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Clinical and CT characteristics of abdominal tuberculous lymphadenopathy: a comparative analysis of hematogenous and non-hematogenous dissemination

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Abstract

Objectives To elucidate the differences in clinical and CT manifestations between abdominal tuberculous lymphadenopathy (ATBL) resulting from hematogenous and non-hematogenous dissemination.

Methods A retrospective analysis was conducted on the clinical records and CT data of 178 untreated ATBL patients from January 2012 to March 2023. Patients were categorized into two groups: hematogenous dissemination (75 cases) and non-hematogenous dissemination (103 cases). The clinical characteristics of the two groups of patients were compared, and the CT imaging features of ATBL (such as location, size, and enhancement degree) were evaluated. Statistical analyses were performed using Student's t-test or Mann-Whitney U test and Chi-squared test to identify significant differences between the groups.

Results The study found that the non-hematogenous dissemination group had a higher prevalence of males, younger patients, abdominal distension, and positive tuberculin skin test (TST) results, along with higher $CD4^+$ T cell counts and lymphocyte counts. Conversely, the hematogenous group exhibited more HIV-positive patients, positive results of smear microscopy for acid-fast bacilli (AFB) staining, pleural effusion, and cough and sputum production. Significant differences were noted in the distribution, size, fusion, and enhancement patterns of ATBL between the two groups. ATBL in the hematogenous dissemination group predominantly involved the upper and lower para-aortic regions, the hepatoduodenal ligament, the portocaval space, the hepatogastric ligament, and the iliac vessels region. In contrast, the non-hematogenous dissemination group had more involvement in the mesenteric region. Lymph nodes in the hematogenous dissemination group were larger diameters, with irregular mass fusion and mixed enhancement pattern, while homogeneous enhancement was more common in the non-hematogenous dissemination group.

Conclusion There are differences in the clinical and CT manifestations of ATBL caused by hematogenous versus non-hematogenous dissemination.

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Keywords CT, Hematogenous dissemination, Non-hematogenous dissemination, Extrapulmonary tuberculosis, Lymphadenopathy

Background

Tuberculosis (TB) remains a significant challenge in global public health. In 2023, the number of newly diagnosed TB cases reported globally reached a new high, with China ranking third [1]. Factors such as HIV/AIDS, drug abuse, and the use of immunosuppressants have led to an increasing incidence rate, imposing a heavy burden on the health of the population and on society. Early detection and treatment are crucial for achieving the World Health Organization's (WHO) goal of eliminating TB [2]. TB can affect various organs throughout the body. The lungs are the most commonly affected site, and pulmonary TB (PTB) is relatively easy to diagnose. However, extrapulmonary TB (EPTB) is more challenging, with approximately 33.4% of cases presenting extrapulmonary involvement [3]. About 11–12% of EPTB occurs in the abdomen, with TB lymphadenopathy being the most common form [4]. It has an insidious onset, lacks specific clinical symptoms, and deep lymph node tissue sampling is difficult, which makes diagnosis challenging [4]. ATBL primarily results from hematogenous and non-hematogenous dissemination (gastrointestinal infection and direct spread), with the majority originating from gastrointestinal infections. However, the clinical manifestations of ATBL are non-specific and easily confused with other diseases. This can lead to frequent misdiagnoses or delays, with some cases even being mistaken for malignancies and undergoing unnecessary abdominal surgeries [5].

Diagnosis of ATBL relies on histopathological evidence, detection of AFB in lesions or ascitic fluid, or growth of mycobacterium TB (MTB) in cultures. These processes are time-consuming, with culture results taking up to six weeks [6]. In TB diagnosis and treatment, time is of the essence. Untreated or delayed treatment can result in complications, increased mortality, and heightened disease transmission risks [7]. Hence, rapid and early diagnostic methods are urgently needed to improve patient outcomes and control the spread of TB.

