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CASE STUDY

Immune thrombocytopenia secondary to tuberculosis: a case and review of literature

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SUMMARY

Immune thrombocytopenia (ITP) is an auto-immune condition that results in isolated thrombocytopenia associated with possibly lethal haemorrhage. In its secondary form, ITP can be triggered by many infectious and non-infectious conditions. Secondary ITP associated with tuberculosis (TB) has rarely been described in the literature. We report on a 22-year-old patient presenting with hypermenorrhoea and petechiae due to ITP secondary to tuberculous lymphadenitis. Normalisation of thrombocytopenia was only achieved after initiation of anti-tuberculosis treatment following failure of thrombocyte substitution and immune-modulatory treatment. A search of the literature available on TB-associated ITP identified 50 cases published between 1964 and 2016. We reviewed all cases using suggested case definitions on the likelihood of association between ITP and TB. A broad spectrum of TB sites was reported to be associated with ITP, and anti-tuberculosis treatment was the most effective therapy for platelet count normalisation. Time from initiation of anti-tuberculosis treatment to platelet count recovery ranged from 2 days to 3 months. In endemic regions, TB should be considered as an underlying cause of ITP. Early diagnosis of TB and initiation of anti-tuberculosis treatment appears crucial for rapid platelet count recovery, and can reduce the risks associated with long-term immunosuppression, transfusions and the time at risk for haemorrhage.

KEY WORDS: ITP; TB; secondary ITP

IMMUNE THROMBOCYTOPENIA (ITP) is an auto-immune condition resulting in isolated thrombocytopenia (platelet count [PC] $< 100 \times 10^3$/μl) due to various defects in immune tolerance against platelet antigens. In secondary forms, the disease is triggered by other auto-immune diseases or infections. Clinical presentation ranges from asymptomatic courses to fatal haemorrhagic diatheses.[1] Diagnosis of ITP is established by ruling out other causes of thrombocytopenia (e.g., bone marrow failure or malignancy); the detection of anti-platelet antibodies is not considered necessary.[2] Treatment aims at reducing the risk of bleeding; first-line treatment consists of corticosteroids, intravenous immunoglobulin (IVIG) or anti-D antibodies; second-line treatment may include splenectomy or thrombopoietin agonists.[1]

Tuberculosis (TB) and its treatment have been associated with a variety of haematological changes, including thrombocytosis (especially in pulmonary TB) and thrombocytopenia (associated with disseminated or miliary TB).[3] Although TB-associated 'thrombocytopenic purpura' was described in the mid-twentieth century, pathophysiological and epidemiological data are scarce.[4,5]

We report a case of ITP secondary to TB and present a literature review of the available data on TB-associated ITP. We searched PubMed (keywords 'tuberculosis' and 'ITP' without additional filters) and ProQuest (keywords 'tuberculosis' AND 'ITP'; 'tuberculosis' AND 'immune thrombocytopenia') for articles published up to May 2016. Other case reports listed in the references were also reviewed.

Table 1 Definitions of causality classification

Causality of TB and ITP	Definition
Highly suggestive	Recovery of PC ($>150 \times 10^3$/μl) not achieved by first-line ITP treatment alone, but by anti-tuberculosis treatment (either as primary therapy without immune-modulation or started after failure of ITP regimen)
Suggestive	Recovery of PC achieved by simultaneous administration of first-line ITP therapy and anti-tuberculosis treatment
Possible	Non-homogeneous group; temporal association is present, however, causality either unlikely or not very suggestive
Unlikely	Unlikely causal association

TB = tuberculosis; ITP = immune thrombocytopenia; PC = platelet count.

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Table 2 Case reports of ITP associated with TB

