Papulonecrotic tuberculid

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Abstract

Papulonecrotic tuberculid (PNT) is an uncommon form of id eruption, which occurs in association with tuberculosis infections in patients with a high degree of immunity and allergic sensitivity to mycobacterial organisms. It commonly presents as recurrent crops of papulonecrotic lesions that crust or ulcerate, and heal with atrophic varioliform scars over time. The differential diagnoses of PNT are wide and varying. Tuberculin test is usually strongly positive. Histology shows tuberculoid histology with endarteritis and thrombosis of dermal blood vessels. One of the hallmarks of PNT is its prompt response to antituberculous therapy. The purpose of this article is to increase awareness of this condition among dermatologists.

Introduction

Cutaneous tuberculosis (TB; including tuberculids) is a rare entity, constituting 0.15% of all reported cases of TB and only 1% of all extrapulmonary TB cases.1,2 Tuberculids are a heterogeneous group of skin lesions that occur in association with TB infections elsewhere in the body, including the skin, in patients with a high degree of immunity and allergic sensitivity to the mycobacterial organism. Tuberculids comprise papulonecrotic tuberculid (PNT), lichen scrofulosorum, erythema induratum–nodular vasculitis, nodular tuberculid and granulomatous phlebitis (phlebitic tuberculid).3 In this review, the pathogenesis, clinical features, differential diagnosis and management of PNT are discussed.

History

The concept of tuberculid was introduced in 1896 by Darier, as representing a form of cutaneous hypersensitivity reaction to TB antigens. Later in 1936, Pautrier established PNT as a distinct TB-associated skin entity, and described its characteristic clinical and histopathological features.4

Epidemiology

Until a few decades ago, the incidence of tuberculids had been declining in western countries; however, a recent upsurge has been noted all over the world. It has been attributed to socioeconomic factors such as overcrowding, poverty, refugee movement, increases in HIV infections and the emergence of drug-resistant Mycobacterium tuberculosis strains. Tuberculids are more common in areas with an increased prevalence of TB such as India, South Africa and Hong Kong.2 The incidence of PNT is uncommon, accounting for approximately 4% of patients with cutaneous TB. PNT chiefly affects children and young adults, with a slight female preponderance.1,2,5

Pathogenesis

PNT represents an immunological reaction to degenerated M. tuberculosis bacilli or their antigenic fragments, which are deposited in the skin and subcutaneous
tissue from occult or inapparent TB elsewhere in the body. Apart from *M. tuberculosis*, PNT-like lesions have also been associated with *Mycobacterium kansasii*, *Mycobacterium bovis* and *Mycobacterium avium* complex, and have also been reported following bacillus Calmette–Guérin (BCG) vaccination. The pathogenesis of PNT represents an Arthus reaction (Type III) accompanied by delayed-type hypersensitivity (Type IV) reaction, as illustrated in Fig. 1. 

**Clinical features**

PNT indicates good immunological status, as it is usually seen in patients with a moderate or high degree of immunity. It presents as recurrent crops of asymptomatic, symmetrical, hard, painless, dusky-red or violaceous, inflammatory papulonodular, papulopustular or pustular lesions, measuring 1–5 mm in diameter, which eventually crust or ulcerate and heal spontaneously over time, with residual pigmented changes and atrophic varioliform scars. The lesions are initially seen on the extensor aspects of the limbs and gluteal region (Fig. 2a), and eventually become widespread. Other sites that can be involved include the face, ears, penis, vulva, perineum and scalp (Fig. 2b–g). In Japan, PNT of the penis is considered a distinct entity; Nishigori *et al.* reported a series of 121 Japanese patients in whom the penis was principally involved. PNT commonly manifests as ulceration or scars. Constitutional symptoms such as pyrexia and asthenia may precede the eruption of PNT lesions.

Without treatment, spontaneous resolution of individual lesions can occur in a few weeks, with simultaneous occurrence of new lesions. Vesicular, pustular, lichenoid and umbilicated lesions, as well as verrucous variants resembling acquired perforating dermatosis, have been described. Koebnerization of PNT lesions has been reported, as has the appearance of PNT lesions at the tuberculin testing site on the arm. In patients with HIV, PNT can occur after the initiation of antiretroviral therapy, due to the improvement in immunity.

