbs normal desquamation and causes keratin plugging. Helou et al. [13] reported that such trauma may disturb keratinocyte maturation, promoting infundibular obstruction and subsequent apocrine gland dilation. This physical anomaly is expected to resemble the intrinsic defect observed in spontaneous FFD, triggering an analogous cascade of duct rupture and inflammatory cell recruitment. Sepaskhah et al. [7] back this up by observing histological similarities between laser-induced and classic instances, such as follicular dilatation, hyperkeratosis, and perifollicular lymphocytic infiltration.

Another plausible mechanism is laser-mediated alteration of the local immune environment. Sammour et al. [5] observed that while laser-induced cases shared structural features with classic FFD, mast cell infiltration commonly reported in idiopathic presentations was notably absent. The disparity shows that laser-induced cases may represent an early-stage or mast cell-independent mechanism of inflammation. Zargari and Azimi [12] discovered that FFD lesions appeared after only two diode laser sessions, implying that even low energy exposure is sufficient to cause immune-epithelial dysregulation. Hanner et al. [14] have stressed the importance of hormonal responsiveness in the apocrine unit, arguing that estrogen- and androgen-sensitive pathways may increase follicular vulnerability to trauma, particularly in premenopausal women.

Laser energy itself may selectively damage in vivo structures based on skin type, follicle density, and laser wavelength, influencing disease risk. For instance, Tetzlaff et al. [11] suggested that the 800 nm diode laser could penetrate deeply enough to reach and disrupt the apocrine-duct junction, which would explain the localized inflammation and obstruction seen in post-treatment histology. In parallel, the systematic review by Mallat et al. [8] categorized FFD among several cutaneous complications of laser hair removal, reinforcing that localized thermal insult to adnexal structures is both plausible and recurrent. These overlapping lines of evidence point toward a damage repair imbalance in the follicular-apocrine complex, where laser-induced stress leads to persistent obstruction and chronic inflammation in susceptible individuals.

Diagnostic Challenges

Fox-Fordyce disease (FFD) can often be overlooked and misdiagnosed as conditions ranging from bacterial or fungal folliculitis, contact dermatitis, miliaria, arthropod bites, syringomas, lichen planus, lichen nitidus, and chronic dermatitis. This is at the cost of its clinical manifestations such as pruritic skin-colored papules, which are common amongst a multitude of dermatoses. For example, folliculitis and FFD are often mistaken for one another due to the presence of inflammation around hair follicles in both conditions. However, what mainly distinguishes FFD from conditions such as folliculitis is the specific dermatologic representation of areas rich in apocrine sweat glands such as the axillae, pubic region, and area around the nipple(areola) [5]. In addition, folliculitis often presents with pus-filled bumps, pustules, that are often absent in patients with FFD [2]. Also, the course of treatment

for these two conditions are vastly different. Folliculitis is caused by bacterial or fungal infections, so it can be treated with topical or oral antibiotics, which would be unnecessary and inappropriate treatment for a patient dealing with FFD. In addition, FFD is also commonly misdiagnosed for a heat rash, miliaria [2]. These two conditions are commonly confused for one another due to the similarity in clinical presentations of small itchy bumps on the patient. Both conditions are also exacerbated by external conditions such as heat and sweating. However, what can distinguish the two is the blockage of the transient eccrine glands in miliaria versus the chronic occlusion of aporicine glands affected in FFD [2]. Treatment wise, typical interventions for miliaria such as cooling and wearing loose clothing will not help FFD patients. Hence, although rare, FFD should always be considered on a differential diagnosis when taking a history for a patient, particularly a younger woman, who presents with these dermatological findings in areas high in concentration of apocrine glands, specifically at the hair removal laser treatment site [1]. A misdiagnosis can lead to serious complications due to the use of inappropriate treatment modalities.

Histopathology is necessary when investigating a potential diagnosis of FFD, especially when the clinical findings are uncertain. The findings most commonly seen include follicular hyperkeratosis, which is an excess of keratin that causes buildup in the follicular infundibulum [2]. Other distinct findings include ductal plugging, which in turn causes dilated apocrine ducts [10]. In addition, perifollicular lymphohistiocytic infiltrates have been seen around the hair follicles, which ultimately can contribute to the inflammation clinically presented within FFD [24]. Lastly, perifollicular xanthomatosis, also known as "foamy" histocytes, or lipid-laden histiocytes, have also been reported [9]. These findings are crucial when distinguishing FFD from other skin conditions that may present similarly such as acne vulgaris, folliculitis, or syringomas. For example, FFD histologically will lack the characteristic comedones seen in acne vulgaris cases. In addition, FFD will mainly be seen in highly concentrated areas of apocrine glands while acne vulgaris will show predominantly where there are many sebaceous glands. Furthermore, it has been noted in scientific literature that FFD has been mistaken for syringomas since they both can present similarly in the axilla region [25]. Unfortunately, biopsies are often underutilized due to aesthetic concerns of patients. This is a significant barrier, given this modalities' usefulness in confirming the diagnosis, and also guiding the appropriate treatment choice.