Country, year, reference	Cases *n*	Age years	Sex	Site of TB	TB diagnosis	Anti-platelet antibodies
Case reports with highly suggestive association of ITP with TB: recovery of platelet count (>150 × 10^3 /μl) achieved by either ATT alone or ATT after failing first-line ITP therapy						
United Kingdom, 1978[11‡]	1	24	F	Spleen	Unknown	Unknown
Saudi Arabia, 1995[31]	9	28–65[§]	[§]	Disseminated (*n* = 3), abdominal (*n* = 3), lung (*n* = 3)	Histology (*n* = 3) and AFB smear (*n* = 6)	Unknown
Spain, 1996[32]	1	70	M	Disseminated	AFB smear	Unknown
Australia, 1998[11‡]	1	74	F	Mediastinum	Unknown	Unknown
Spain, 1998[11‡]	1	27	M	Lung	Unknown	Unknown
Turkey, 2009[6]	1	4	M	Lung	AFB smear	Unknown
Turkey, 2013[9]	1	58	F	Disseminated	AFB smear	Unknown
Brazil, 2014[10]	1	69	M	Intestinal	AFB smear	Unknown
USA, 2001[11]	1	49	M	Disseminated	AFB smear	Unknown
Bangladesh, 2009[13]	1	21	M	Knee joint	Histology	Unknown
India, 2007[14]	1	8	F	Mediastinum	Clinically	Yes
China, 2007[15]	1	48	M	Lymph node	Histology	Unknown
India, 2016[20]	1	23	M	Pleura	Mycobacterial culture	Unknown
India, 2016, this study	1	22	F	Lymph node	AFB smear	No
Case reports with suggestive association of ITP with TB recovery of PC achieved by simultaneous administration of first-line ITP therapy and ATT						
Japan, 1994[11‡]	1	**	**	Lung	Unknown	**
India, 2012[7]	1	25	F	Lung	AFB smear	Unknown
Turkey, 2010[27]	1	46	M	Lung	AFB smear	Unknown
Turkey, 2004[19]	1	29	M	Lung	AFB smear	No
Iran, 2010[25]	1	17	F	Lung	AFB smear	Unknown
Japan, 2006[26]	1	22	F	Lung	AFB smear	Yes
India, 2007[29]	1	24	M	Lung	AFB smear	Unknown
Japan, 2010[28]	1	30	F	Lung	AFB smear	Yes
India, 2014[23]	1	19	F	Disseminated, including bone marrow	Histology	Unknown
India, 1986[11‡]	1	16	F	Lymph node	Unknown	Unknown
Australia, 1992[33]	1	20	M	Lung	Mycobacterial culture	Yes
India, 1993[11‡]	1	36	M	Lymph node	Unknown	Unknown

* If ATT is administered simultaneously, distinguishing not possible; poor response: PC < 50 000/μl; moderate response < 150 000/μl.
† Either direct response to ATT or sustained PC under ATT.
‡ Papers not available, data derived from Ghobrial et al.[11]
§ Ages of patients 28, 40, 42, 47, 50, 51 (*n* = 2), 55, 65 years; sex of patients: female (*n* = 7), male (*n* = 2).
¶ Pre-treatment due to liver transplantation.
# Simultaneous with ATT.
** Language barrier.
†† PC not available until 2 days after ATT initiation.
ITP = immune thrombocytopenia; TB = tuberculosis; PC = platelet count; ATT = anti-tuberculosis treatment; F = female; AFB = acid-fast bacilli; M = male; IVIG = intravenous immunoglobulin.

CASE REPORT

A 22-year-old woman presented with a history of fever, cervical swelling and fatigue for around 6 weeks and 6 days of hypermenorrhoea. On presentation the patient was afebrile and anorectic, and vital signs were normal. Enlarged, mobile, non-tender cervical, axillary and inguinal lymph nodes were palpable. Petechiae were visible on all limbs. Initial blood count showed severe thrombocytopenia (PC 7 × 10^3/μl), mild microcytic anaemia (haemoglobin [Hb] 11.4 g/dl) and normal white blood cell count. Peripheral blood smear confirmed thrombocytopenia. Coagulation, renal and liver function tests were normal. Ultrasound confirmed multiple enlarged cervical and abdominal lymph nodes.

Table 2 (continued)