**Association**

The tuberculids often act as sentinel lesions of visceral or cutaneous TB, thus a search for overt or occult tubercular foci is warranted. An associated TB focus has been found in 38–75% of patients with PNT. It is also associated with the occurrence of other forms of cutaneous TB, such as scrofuloderma, disseminated lupus vulgaris, lichen scrofulosorum and erythema induratum of Bazin and nodular granulomatous phlebitis. Reports of transformation of PNT to lupus vulgaris and lichen scrofulosorum have been reported.

There are cases of PNT associated with erythema nodosum, limb gangrene, Takayasu arteritis, arthritis, uveitis, optic neuritis, phlyctenular conjunctivitis, discoid lupus erythematosus, leucocytoclastic vasculitis, lymphadenopathy and Hodgkin lymphoma reported in the literature.

**Diagnosis**

**Differential diagnoses**

Commonly considered differential diagnosis of PNT includes lymphomatoid papulosis, pityriasis lichenoides
et varioliformis acuta, lichen urticatus, insect bite, pruritus, prurigo, necrotizing leucocytoclastic vasculitis, suppurative folliculitis, secondary syphilis, miliary TB, infectious causes of palisade granuloma, perforating dermatoses and perforating granuloma annularis.1,3,9,27 The salient clinical and histopathological features of diseases that can mimic PNT are shown in Table 1, and the differences between the various tuberculids are shown in Table 2.

Diagnostic methods

The tuberculin test is usually positive in patients with PNT, frequently with a severe and even necrotic reaction within 8–12 h (Fig. 3a).1 However, tuberculin skin testing has disadvantages such as false-positive results in patients with prior BCG vaccination or infection with nontuberculous mycobacteria.28 Thus, a lesional biopsy should be carried out to confirm the diagnosis (Fig. 3b, c). In early lesions, vascular involvement consisting of leucocytoclastic vasculitis or lymphocytic vasculitis associated with fibrinoid necrosis and thrombotic occlusion of individual vessels are seen. Later, a wedge-shaped infarct-like lesion starts in the dermis with a large central zone of coagulation necrosis surrounded by inflammatory cells. As the wedge casts off, the epitheloid cells and giant cells collect around its periphery, even though focal granuloma formation is poor. In approximately 20% of cases, follicular necrosis or suppuration is present. Immunohistochemistry shows a predominance of T lymphocytes, along with macrophages, scanty antigen-presenting cells and an absence of B lymphocytes.1,3,5 Dermoscopy of PNT is described in Fig. 3d.

The smaller numbers of bacilli, along with the local delayed hypersensitivity reaction, the killing of bacilli upon arrival to the skin and the robust inflammation associated with PNT make it difficult to isolate acid-fast bacilli from skin biopsy specimens.4 However, Jun et al. reported finding small numbers of acid-fast bacilli in a pathological section of PNT lesion.6

The reliability of PCR as a diagnostic technique in PNT tissue samples is controversial.28 The sensitivity varies from 0 to 80% in PNT lesions.29,30 For M. tuberculosis complex, an amplified 123-bp sequence

Figure 2 (a) Papulonecrotic tuberculid of the gluteal region; (b) multiple, symmetrical, dusky-red papules with central ulceration on the back and both upper arms; (c) papulonecrotic tuberculid of the genitalia and perineum in a patient with pulmonary tuberculosis; (d) papulonecrotic tuberculid of the face in a patient with tuberculous lymphadenitis; (e) extensive areas of atrophic varioliform scars with few active papulonecrotic lesions on the back; (f) papulonecrotic tuberculid over abdomen with a positive tuberculin test.
is considered specific.\textsuperscript{16} PCR-negative PNT cases have been reported as papulonecrotic TB in the literature, and such cases call into question the categorization of this entity as a tuberculid.\textsuperscript{5} Interferon-\gamma release assay can detect latent tuberculous infection where PCR is negative.\textsuperscript{26} A recommended approach to a patient with clinical suspicion of PNT is outlined in Table 3.