Currently, there are no rapid or early diagnostic methods for ATBL. Although imaging is not the gold standard for ATBL diagnosis, CT scanning is the preferred method for evaluating the extent and type of abdominal TB [6]. Its non-invasive, cost-effective, and convenient nature has made CT widely used in abdominal examinations. Because of the varying infection routes, the clinical and CT presentations of ATBL can be inconsistent. Hence, dividing patients into hematogenous and non-hematogenous dissemination groups facilitates a better understanding of the clinical and CT differences associated with these infection routes, potentially aiding early diagnosis and treatment.

Materials and methods

Data collection

This retrospective study was approved by the institutional ethics committees of the participating centers, with patient consent waived. From January 2012 to March 2023, clinical and CT data of 178 ATBL patients from three centers were reviewed. Patients who had received anti-tuberculosis treatment or were under 18 years old were excluded.

The diagnosis of 178 ATBL cases was based on the following criteria: histopathological confirmation through tissue biopsy ($n=96$), microbiological confirmation via tissue or ascitic fluid culture ($n=41$), and satisfactory clinical response to anti-tuberculosis treatment ($n=41$) [8, 9].

Currently, ATBL is recognized as having three primary transmission pathways: the most common is gastrointestinal infection (via ingestion of food or sputum containing MTB), followed by hematogenous dissemination (where tubercle bacilli spread from distant sites, usually the lungs, through the bloodstream), and direct invasion (from adjacent infected organs or tissues) [10–12].

Hematogenous dissemination TB was defined as active TB detected in at least two non-contiguous anatomical sites, miliary PTB, or isolation of MTB from blood samples [3]. Based on these criteria, patients were divided into two groups: the hematogenous dissemination group and the non-hematogenous dissemination group. Clinical and CT examination data were extracted from electronic medical records by three evaluators and compiled for further analysis.

Imaging protocol

All patients underwent abdominal CT scans using 64-slice multidetector CT devices (Revolution HD, GE Healthcare, Wisconsin, USA; LightSpeed VCT, GE Healthcare, Chicago, Illinois, USA; or Philips Incisive, Philips Medical Systems, Best, Netherlands). Before scanning, patients were routinely given 800–1000 mL of water to drink 30 min prior to the examination to distend the gastrointestinal tract, except those with gastrointestinal obstruction or suspected obstruction. For patients with mild to moderate chronic heart disease and chronic kidney disease whose cardiac and renal functions are in the compensatory stage, we have implemented an individualized water-intake adjustment plan. Their water intake was moderately reduced to 400–600 mL, and the

time for drinking water was extended from 30 min to 45–60 min. Patients in the decompensated stage should not be given water. Patients were scanned in the supine position. Non-ionic iodinated contrast agent (Visipaque 320, GE Healthcare; or Ioversol 350, Hengrui Pharmaceuticals, Lianyungang, Jiangsu, China) was administered intravenously at a dose of 1–2 mL/kg body weight and an injection rate of 3–3.5 mL/s. Three-phase enhanced scanning was performed (25–30 s, 50–60 s, and 120 s after injection), followed by data reconstruction with a slice thickness of 0.625 mm–0.5 mm.