Initial PC /μl	Haemorrhagic symptoms	Therapy other than ATT	PC response to therapy other than ATT*	Response to ATT†	Time from ATT initiation until PC recovery
<1 000	Unknown	Prednisone, splenectomy	No	Recovery	Unknown
4 000–21 000	Purpura ($n = 8$), menorrhagia ($n = 1$), haematuria ($n = 3$), epistaxis ($n = 1$), gum bleeding ($n = 1$)	All cases: prednisone one case: cyclophosphamide	Poor (no rise above 40 in any patient)	Recovery	2–6 weeks
8 000	Mucosal bleeding, petechiae	Methylprednisolone, IVIG	Poor (8–21)	Recovery	5 days
2 000	Unknown	Prednisolone, IVIG	Improvement	Recovery	Unknown
2 000	Unknown	Prednisolone, IVIG	Poor	Recovery	Unknown
2 000	Epistaxis, petechiae	IVIG, methylprednisolone	Poor	Recovery	1 month
6 000	Gum bleeding, petechiae, ecchymosis, epidural	IVIG	Moderate (6–100)	Recovery	Unknown
1 300	Bruises, petechiae	Tacrolimus,¶ ciclosporine, IVIG, prednisone	Poor (1.3–27)	Recovery	1 month
5 000	Epistaxis, purpura	High-dose steroids, IVIG	No	Recovery	2 months
10 000	Haemoptysis, melaena, haematuria, purpura	Dexamethasone, prednisolon#	Moderate (10–60)	Recovery	10 days
21 000	Petechiae	None	No	Recovery	10 days
6 000	Petechiae	Dexamethasone	Moderate (6–76)	Recovery	Unknown
<5 000	Haematuria, petechiae, mucosal and retinal bleeding	Methylprednisolone	No	Recovery	5 days
7 000	Menorrhagia, petechiae	Prednisolone	Poor (7–33)	Recovery	7 weeks
7 000	**	IVIG, splenectomy	Improvement	**	Unknown
36 000	Purpura	IVIG#	Recovery	Yes	3 days
4 000	Haemoptysis, purpura	Prednisolone#	Increase (to 124 by day 10, normal after 2 months)	Yes	2 months
7 600	Haemoptysis, haematuria, purpura, mucosal bleeding	IVIG,# prednisolone	Recovery	Yes	12 days
36 000	Epistaxis, petechiae, vaginal bleeding	IVIG, corticosteroids (not specified)#	Recovery	Yes	Unknown
2 000	Purpura	IVIG#	Recovery	Yes	5 days
8 000	Purpura	Prednisolone#	Revovery	Yes	1.5 months
19 000	Purpura	Methylprednisolone#	Recovery	Yes	2 months
40 000	Asymptomatic	Dexamethasone (1 day after ATT)	Recovery	Yes	3 months
29 000	Unknown	Prednisone#	Recovery	Yes	Unknown
5 000	Rectal and other mucosal bleeding	IVIG 2 days before ATT	Recovery††	Yes	2 days
46 000	Unknown	Prednisone#	Recovery	Yes	Unknown

Diagnostic workup included blood culture, urine examination and multiple tests for infectious diseases, all of which were negative. The Mantoux skin test showed 16 mm induration. No anti-platelet antibodies were detected. A bone-marrow biopsy showed neither atypical cells nor granuloma; TB polymerase chain reaction was negative. Lymph node biopsy results were pending.

Clinically relevant hypermenorrhoea (Hb 7.9 g/dl on day 3) required multiple platelet and erythrocyte transfusions transiently raising the PC to 10×10^3/μl. Oral prednisolone (1 mg/kg body weight) was started on day 6 due to suspected ITP. The next day, PC stabilised at around 30×10^3/μl and the menorrhoea decreased. Ten days later, PC was still low, at 33×10^3/μl. Lymph node biopsy yielded granuloma with caseous necrosis and acid-fast bacilli, and a diagnosis of tuberculous lymphadenitis was established. Anti-tuberculosis treatment was started and steroids were tapered off. After 3 days of anti-tuberculosis treatment, a prompt and sustained rise of PC was observed; after 7 weeks, PC had normalised to 297×10^3/μl.

LITERATURE REVIEW AND DISCUSSION

The literature search identified a total of 50 cases of TB-associated ITP published between 1964 and 2016;[4,6–34] 21 cases were summarised by Ghobrial

et al. in 2001.[11] For all cases, an association of TB and ITP was suggested by the authors. However, as in our view the likelihood of causality between TB and ITP varies, we established four case definitions of the likelihood of causality ('highly suggestive', 'suggestive', 'possible' and 'unlikely') (Table 1). We reviewed all cases based on these case definitions to only include cases with 'highly suggestive' and 'suggestive' causality in our analysis.