### Treatment

The treatment of tuberculids involves antituberculous therapy (ATT) with a 2-month intensive phase followed by a 4-month maintenance phase. One of the hallmarks of PNT is its prompt response to ATT. The lesions usually start to clear within 3–12 weeks of initiating ATT.\textsuperscript{1} Owing to a high degree of immunity, reports of spontaneous rapid resolution of skin lesions without any ATT have been reported.\textsuperscript{31} In doubtful cases, a therapeutic trial of ATT is decisive. The use of a single drug should be avoided as it can result in recurrence.\textsuperscript{1} In instances where treatment was provided for a shorter duration and fewer drugs were used, a higher incidence of recurrence was noted. Resistant cases require antituberculous treatment for a prolonged period of time.\textsuperscript{3}

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**Table 1** Differential diagnosis of papulonecrotic tuberculid.

| Disease                        | Clinical features                                                                 | Histopathological features                                                                 |
|--------------------------------|----------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------|
| Papulonecrotic tuberculid       | Common in adults. Presents as chronic, recurrent crops of self-healing papulonecrotic lesions, predominantly affecting the trunk, which heal with fine atrophic circular or varioliform scars | Features of CD30-positive cutaneous lymphoma. Epidermis may be ulcerated. Mixed infiltrate composed of atypical lymphocytes with large nuclei and frequent abnormal mitoses, eosinophils, neutrophils and extravasated RBCs and large histiocytic cells in the dermis |
| Pityriasis lichenoides et varioliformis acuta | Polymorphous crops of eruption, which start as an oedematous pink papule that undergoes central vaculization and haemorrhagic necrosis and heals with varioliform scarring. Chieflily affects the trunk, thighs and flexural aspects of upper arms; the mucosa can also be involved. Systemic symptoms may precede or accompany the onset of the lesions. | Interface dermatitis with deep, dense and wedge-shaped predominantly perivascular lymphocytic infiltrates. Necrotic keratinocytes are common. Presence of intraepidermal and perivascular erythrocytes is typical |
| Perforating granuloma annulare  | Localized or generalized papules, which develop yellowish centres and discharge a small amount of clear, viscous fluid that later forms a crust, healing with hypopigmented or hyperpigmented scar | Superficial area of necrobiosis surrounded by palisading histiocytes situated beneath a perforation. Varying degrees of epidermal hyperplasia at the margins. Exudation of necrotic material via the perforation. Necrotizing tuberculoid granulomas with multiple acid-fast bacilli |
| Acute cutaneous miliary tuberculosis | Crops of minute blush papules, vesicles, pustules or haemorrhagic lesions. Vesicles may become necrotic to form small ulcers. More frequently seen on trunk, thighs, buttocks and genitalia. Usually seen in advanced visceral tuberculosis | Leucocytoclastic vasculitis with segmental inflammation in an angiocentric pattern, endothelial swelling, fibrinoid necrosis of vessel walls, extravasation of RBCs and neutrophilic infiltration with karyorrhexis of the nuclei |
| Leucocytoclastic necrotizing vasculitis | Palpable purpura that progresses to papules, vesicles, plaques, bullae, or pustules with secondary findings of ulceration and necrosis. Commonly seen on the legs and ankles | Thick compact orthohyperkeratosis, irregular epidermal hyperplasia, focal parakeratosis, hypergranulosis, fibrosis of papillary dermis with vertically oriented collagen fibres, increased numbers of fibroblasts and capillaries |
| Prurigo nodularis               | Pink nodule or plaque with hyperkeratotic or eroded surface and hyperpigmented border. Common on the limbs, back and gluteal region | Varying degrees of oedema and spongiosis. Mixed inflammatory cell infiltration composed of lymphocytes, eosinophilic and neutrophilic involvement around the blood vessels. Occasional RBC extravasation |
| Lichen urticatus                | Common on exposed parts of the body. Characterized by recurrent crops of weals, papules, or weals surmounted by papules, with a central haemorrhagic punctum, excoriating and crusty | Leucocytoclastic vasculitis with segmental inflammation in an angiocentric pattern, endothelial swelling, fibrinoid necrosis of vessel walls, extravasation of RBCs and neutrophilic infiltration with karyorrhexis of the nuclei |
| Acquired perforating collagenosis | Keratotic dome-shaped papules with central crust. Can develop anywhere on the body, but more frequently on extensor aspects of the limbs and trunk. Strongly linked to chronic kidney disease and diabetes | Thick compact orthohyperkeratosis, irregular epidermal hyperplasia, focal parakeratosis, hypergranulosis, fibrosis of papillary dermis with vertically oriented collagen fibres, increased numbers of fibroblasts and capillaries |
| Papulopustular syphilid         | Occur in secondary syphilis. Papulopustular lesions undergo central necrosis in debilitated patients. Lesions may be papuloid with heaped-up crusts | Plasma cell infiltrates around the blood vessels in the dermis. Tuberculoid infiltrate consisting of epitheloid giant cells |

RBC, red blood cell.
The diagnosis of PNT is often delayed due to its rarity, lack of awareness among dermatologists, wide differential diagnosis and lack of evident TB foci in patients. However, the following diagnostic criteria may help: (i) presence of characteristic skin lesions, (ii) evidence of current or past TB infection, (iii) strongly positive Mantoux test, (iv) tuberculoid histology with endarteritis and thrombosis of dermal blood vessels, and (v) good response to ATT. With the increasing trend of TB all over the world, a high suspicion of PNT should be kept in mind in any patient with papulonecrotic lesions.

