

## Tattoo-Induced Cutaneous Lymphoid Hyperplasia: A Case Series and Review of the Literature

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**ABSTRACT Introduction:** The increasing popularity of tattoos has been paralleled by a rise in tattoo-related skin reactions. Among these, tattoo-induced cutaneous lymphoid hyperplasia (CLH) remains a rare but increasingly recognized complication. Its pathogenesis and incidence are not yet fully understood, and diagnosis relies on histopathological confirmation via skin biopsy. Treatment approaches include topical or intralesional corticosteroids, surgical excision, and ablative CO<sub>2</sub> laser therapy in refractory cases.

**Objectives:** To analyze and describe the clinical and histopathological features of tattoo-induced CLH, including patient demographics, tattoo characteristics (e.g., location, ink color), latency periods, and lymphocyte predominance.

**Methods:** This study presents a case series of 15 patients with histologically-confirmed tattoo-induced CLH observed at dermatology departments in Brescia, Italy, and Gdańsk, Poland, between May 2017 and June 2023. These cases were integrated with a comprehensive literature review, resulting in a total of 58 patients analyzed. Data collected included patient demographics, tattoo location, latency period between tattooing and lesion onset, clinical features, ink color involved, lymphocyte predominance on histology, treatment modalities, and outcomes. The median patient age was 39 years, with an average latency of 33 months.

**Results:** Red ink was the most frequently implicated pigment. The predominant histopathological pattern was T-cell infiltration. Treatment responses varied. While topical and intralesional steroids showed inconsistent efficacy, surgical excision and ablative CO<sub>2</sub> laser treatments yielded the most favorable and sustained outcomes.

**Conclusions:** These findings underscore the importance of recognizing CLH as a delayed immune-mediated reaction, particularly to red pigments. Accurate diagnosis through biopsy is essential to distinguishing CLH from other tattoo-related dermatoses or cutaneous lymphomas, guiding appropriate management strategies.

## Introduction

The rising prevalence of tattooing in society has consequently led to a corresponding rise in adverse skin reactions associated with this practice [1].

However, data on the incidence of tattoo-related complications remain limited, likely due to underreporting in both the medical literature and health departments. The most common complications of tattoos include infections, allergic dermatitis, autoimmune dermatoses, and chronic inflammatory reactions [2,3]. Less frequent adverse effects include keloid formation, hypertrophic scarring, photodermatitis, neoplasm, and neurosensitive reaction [3,4].

Pseudolymphomatous reactions have been described among the less frequent complications, with, to our knowledge, only 43 cases of tattoo-induced cutaneous lymphoid hyperplasia (CLH or pseudolymphoma) reactions having been reported in the literature [5-36]. CLH is a rare entity consisting of a heterogeneous group of benign B or T lymphoproliferative reactions with clinical-histological characteristics mimicking malignant cutaneous lymphomas [6]. CLH typically occurs several months to years after tattooing [8], though cases have been documented as early as one month or as late as 42 years [14]. While red pigment is the most frequently involved pigment, other ink colors may also be responsible. The etiopathogenesis of tattoo-induced CLH remains unknown. Historically, metal components in red tattoo dyes (e.g., nickel, mercury, and cadmium) were suspected of acting as chronic antigenic stimulants, leading to polyclonal lymphoid cell proliferation [7]. While modern tattoo inks primarily contain organic azo or polycyclic pigments, heavy metals may still be present as a contaminant [37].

Accurate data on the incidence and prevalence of tattoo-induced CLH are lacking [5]. Clinically, tattoo-induced CLH presents as dome-shaped papules, nodules, or plaques at the tattoo site, which may be single or multiple. Occasionally, diffuse infiltration of the entire tattoo can be observed [5]. Since tattoo-induced CLH lacks a distinct clinical pattern, it can be indistinguishable from lichenoid or granulomatous tattoo reactions [16]. While pruritus has been reported in some cases, most lesions are asymptomatic [5].