Of the 50 cases identified, 34 were included in our review: 22 (44%) were categorised under 'highly suggestive causality of ITP and TB' and 12 (24%) were categorised under 'suggestive causality of ITP and TB' (Table 2); 13 (26%) were categorised under 'possible association of ITP and TB',[4,8,11,16,17,21,23,24,34] and 3 (6%) were categorised under 'unlikely causality of ITP and TB'.[12,18,22]

Gender was evenly distributed (females $n = 18$, males $n = 15$, one case unknown); age ranged from 4 to 74 years (median 30, interquartile range [IQR] 22–50.5). A quarter of all cases were reported from India ($n = 8$); the remainder were reported from 10 different countries from all continents, except Africa.

Initial PC ranged between $<1000/\mu l$ and 46 000/μl (median 7800/μl, IQR 4750–18 250). Haemorrhagic symptoms were present in most cases; 27/28 cases had bleeding symptoms, with skin manifestations (petechiae/purpura/ecchymosis) being the most common ($n = 26$); mucosal bleeding and haematuria ($n = 6$) and epistaxis ($n = 4$) were also reported.

TB was microbiologically ($n = 21$) or histologically ($n = 6$) confirmed in 27/28 patients for whom the method of TB diagnosis was reported. More than a third (14/34, 41%) of patients had pulmonary TB (PTB), seven (21%) patients had disseminated TB and the remaining patients had extra-pulmonary TB (EPTB) at various sites. Our review did not show a specific TB site to be exclusively associated with ITP; however, in the majority of cases ITP was reported secondary to disseminated or EPTB.

The pathophysiological mechanisms of secondary ITP associated with TB remain unknown. Previously suggested mechanisms include production of anti-platelet antibodies and molecular mimicry in the course of regular immune response to TB.[12,28,30] The detection of antibodies against platelet antigens was only reported in four cases; in two cases, the detection of anti-platelet antibodies was attempted but was unsuccessful, and in the remaining cases no information on anti-platelet antibodies was provided. Most patients with anti-platelet antibodies had PTB ($n = 3$).

In most cases, treatment for ITP (steroids or other immune-modulating agents) was started prior to anti-tuberculosis treatment, as the diagnosis of TB was only established later. Generally only a minor increase in PC was achieved under exclusive immune-modulatory medication; therapy extension or switching to anti-tuberculosis treatment, however, resulted in a significant increase in PC. Simultaneous initiation of immune-modulatory treatment for ITP and anti-tuberculosis treatment tended to lead to a sustained and significant PC response.

The time period from initiation of anti-tuberculosis treatment to PC recovery (defined as PC $\geqslant 150 \times 10^3/\mu l$) ranged from 2 days to 3 months. However, in many cases PC was frequently not determined after initial stabilisation of PC. In 15/25 (60%) patients, PC recovery was achieved within $\leqslant 1$ month, and in a further 9 (36%) patients within $\leqslant 2$ months; in one case, PC normalisation was documented only after 3 months.

Based on the available therapeutic experience of TB-associated ITP, the beneficial effect of immune modulatory treatment remains unclear, as PC recovery has been shown to be achievable by anti-tuberculosis treatment alone.[14] However, in patients presenting with symptomatic ITP, physicians are mostly faced by the acute need for therapeutic intervention and a usually time-consuming diagnostic TB workup, especially in cases with suspected EPTB. As anti-tuberculosis treatment appears to be the most effective therapy for PC normalisation in TB-associated ITP, diagnostic workup for TB should be given high priority in endemic areas. Furthermore, if TB remains undiagnosed and anti-tuberculosis treatment is not initiated, immune-modulatory therapy may even result in the exacerbation of TB disease. Nonetheless, immune-modulatory therapy may be necessary to achieve PC normalisation.[23]

ITP secondary to TB has been considered a very rare event. It is likely that the true number of cases with ITP secondary to TB is actually higher; many cases may remain undetected, especially in asymptomatic patients and in areas without a routinely available laboratory infrastructure. To date, no prospective study investigating the prevalence of ITP secondary to TB has been conducted. The only retrospective study assessing the incidence of ITP secondary to TB was published in 1995.[31] This study, from Saudi Arabia, found that in that study setting, around 1% (9/846) of TB patients had symptomatic ITP related to TB; these nine cases constituted around 7% (9/138) of all cases of ITP diagnosed over the same period.