Challenges are even greater when diagnosing FFD on patients with darker skin types. For example, in patients with darker skin types, papules may be less obvious and lack the same erythematous quality as the typical presentation [26]. In these patients, post-inflammatory changes may be the most obvious and dominant clinical presentation. Instead of the typical red lesions, patients with darker skin tones might present with lesions shaded more brown, grey, purple, blue, or black lesions [27]. This poses complications such as overlooking the diagnosis, especially when clinicians have not been exposed to skin manifestations on darker complexions [28]. In turn, darker-skinned patients are at higher risk of a misdiagnosis, delayed treatment, and higher emotional and psychological distress stemming from skin disease [26] This has also been noted in the lack of dark skin images in various textbooks and atlases, posing a racial discrepancy and underrepresentation of dark skinned patients in medicine [29-31]. In order to potentially solve this issue, widespread changes that call for inclusive photographic documentation and training can be the first step to improve diagnostic accuracy across skin tones. In addition, artificial intelligence can be used to the advantage of medical professionals to expand their exposure to the dermatologic manifestations of skin conditions amongst a broad range of skin tones.

Treatment Strategies & Gaps

There are no established clinical management guidelines for Fox–Fordyce disease (FFD). Treatment is largely symptomatic and based on anecdotal case reports. Approaches are heterogeneous, with variable success and recurrence rates. First-line therapies include benzoyl peroxide, clindamycin, intralesional or topical corticosteroids, calcineurin inhibitors, retinoids, and oral contraceptives [2]. The mechanism by which clindamycin treats FFD is unclear. Retinoids are believed to reduce follicular and ductal occlusion. Given the predominance of FFD in females, the condition may be hormone-responsive; oral contraceptives have been trialed with limited success. Other treatments include antihistamines, calcipotriol, aluminum hydroxide, and vaginal estrogen [1]. More invasive interventions include surgical excision, fractional lasers, botulinum toxin injections, phototherapy, electrocoagulation, copper vapor or CO_2 lasers, liposuction-assisted curettage, and microwave therapy [2]. Botox may alleviate symptoms by reducing sweat and inhibiting pruritogen release [32-34]. Although laser treatment may occasionally trigger FFD, it can also be therapeutic by destroying obstructed follicular

apocrine units. This highlights the importance of identifying which patients are likely to benefit and which are at risk.

Comparative studies of treatment efficacy are limited. In a systematic review, topical retinoids were the most commonly reported treatment, used in 18% of cases, with 64% of patients reporting symptomatic improvement either as monotherapy or in combination [1]. Approximately half of the patients treated with topical clindamycin achieved resolution. Topical calcineurin inhibitors showed promise in limited cases, whereas topical corticosteroids demonstrated minimal efficacy. A case report comparing PDL and fractional CO_2 laser in the same patient found greater improvement with PDL [6]. PDL may possibly alter the inflammatory cell response, leading to a better response than CO_2 laser.

FUTURE RESEARCH & RECOMMENDATIONS

Despite the growing recognition of Fox-Fordyce disease (FFD) as a potential side effect of laser hair removal, major information gaps remain in understanding its genesis, development, and therapy. Current data are primarily generated from single case reports or small series, which limits generalizability and obscures the prevalence of laser-induced FFD in varied populations. Sammour et al. [5] emphasize that laser-induced FFD is likely underdiagnosed, suggesting that the real incidence may be significantly higher than reported. A broader investigation into predisposing factors, such as skin phototype, hormonal status, and laser wavelength parameters, is urgently needed. As noted by Mallat et al. [8], cutaneous complications of laser hair removal including FFD vary widely by individual and device characteristics, underlining the importance of standardizing safety protocols in cosmetic dermatology.

Future investigations should look into the immunological differences between spontaneous and laser-induced instances. For example, the absence of mast cells in post-laser histology [5] against their presence in chronic idiopathic FFD [9] raises the question of whether laser-induced instances constitute a distinct inflammatory pathway. Investigating cytokine profiles, barrier disruption markers, and in vivo skin immune responses following laser treatment could provide valuable insights into pathogenesis. Furthermore, research trials to determine the efficacy of preventative or early-intervention techniques, such as lower-fluence laser settings or prophylactic topical treatments, are needed. As Hanner et al. [14] and Sepaskhah et al. [7] both suggest, hormonal

modulation may influence susceptibility, which could guide personalized risk assessments prior to treatment. These avenues offer tangible steps toward refining both diagnosis and prevention in patients seeking cosmetic procedures.

CONCLUSION

Fox-Fordyce disease continues to be a clinically hard and unrecognized disorder, made more complicated by its appearance as an adverse event following laser hair removal. Recent investigations show that laser-induced FFD shares histological and clinical aspects with classic presentations, but may have unique immunological trajectories. Understanding how follicular damage, apocrine duct obstruction, and hormonal influence interact is critical to improving diagnosis and patient outcomes. As the popularity of cosmetic laser procedures grows, physicians must be attentive in monitoring post-treatment problems and include FFD in the differential diagnosis of persistent pruritic papules. Future research should strive to elucidate risk factors, validate diagnostic markers, and assess targeted treatments for both the idiopathic and iatrogenic forms of this disease.

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CONFLICTS OF INTEREST

The authors declare that there is no conflict of interest.

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