The gold standard in the diagnosis of tattoo-induced CLH is represented by skin biopsy followed by histological examination. However, it is important to note that different pathological reactions can coexist within the same allergic tattoo reaction. For instance, a single biopsy may reveal a combination of lichenoid and pseudolymphomatous infiltration [38].

Treatment options for tattoo-induced CLH include topical or intralesional corticosteroids, although results are often unsatisfactory, and recurrences are common. In persistent cases, surgical excision or ablative CO<sub>2</sub> laser therapy is recommended.

## Materials and Methods

### Case Series

A total of 15 cases of CLH were observed during the period from May 2017 to June 2024. Eight cases were collected at the Dermatology Department of the University of Brescia, Italy, and seven cases were collected at the Dermatology Department of the University Gdańsk, Poland. Patient data, including general characteristics, tattoo location, time of lesion onset post-tattooing, clinical presentation, ink color associated with the reaction, predominant lymphocyte population on histology, treatment, and therapeutic outcomes, were retrospectively analyzed.

All patients underwent skin punch biopsies, with histological examination confirming the diagnosis of tattoo-induced CLH. Histopathological examination was performed using specific immunohistochemical staining for B and T lymphocytes (CD20, CD79, CD3, CD4, CD8). Clonality studies were not conducted. None of the patients had a notable medical history prior to the reaction. Patch testing was performed in eight patients at the University of Brescia's Dermatology Department using the standard SIDAPA baseline series, following Italian guidelines for patch testing [39].

### Literature Research and Evaluation of the Data

The literature study was performed on PubMed using the keywords: “tattoo(ing)”, “tattoo”, “pigment(ed)” combined with “cutaneous pseudolymphoma”, “cutaneous lymphoid hyperplasia”, and “cutaneous lymphoid reaction”.

A total of 43 cases of CLH reported in the literature up to 31 December 2024 were reviewed. When available, data on patient characteristics, tattoo location, time of lesion onset, clinical presentation, ink color responsible for the reaction, predominant lymphocyte population on histology, treatment, and therapeutic outcomes were recorded. Histological confirmation was a mandatory criterion for inclusion.

## Results

From the review of the literature (our cases are included), a total of 58 patients were analyzed (Table 1). Three of the patients presented reactions in more than one tattoo, bringing the total number of tattoos to 62. The study included 30 males and 28 females, with a median age of 39 years (range: 20–67 years). The average onset delay after tattooing was 33 months (median: 12 months, range: 1 month–41 years).

Tattoo location was documented in 56 of 62 cases: upper limbs (31/56, 55.4%), lower limbs (14/56, 25%), trunk (9/56, 16%), and vermilion of the lips (2/56, 3.6%). Nine cases had unknown tattoo locations. The clinical presentation included a papulonodular pattern (34/58, 58.6%), infiltrative pattern (9/58, 15.5%), and plaque-like pattern (15/58, 25.9%). Photosensitivity was reported in five cases (Figure 1 A-H). Additional symptoms were noted in 35/58 (60.3%) of cases, with itching being the most common (25/58, 43.1%). Swelling occurred in 10/58 (17.2%) cases. Red ink was the most frequently implicated (49 cases), followed by blue (5), black (3), green (3), and pink/purple (3). Five cases involved multiple ink colors.

Patch tests were performed in 28 patients, with positive results in nine cases.

Metals such as nickel and mercury showed the highest positivity rates in patch tests.

**Table 1. Main characteristics of the present series and the reported cases of tattoo-induced Cutaneous lymphoid hyperplasia (CLH) of literature on PubMed.**