### Table 2 Differences between various tuberculids.

| Features                  | Papulonecrotic tuberculid | Lichen scrofulosorum | Erythema induratum of Bazin |
|---------------------------|---------------------------|----------------------|-----------------------------|
| Described by              | Pautrier                  | Von Hebra            | Ernest Bazin                |
| Synonym                   | Tuberculosis papulonecrotica | Tuberculosis cutis lichenoides | Tuberculosis cutis indurativa |
| Age group                 | Children and young adults | Children and adolescents | Young to middle-aged adults |
| Sex predilection          | Female                    | No predilection       | Female                      |
| Cause                     | Associated with tuberculosis and BCG vaccination | Associated with tuberculosis and BCG vaccination | Associated with tuberculosis, and with infectious nontuberculous and noninfectious disorders |
| Underlying tuberculosis focus | 38–75%                   | 72%                  | Not exactly known           |
| Symptom                   | Asymptomatic              | Asymptomatic         | Tender                      |
| Clinical feature          | Recurrent crops of symmetrical, hard, inflammatory papulonodular lesions that crust or ulcerate | Discrete, flat-topped, follicular and perifollicular, usually grouped papules with fine scaling | Recurrent crops of ill-defined nodules or subcutaneous indurated plaques with scaly surface. Usually ulcerate, resulting in lesions that are ragged, irregular and shallow, with a bluish edge |
| Colour                    | Dusky-red or violaceous   | Usually skin-coloured, but may vary from erythematous to yellowish to reddish-brown | Usually violaceous |
| Clinical variants         | Vesicular, pustular, lichenoid, umbilicated, verrucous variant | Lichenoid, psoriasiform, granuloma annulare-like | Nodular tuberculid; nodular granulomatous phlebitis |
| Common sites              | Extensor aspects of the limbs and the gluteal region | Trunk, proximal limbs, gluteal region | Posterior aspects of the lower legs |
| Resolution                | Heals with atrophic varioliform scar | Heals without scar | Atrophic, hyperpigmented scarring |
| Pathology                 | Subacute lymphohistiocytic vasculitis: thrombosis and destruction of blood vessels. Wedge-shaped infarct-like lesion with central necrosis surrounded by inflammation in superficial to deep dermis | Superficial epitheloid granulomas around hair follicles and sweat ducts in the dermis | Septolobular panniculitis with primary neutrophilic vasculitis of nearby vessels |
| Differential diagnoses    | Pityriasis lichenoides et varioliformis acuta, lymphomatoid papulosis, lichen urticatus, pruigo, suppurative folliculitis, necrotizing leucocytoclastic vasculitis, perforating dermatoses, perforating granuloma annulare | Lichen nitidus, lichen spinulosus, follicular lichen planus, papular or lichenoid sarcoidosis, secondary syphilis | Erythema nodosum, pancreatic panniculitis, polyarteritis nodosa, lupus profundus, subcutaneous sarcoïd, pemphigus cutaneous T-cell lymphoma |

BCG, bacille Calmette–Guérin.

## Conclusion

The diagnosis of PNT is often delayed due to its rarity, lack of awareness among dermatologists, wide differential diagnosis and lack of evident TB foci in patients. However, the following diagnostic criteria may help: (i) presence of characteristic skin lesions, (ii) evidence of current or past TB infection, (iii) strongly positive Mantoux test, (iv) tuberculoid histology with endarteritis and thrombosis of dermal blood vessels, and (v) good response to ATT. With the increasing trend of TB all over the world, a high suspicion of PNT should be kept in mind in any patient with papulonecrotic lesions.

