CASE REPORT

A case of tuberculous gumma: there is more to it than meets the eye

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SUMMARY

We present a case of a young girl who presented with multiple cutaneous abscesses over 2 years at different sites. She had no constitutional symptoms or history of tuberculosis. On investigation, she was found to have rifampicin-sensitive tuberculosis presenting as tubercular gumma, a rare form of cutaneous tuberculosis which occurs due to haematogenous spread of the bacilli. She had disseminated disease involving the spinal column with associated psoas abscess. A thorough evaluation was done for immune-deficiency workup but was all negative. She was given antitubercular therapy and showed a good response to therapy at a follow-up of 1 month.

BACKGROUND

Tuberculosis (TB) is a common infection in our country India but its presentation can be highly variable. It may have manifestations including any system of the body. Cutaneous TB accounts for nearly 1% off all TB cases. Cutaneous TB is further divided into exogenous and endogenous. Exogenous includes inoculation of tubercle bacilli from the external environment and include TB chancre, TB verrucosa cutis (TBVC) and lupus vulgaris. Endogenous forms include lupus vulgaris, scrofuloderma, tuberculides and metastatic tubercular abscess (also known as tuberculous gumma). The order of frequency includes lupus vulgaris (55%), scrofuloderma (27%), tuberculides (6.8%), TBVC (6%) and the least common being tuberculous gumma (5.4%).¹

Skeletal and lymph node TB are other forms of extrapulmonary TB most commonly seen due to haematogenous spread of mycobacterium. A combination of these rare forms of TB without pulmonary involvement is reported very rarely and may lead to missing the diagnosis when classical constitutional symptoms may be absent. We present such a case of a young immunocompetent girl with metastatic cutaneous abscess associated with skeletal and lymph node TB.

CASE PRESENTATION

The patient was a 16-year-old girl with a history of recurrent cutaneous abscess for 2 years. It initially started in the left cubital fossa followed by the right forearm 3 months later. Later on, there was also appearance of abscesses on her foot, leg and left axilla. Two of these had been drained and patient had also received multiple courses

of antibiotics but with only partial healing with scarring. The axillary abscess ruptured and was oozing pus at which point the patient presented to our centre. She had no history of TB, fever, weight loss or other constitutional symptoms. On examination, she had tenderness in the left axilla with lymphadenopathy. The collections were fluctuant, filled with pus and warm to touch. There was no scar of BCG vaccination but records of vaccination were unavailable. Rest of the systemic examination was normal.

INVESTIGATIONS

Haematological investigations revealed a normocytic normochromic anaemia with haemoglobin of 9 g/dL. Other parameters of the complete blood count, liver and kidney function tests were all within normal limits. The axillary abscess was drained and cultures from the pus were sent to the laboratory for investigation. Gram stain was negative but acid-fast bacilli were seen on Ziehl-Neelsen staining. The pus was also positive for *Mycobacterium tuberculosis* by the method of Gene Xpert thus also confirming the species.

With the diagnosis of metastatic tubercular gumma, the patient was investigated for disseminated disease and for immune-suppressed state. The chest X-ray was normal but a contrast enhanced CT chest and abdomen revealed disseminated disease involving lymph nodes and vertebrae at levels D9–D11 (figure 1). We worked her up for immune-deficiency states, both primary and secondary. Summary of the results is provided in table 1. She was planned for evaluation of chronic granulomatous disease and Mendelian susceptibility to mycobacterial disease (MSMD) but could not be done due to cost constraints.

DIFFERENTIAL DIAGNOSIS

Our initial differentials included primary immune deficiency with recurrent bacterial infection (like staphylococcal), acquired immune deficiency or disseminated TB, deep fungal infection, non-tubercular mycobacteria.

TREATMENT

She was started on antitubercular therapy with four drugs which included isoniazid, rifampicin, pyrazinamide and ethambutol. She is being planned for at least 12 months of therapy in view of involvement of spine.

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Figure 1 Axial sections from CT scan showing (A) conglomerated lymph node in left axilla with abscess tracking up to the skin surface (marked with arrow) and (B) left psoas abscess with lytic lesion in vertebral body (marked with arrows).

OUTCOME AND FOLLOW-UP

After 1 month of therapy, there was significant benefit in the form of weight gain, increased appetite and also cessation of discharge from the axillary lesion. The other abscesses also appeared to be healing ([figure 2](#)).