Data	Variable	Value
Number of patients N		58
Number of tattoos N		62
Sex N (%)	M	30 (51.7)
	F	28 (48.3)
Median age (years) [range]		39 [20-67]
Time of onset delay after tattooing		Mean: 33 months Median: 12 months Range: 1 month–41 years
Body site localization of the tattoo N (%)	Head and neck	2 (3.6%)
	Trunk	9 (16%)
	Upper limbs	31 (55.4%)
	Lower limbs	14 (25%)
	Unknow	9 (16%)
Causative tattoo ink color N (%)	Red	49 (84.4%)
	Black	3 (5.17%)
	Blue	5 (8.62%)
	Green	3 (5.17%)
	Pink/Purple	3 (5.17%)
Clinical patterns N (%)	Papulonodular	34 (58.6%)
	Infiltrative	9 (15.5%)
	Plaque-like	15 (25.9%)
	Photosensitivity	5 (8.6%)
Symptoms N (%)	Pruritus	25 (43.1%)
	Swelling	10 (17.2%)
Histological pattern N (%)	Predominant T pattern	33 (56.9%)
	Mixed T+B pattern	18 (31.1%)
	T pattern	5 (8,6%)
	Predominant B pattern	1 (1.7%)
	Not reported	1 (1.7 %)



**Figure 1.** Clinical presentation of tattoo-induced cutaneous lymphoid hyperplasia (CLH). Papulonodular presentation (A, D, H). Nodular lesion presentation (B). Infiltrated scaling plaque presentation (C, F, G). Diffuse infiltrated plaque presentation (E). In panel A, tattoo-induced CLH arises on a black pigment tattoo, in panels B-H, tattoo-induced CLH arises on red pigment tattoos.

Histology was performed with different immunostagings in the various cases, highlighting the lymphocytes and their distribution. Immunophenotype of the lymphocytes was divided into the T pattern when the lymphocytic infiltrate was composed exclusively of T lymphocytes (CD20-) and into predominant T pattern when the lymphocytic infiltrate was predominantly composed of T lymphocytes. A mixed T+B pattern was observed when the lymphocytic infiltrate was composed of both T and B lymphocytes without an evident prevalence of either population (Figure 2 A-F). Finally, a predominant B pattern signified that the lymphocytic infiltrate was predominantly composed of B lymphocytes. In none of the cases, a pure B lymphocyte infiltrate was noted.

The predominant T pattern was the most frequently observed, reported in 33 cases; a mixed T+B pattern was observed in 18 cases, a T pattern was seen in five cases, and a predominant B pattern was present only in one case. In another one case, information regarding the lymphocyte population was not reported.

Various treatment methods reported in the literature varied across cases, often involving multiple approaches. Topical steroids (mostly clobetasol propionate) were used in 28 cases, with improvement in only 7 (Figure 3 A-B). Triamcinolone injections were administered in 11 cases, showing effectiveness in five and partial improvement in two. Topical tacrolimus 0.1% was prescribed in two cases, with one

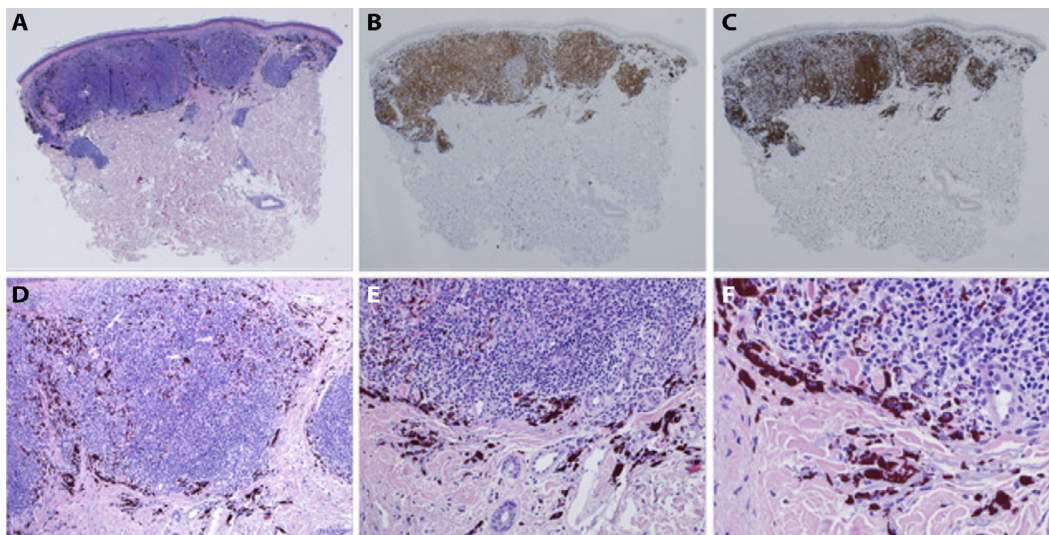
showing improvement. Surgical excision was performed in 13 cases, achieving complete resolution without recurrence. Oral prednisone was effective in one of two cases, while hydroxychloroquine (200 mg twice daily) showed no benefit in two cases. Laser treatment (Q-switched Nd:YAG laser, 595-nm pulsed dye laser, CO<sub>2</sub> laser) was used in seven patients, resulting in remission of tattoo-induced CLH in six cases (Figure 3 C-D). The ablative CO<sub>2</sub> laser treatment (10,600 nm, 18 mJ pulse energy) was performed on an infiltrated red tattoo on the calf, resulting in the subsidence of the reaction and significant flattening of the lesion. However, complete tattoo removal was not achieved even after five sessions, spaced at 1–3-month intervals. In two of the cases, laser treatment was administered simultaneously with triamcinolone acetone injections. In the remaining case, the treatment did not lead to any changes in the skin condition.