Our review is limited by the paucity of information available for some case reports and the non-standardised reporting. The strength of this review is that all available reports on TB-associated ITP were critically reviewed and categorised according to case definitions. To further increase our understanding of TB-associated ITP and to improve its management, more prospective and standardised data are needed; the case definitions suggested in this review may support a standardised method of reporting.

In summary, thrombocytopenia in our patient was very likely secondary to tuberculous lymphadenitis;

its main features were haemorrhagic thrombocytopenia, failure of first-line ITP treatment, prompt response to anti-tuberculosis treatment and the exclusion of other aetiologies. TB should always be considered as the underlying cause of ITP, especially in patients from endemic areas. As anti-tuberculosis treatment appears to be the most effective intervention for recovery of thrombocytopenia, rapid establishment of TB diagnosis and initiation of anti-tuberculosis treatment should be given priority. This may reduce the hazards associated with long-term immunosuppression, transfusions and the time of risk for haemorrhage.

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RESUME

La thrombocytopénie auto-immune (ITP) est une maladie auto-immune entraînant une thrombocytopénie isolée avec un risque d'hémorragie mortelle. Dans sa forme secondaire, l'ITP peut être déclenchée par de nombreux états infectieux et non infectieux. L'ITP secondaire associée à la tuberculose (TB) a rarement été décrite dans la littérature. Nous rapportons le cas d'une patiente de 22 ans se présentant avec une hyperménorrhée et des pétéchies dues à une ITP secondaire à une adénopathie tuberculeuse. La normalisation de la thrombocytopénie n'a été obtenue qu'après mise en route du traitement antituberculeux précédée d'un échec de l'administration de plaquettes de substitution et d'un traitement immunomodulateur. Nous avons ensuite recherché la littérature disponible relative à l'ITP associée à la TB et nous avons identifié 50 cas publiés entre 1964 et 2016. Nous avons revu tous les cas en utilisant les définitions de cas suggérées sur la probabilité d'une association entre ITP et TB. Une vaste gamme de sites de TB s'est avérée associée à l'ITP et le traitement antituberculeux a été le plus efficace pour normaliser le nombre des plaquettes. Le délai entre la mise en route du traitement antituberculeux et le retour à la normale des plaquettes a varié de 2 jours à 3 mois. Dans les régions d'endémie, la TB devrait être considérée comme une cause sous-jacente d'ITP. Un diagnostic précoce de la TB et la mise en route rapide du traitement antituberculeux apparaissent cruciaux pour une normalisation du nombre des plaquettes et peuvent réduire les risques associés à une immunosuppression prolongée, à des transfusions ainsi que la durée du risque d'hémorragie.

RESUMEN

La trombocitopenia inmunitaria (ITP) es una enfermedad de tipo autoinmunitario caracterizada por una trombocitopenia aislada que da lugar a hemorragias que pueden ser mortales. Muchas afecciones infecciosas y no infecciosas pueden desencadenar una trombocitopenia inmunitaria secundaria. Existen muy pocas publicaciones científicas sobre la trombocitopenia inmunitaria secundaria a la tuberculosis (TB). En el presente artículo se presenta el caso de una paciente de 22 años de edad que consultó por hipermenorrea y petequias causadas por una ITP secundaria a linfadenitis tuberculosa. La corrección de la trombocitopenia solo se logró después de haber iniciado el tratamiento antituberculoso, pues la transfusión de eritrocitos y plaquetas y el tratamiento con inmunomoduladores fueron ineficaces. También se llevó a cabo una revisión de las publicaciones científicas sobre ITP asociada con la TB y se encontraron 50 casos publicados de 1964 al 2016. Se examinaron todos los casos según las definiciones de caso recomendadas a fin de evaluar la probabilidad de la asociación de la ITP con la TB. Se encontraron informes con un amplio espectro de localizaciones de la TB asociadas con ITP y el tratamiento antituberculoso fue la estrategia terapéutica más eficaz para corregir el recuento de plaquetas. El lapso entre el comienzo del tratamiento antituberculoso y la normalización de las plaquetas osciló entre 2 días y 3 meses. En las regiones endémicas, se aconseja tener presente la TB como una causa subyacente de ITP. Un diagnóstico oportuno de la TB y la pronta iniciación del tratamiento parece fundamental, con el fin de obtener una rápida normalización del recuento de plaquetas, disminuir los riesgos asociados con la inmunodepresión a largo plazo, las transfusiones y acortar el período con riesgo de hemorragia.