### Learning points

- PNT represents immunological reaction to degenerated *M. tuberculosis* bacilli or their antigenic fragments, which are deposited in the skin and subcutaneous tissue.
- PNT is usually seen in individuals with moderate or high degree of immunity.
- Clinically, PNT presents as symmetrical crops of recurrent inflammatory papulonodular, papulopustular or pustules, which ulcerate and heal with residual atrophic varioliform scars.
Figure 3 (a) Strongly positive Mantoux test in a patient with papulonecrotic tuberculid. (b) Orthokeratosis, mild spongiosis with basal cell pigmentation, and perivascular cuffing of mixed inflammatory infiltrates composed of neutrophils and lymphocytes. A few of the neutrophils can be seen infiltrating the vessel wall, along with the formation of neutrophilic debris (nuclear dust). (c) Skin with deep dermis showing epitheloid cell granuloma, Langhans giant cell, and perivascular lymphocytic infiltrate. Haematoxylin and eosin, original magnification (b,c) × 40. (d) Dermoscopy showing targetoid lesion with central ulceration with yellowish pink globules, surrounding hypopigmented rim, and outer ring of hyperpigmentation (blue arrow). Central yellow globules and white streaks with surrounding hyperpigmented halo (yellow arrow), depicting scar of tuberculid lesion (image taken with Dermlite D4 dermoscope, polarized image, original magnification × 10).
• Initially, the lesions are seen on the extensor aspects of the limbs and gluteal region, eventually becoming widespread.
• Tuberculin test is usually strongly positive.
• Leucocytoclastic vasculitis or lymphocytic vasculitis associated with fibrinoid necrosis is the early histological finding.
• PNT shows prompt response to ATT.

Table 3 Recommended approach for a patient with clinical suspicion of papulonecrotic tuberculid.

| Procedure                                                                 |
|---------------------------------------------------------------------------|
| Complete history and physical examination                                   |
| Excision biopsy taken from one of the lesions for histological examination |
| with haematoxylin and eosin                                                 |
| PCR for Mycobacterium tuberculosi DNA from formalin-fixed, paraffin-embedded specimens |
| Tuberculin skin test                                                       |
| Screening for tuberculosis focus with relevant investigations              |
| Exclusion of all other causes of cutaneous lesions with central necrosis   |
| In doubtful cases of PNT, a therapeutic trial of antituberculous therapy can be considered |

PNT, papulonecrotic tuberculid.

Conflict of interest
The authors declare that they have no conflict of interest.

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Ethics statement
Ethics approval not applicable. The patients have provided informed consent for publication of their case details and images.

Data availability
Data are available on request from the corresponding author. Data openly available in a public repository that issues datasets with DOIs.

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**CPD questions**

**Learning objective**

To gain up-to-date knowledge on the features and treatment of papulonecrotic tuberculid.

**Question 1**

Which of the following types of hypersensitivity reaction does the pathogenesis of papulonecrotic tuberculid (PNT) represent?

(a) Type 2.
(b) Type 3.
(c) Type 4.
(d) Combination of Types 2 and 3.
(e) Combination of Types 3 and 4.

**Question 2**

Which of the following describes the presentation of papulonecrotic tuberculid (PNT)?

(a) Recurrent crops of ill-defined nodules or subcutaneous indurated plaques with scaly surface.
(b) Recurrent crops of symmetrically distributed, hard, painless, dusky-red or violaceous inflammatory papules with or without central necrotic crust.
(c) Discrete, flat-topped, follicular or perifollicular grouped papules with fine scaling.
(d) Crops of minute bluish papules, vesicles, pustules or haemorrhagic lesions.
(e) Erythematous tender nodular lesions.

**Question 3**

Papulonecrotic tuberculid (PNT) heals with which of the following type of scar?

(a) Atrophic varioliform.
(b) Pock-like.
(c) Hypertrophic.
(d) Cribriform.
(e) Papyraceous.

**Question 4**

Which of the following is an early histological feature of papulonecrotic tuberculid (PNT)?

(a) Noncaseating epitheloid granulomas involving sweat glands.
(b) Superficial area of necrobiosis surrounded by palisading histiocytes.
(c) Leucocytoclastic vasculitis associated with fibrinoid necrosis.
(d) Plasma cell infiltrates around the blood vessels in the dermis.
(e) Lobular panniculitis with predominant lymphocytic infiltrates.
**Question 5**

In papulonecrotic tuberculid (PNT), which of the following statements about antituberculous therapy (ATT) is correct?

(a) Lesions start to heal within 3–12 weeks of initiating ATT.
(b) PNT is very slow to respond to ATT.
(c) PNT is recalcitrant to ATT.
(d) PNT does not require ATT owing to the high level of immunity in the patients.
(e) ATT is indicated only in severe, extensive cases.

**Instructions for answering questions**

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