In five patients, no treatment was administered. Spontaneous remission was observed over time in three cases.

## Discussion

To the best of our knowledge, this study represents the largest case series of patients with tattoo-induced CLH and the most comprehensive review of the literature.

The global rise in tattoo popularity over the past two decades has led to an increase in tattoo-associated complications



**Figure 2.** Histological presentation of tattoo-induced cutaneous lymphoid hyperplasia (CLH) with a mixed T+B pattern. Panel A shows a 2x magnification in hematoxylin and eosin (HE) staining with a diffuse lymphocytic infiltrate in the superficial dermis associated with red pigment accumulations. Panel B displays immunohistochemical staining with positivity for CD3 (2x magnification). Panel C exhibits immunohistochemical staining with positivity for CD20. Panel D, at 10x magnification, and panel E, at 20x magnification in HE staining, demonstrate a widespread lymphocytic infiltrate and red pigment accumulations. Panel F, at 40x magnification in HE, shows scattered deposits of free red pigment and macrophages laden with pigment.

and a growing interest in their etiology and treatment. Various presentations of tattoo-induced CLH have been described in the literature. The purpose of this study was to review documented cases of tattoo-induced CLH and incorporate 15 additional cases collected by the authors to analyze their clinical and microscopic characteristics and to assess the effectiveness of different treatment methods.

The clinical presentation of CLH varies, ranging from single or multiple nodular, papular, or plaque-like lesions with varying degrees of infiltration. Erythema is a common manifestation, and itching is the most frequently reported additional symptom. The reaction can develop at different times following tattooing, though it most commonly appears several months later. There is no sex predominance according to the previous studies [8]; this is probably due to the increased popularity of tattoos among females and the likely higher use of red pigment in female tattooing. Given the clinical variability, biopsy and histological analysis remain mandatory and are currently the gold standard for diagnosis. Recently, diagnostic support techniques such as confocal microscopy and LC-OCT have been described for tattoo-induced CLH [40,41].

The exact pathogenesis of CLH remains unclear. It is hypothesized that an immunogenic pigment may induce lymphocytic proliferation, triggering sensitization and a delayed hypersensitivity reaction. The tattooing procedure itself does not seem to play a role in the development of CLH [8].