DISCUSSION

Cutaneous TB is an uncommon entity and the presentation as gumma (metastatic abscess) is even rarer. The diagnosis of TB when made by Gene Xpert is highly specific and the sensitivity depends on the sample used. When aspirated pus is used, its sensitivity is well above 80% in all studies, making it a useful diagnostic modality.²

Tuberculous gumma has been reported few times, mostly as individual case reports. They are thought to occur due to haematogenous spread of the bacilli and so it is important in these cases to look actively for disseminated disease.³ In our case

Figure 2 Photographs showing response to treatment. (A) Leg and (B) axillary abscess before treatment. (C) and (D) Leg and axilla after 1 month of therapy.

too, the patient had a disseminated disease involving the bones and lymph nodes. It was surprising that there was no pulmonary involvement and no constitutional symptoms. Absence of constitutional features would be more likely in atypical mycobacteria.⁴ Skin TB is known to have >60% association with pulmonary TB. Of the reported cases, there is only one other case which has tubercular gumma with lymph node and bony involvement but in this case there was pulmonary involvement.⁵ Bony involvement is more common in extremities than in axial skeleton, like dactylitis.⁶ In our case, there was spine involvement (lytic lesions) with psoas abscess formation. In our case, the duration planned was extended to 12 months due to bony involvement.⁷

Gumma has been shown to occur in some immunocompromised patients with conditions like lymphoma, polymyositis (on immunosuppressant therapy) and immune thrombocytopenic purpura.^{8–10} We did not find any immune-suppressed condition in our patient. One factor that may have contributed to her immune-suppressed condition is her malnourished state as she had a body mass index of 15.5. The CD4 count in our case was less than normal (500–1500 cells/mm³) but it did not fulfil the criteria for CD4 lymphopenia. The explanation for this may be that TB itself may lead to low CD4 count as has been shown in previous studies.¹¹ We could not investigate for the interferon gamma and interleukin 12 pathway which when deficient or ineffective may lead to MSMD. These are a rare group of inherited immune deficiencies which predispose the patient to weakly virulent microbes including tubercle bacilli in the BCG vaccine or from the environment. They are also at a slightly higher risk of *Salmonella*, *Candida* and rarely fungal

Table 1 Summary of investigations

Tests done from pus aspirated	Immune-deficiency workup
Gram stain and bacterial culture—negative	HbA1c—5.2
KOH mount and fungal culture—negative	HIV 1 and 2—negative
Acid-fast bacilli—visualised	HbsAg and anti-HCV antibody—negative
Gene Xpert (RT-PCR) for <i>Mycobacterium tuberculosis</i> —positive and sensitive to rifampicin	Serum immunoglobulins IgA—3.52 g/L (normal) IgG—28.50 g/L (slightly elevated) IgM—1.64 g/L (normal) CD4 count—445 cells/mm ³

HCV, hepatitis C virus; HbA1c, Glycated hemoglobin; HbsAg, Surface antigen of Hepatitis B virus; RT- Real time

infections. In total, 18 different disorders are identified and nine genes have been implicated. Family history if present is a marker but is absent more commonly due to incomplete penetrance.¹² This could be a cause in our case as she presented with disseminated TB (with haematogenous spread at a young age, without any history of pulmonary TB, lacked constitutional features (which occur in response to immune activation against TB)). Possible source of infection can be BCG vaccine (which may not have left a scar) or even inoculation of environmental bacilli from epithelial breakage. In India, recent data suggest that BCG coverage was 93% in 2016 while it was 78% in 2005.¹³

Cutaneous TB responds to standard antitubercular therapy but the lesions take an average of 4–5 months to heal. Scrofuloderma lesions may take more time approximately 7–9 months. Larger abscess may also require surgical drainage.⁵

In conclusion, we want to stress that recurrent cutaneous abscesses are infrequently seen in simple bacterial infections and should raise the alarm to look for some atypical infections like fungal, atypical bacterial or drug-resistant bacteria. In endemic countries especially, it would be rationale to add TB to the list of differential diagnosis. Recurrent abscesses are also an indirect marker suggesting that the immune status of the patient is poor, so effort must be made to rule out the important

causes like diabetes and HIV first followed by primary immune deficiencies (depending on age of presentation).

Contributors SM and NS were the primary physicians looking after the patient and were responsible for collection of data and writing of the case report. AK and NW proof-read the report and gave expert advice.

Competing interests None declared.

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Learning points

- Gene Xpert has an important role to play in diagnosis of extrapulmonary tuberculosis especially where pus is involved.
- Tuberculosis is an important differential for recurrent superficial abscesses in endemic regions.
- Disseminated tuberculosis may present without cutaneous manifestations sans pulmonary manifestations even in immunocompetent patients.
- Thorough search for immune deficiency is a must in all apparently immunocompetent patients with disseminated disease.

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