Epicutaneous testing results are inconsistent. Patch testing for tattoo-related allergies often yields negative results,

making it an unreliable diagnostic tool for pseudolymphoma. The complex composition of tattoo inks and the presence of multiple allergens complicate the identification of specific triggers. The observed positivity is difficult to interpret. In some reported cases, there is evidence of positivity to substances not present in tattoo inks, while on the other hand, metal salts, including nickel, mercury, and cobalt, have not been present in modern tattoo inks for some time [8,42]. Although tattoo needles contain significant amounts of sensitizing elements like nickel and chromium, their role in tattoo-related cutaneous reactions remains unclear [43]. According to Shubert et al., the current lack of available pigment allergens and insufficient understanding of relevant epitopes make patch testing ineffective for diagnosing tattoo hypersensitivity [44].

The phenomenon of photosensitivity reported by some patients may be explained by sunlight-induced pigment degradation and the subsequent release of reactive oxygen species (ROS) [45].

Tattoo inks, composed of organic and inorganic pigments, are deliberately implanted into the skin and may provoke chronic abnormal immune responses.

These “foreign bodies” can elicit various inflammatory reaction patterns, including granulomatous, lichenoid, or pseudolymphomatous reactions. In the granulomatous reactions, the tissue response to a foreign substance involves an accumulation of macrophages that cannot sequester a persistent indigestible material and lead to the formation of a chronic granuloma. On the contrary, the pathogenesis of



**Figure 3.** Tattoo-induced cutaneous lymphoid hyperplasia (CLH) outcome after treatment. Infiltrated scaling plaque presentation in the red part of a tattoo depicting a scorpion on the left arm before treatment (A) and after treatment with topical clobetasol propionate 0.05% twice a day for one month (B), with a good response. Infiltrated scaling plaque presentation in a red tattoo on the calf before treatment (C) and after five sessions of removal with a CO<sub>2</sub> laser (D).

pseudolymphomatous reactions remains obscure. The persistent antigenic stimulus of tattoo ink is believed to drive lymphocyte accumulation and CLH development in tattooed skin. Histopathological findings suggest that red tattoo reactions are characterized by a predominantly histiocytic infiltration with epidermal interface dermatitis, highlighting an immune mechanism mediated by type IVa and IVc hypersensitivity. These findings support the hypothesis that red pigment acts as a potent antigenic stimulus, contributing to lymphoproliferative reactions [46].

The red pigment and its various shades are known to be the predominant ink color responsible for the majority of tattoo hypersensitivity reactions. Nevertheless, little is known about the etiology of the allergy to red ink. In vitro studies have observed increased IL-18 levels following red pigment exposure, suggesting a role in chronic allergy development

[47]. The exact allergen responsible for causing the reactions to red ink has not been identified; however, some pigments seem to be more suspected than others [48]. Standard series skin patch testing in patients with tattoo reactions to red ink resulted in being ineffective in the recognition of the responsible allergen [49]. Red tattoo reactions can develop months or even years after getting a tattoo, which was also in accordance with our findings. The delay between tattooing and the appearance of the reaction supports the hypothesis of a slow allergen formation process in the skin due to enzymatic processes or photodegradation [50].

Historically, heavy metals present in ink were thought to be a culprit of tattoo allergic reactions; however, to date, organic azo dyes are the most common colorants used in tattoo inks. Nevertheless, azo dyes are increasingly acknowledged as a leading cause of hypersensitivity reactions to red tattoo

pigments, with numerous cases reported in the literature<sup>51</sup>. Bearing this in mind, chronic stimulation of the skin by an azo red pigment could be considered a potential trigger of tattoo CLH in predisposed individuals.

In addition, the study investigating the lymphatic drainages under tattoos with the use of infrared fluorescence imaging showed that alterations in the cutaneous lymphatic channels were significantly more frequent in the multicolored tattoos than in black and white tattoos. This may be explained by the deeper injection by tattooists of the colorful inks compared to black inks, which are usually injected more superficially [52]. We can assume that deeper penetration of the skin can provoke greater trauma to the tissues and, consequently, induce a more severe inflammatory response. This hypothesis, as well as the allergenic properties of the red azo pigment, may be theoretically responsible for the higher incidence of CLH in red tattoos.

From a histological perspective, CLHs are characterized by a polymorphic lymphocytic infiltrate, in the majority of cases consisting of a mixed population of T and B lymphocytes with a frequent predominance of the T subpopulation. In fewer cases, the infiltrate consists exclusively of T lymphocytes, and no case of pure B-cell infiltrates has been reported.

A single case of CLH progressing to B-cell lymphoma has been documented [53]. However, as noted by Kluger, this case appears to be isolated and atypical [53]. The clinical course of the disease is critical in distinguishing pseudolymphoma from malignant lymphoma. Indicators of malignancy include lymph node enlargement, deep skin infiltration extending into the subcutaneous tissue, a monomorphic lymphocytic pattern, and loss of cell antigens such as CD2 or CD5 [54]. Long-term follow-up is recommended, however, though only one documented case of tattoo-induced CLH progressing to lymphoma has been described, by Sanguenza et al [25].

In the absence of a previously documented pseudolymphoma, two cases of cutaneous lymphoma development in the context of tattoos have been described: one case of primary cutaneous follicular center lymphoma (PCFCL) and another of primary cutaneous marginal zone B-cell lymphoma (PCMZL). However, the authors conclude by stating that “the relatively small number of reported cases compared to the millions of tattooed individuals suggests a coincidental association [55,56]”.

A recent Danish study highlights a potential link between tattoo ink exposure and an increased risk of skin cancer and lymphoma. By analyzing data from two twin study designs—a cohort of 2,367 randomly selected twins and a case-control study of 316 twins—the researchers were able to control for confounding factors more effectively. Their findings suggest that tattooed individuals may have a 1.62 times higher risk of developing skin cancer (excluding basal

cell carcinoma), with even greater risks observed for larger tattoos. Specifically, tattoos larger than the palm of a hand were associated with a 2.37-fold increase in skin cancer risk and a 2.73-fold increase in lymphoma risk [57].

A recent case-control study conducted in Sweden, which involved 5,591 participants, sought to assess the potential link between tattoos and lymphoma, encompassing over 50 different subtypes of lymphoma. This study found that tattooed individuals had a 21% higher risk of developing lymphoma compared to those without tattoos [58]. However, it is important to interpret the results and conclusions of this study with caution. After fully adjusting for potential confounders in the matched analysis, the primary outcome of the study was not statistically significant, and no significant association was found between lymphoma and tattoos [59].

There are no established guidelines for CLH treatment, and therapeutic decisions should consider the tattoo's location, size, and the patient's preferences.

Medium-to-high-potency topical steroids can be a choice that adapts to all conditions, except for contraindications, resulting in a noninvasive treatment that preserves the tattoo. However, their effectiveness varies in the reported cases in the literature. Even in our experience, this therapy has yielded conflicting and often transient results. Intralesional triamcinolone injections have demonstrated greater efficacy, though they are limited to treating small areas and are more invasive than topical therapy.

In our centers, satisfactory results have been achieved by administering triamcinolone acetonide diluted 1:3 with saline solution directly into the lesions using the schedule of one injection every three weeks for a total of 18 weeks.

Surgical excision remains the most effective treatment, as it completely removes the affected tissue. However, this procedure alters or eliminates the tattoo, making it an undesirable option for many patients. Additionally, surgery may not be feasible for large tattoos or certain anatomical locations and is undoubtedly the most invasive option. Laser therapy is considered a valid alternative, with ablative lasers preferred over nano- and picosecond lasers due to the potential risk of triggering a systemic allergic reaction from ink particle release [60].

## Conclusion

According to our analysis, tattoo-induced pseudolymphomas are delayed cutaneous reactions that most frequently affect red tattoo pigment and exhibit a predominant T-cell histology. A biopsy is essential to distinguishing red ink hypersensitivity reactions from CLH or sarcoidosis. Although the exact incidence of CLH remains unknown, its increasing recognition suggests it may be more common than previously believed. The etiology of tattoo-induced CLH is still

unclear, necessitating further research. First-line treatments, including topical and intralesional steroids, show variable efficacy, while surgical excision remains the most reliable option when feasible.

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