Image analysis

Two radiologists with over eight years of experience conducted the image analysis in a double-blind manner. Lymph nodes with a short-axis diameter of ≥ 10 mm or those presenting peripheral rim, non-homogeneous enhancement patterns, or fusion were regarded as abnormal. For measurement, the lymph node with the largest short-axis diameter was selected. In cases of lesion fusion, the radiologists would attempt to identify the boundaries of individual lymph nodes for measurement. If it was impossible to distinguish, the maximum short-axis diameter of the fused mass on the axial image was recorded. Continuous data were calculated as average values, while any discrepancies in categorical data were re-evaluated by a third senior radiologist with 25 years of experience. CT features of abdominal lymph node lesions were observed, including size (short-axis diameter), anatomical location, enhancement patterns (such as peripheral rim, homogeneous, non-homogeneous, homogeneous non-enhancement or mixed enhancement) [13, 14], enhancement degree (low, iso-, high), and the presence or absence of fusion and multiplicity. Lymph node locations were categorized based on anatomical sites, including the hepatogastric ligament, hepatoduodenal ligament, portocaval space, gastro-splenic ligament, greater omentum, mesentery, peripancreatic region, upper and lower paraaortic regions (divided by the upper edge of L3), iliac vessels region, pelvic cavity, and inguinal region [12, 15]. On contrast-enhanced CT images, lymph nodes with thick, irregular, or thin rims were defined as showing peripheral enhancement. Homogeneous enhancement referred to entirely necrotic or non-necrotic nodes. Heterogeneous enhancement was defined as nodes without peripheral or homogeneous enhancement. If at least two enhancement patterns were present, the lymph node was classified as mixed enhancement [12]. Enhancement degree was assessed using skeletal muscle as a reference, as detailed in our previous publications [16].

Table 1 Demographic and clinical information of ATBL in the hematogenous dissemination group and the non - hematogenous dissemination group

Clinical characteristics	Hematogenous Dissemination group ($n=75$)	Non-hematogenous Dissemination group ($n=103$)	*P*
Age (years)	32 (24–51)	27 (22–40)	0.016
Males	40 (53.3%)	70 (68.0%)	0.047
Smoking history	21 (28.0%)	37 (35.9%)	0.265
Alcohol intake	10 (13.3%)	25 (24.3%)	0.070
Diabetes mellitus	3 (4.0%)	3 (2.9%)	0.698
Malignant tumor	2 (2.7%)	2 (1.9%)	1.000
Solid-organ or hematopoietic stem cell transplantation	1 (1.3%)	0 (0.0%)	0.421
Immunosuppressant Use	5 (6.7%)	4 (3.9%)	0.624
Chronic liver disease	13 (17.3%)	15 (14.6%)	0.616
Chronic heart disease	2 (2.7%)	2 (1.9%)	1.000
Chronic pulmonary disease	2 (2.7%)	0 (0.0%)	0.176
Chronic kidney disease	3 (4.0%)	2 (1.9%)	0.651
HIV (+)	19 (25.3%)	7 (6.8%)	0.001

ATBL, abdominal tuberculous lymphadenopathy

Statistical analysis

Statistical analyses were performed using SPSS 25.0 (SPSS Inc., Chicago, Illinois, USA). Categorical variables were expressed as numbers and percentages (%) and analyzed using the Chi-squared test or Fisher's exact test. The Kolmogorov-Smirnov test was conducted on continuous variables. Normally distributed continuous variables were expressed as mean ± standard deviation and analyzed using Student's *t*-test; non-normally distributed variables were expressed as medians and interquartile ranges (IQR) and analyzed using the Mann-Whitney U test. A two-tailed *P* value of <0.05 was considered statistically significant.

Results

Patients

Out of 244 diagnosed ATBL cases, 66 were excluded (either under 18 years old [17] or previously treated). A total of 178 patients were included, divided into the hematogenous dissemination group ($n=75$) and the non-hematogenous dissemination group ($n=103$). The median age was 29.5 years (IQR, 23.0–46.3), and 110 patients (110/178, 61.8%) were male. In the hematogenous dissemination group, the median age was 32.0 years (IQR, 24.0–51.0), with 40 males (40/75, 53.3%). In the non-hematogenous dissemination group, the median age was 27.0 years (IQR, 22.0–40.0), with 70 males (70/103, 68.0%). Male predominance was observed in both groups, and patients in the hematogenous dissemination group were older than those in the non-hematogenous dissemination group (Table 1).

Smoking and alcohol consumption were not significantly different between the groups. Similarly, no statistical differences were observed in comorbidities such as

Diabetes mellitus, malignant tumor, solid-organ or hematopoietic stem cell transplantation, immunosuppressant use, chronic liver, heart, pulmonary, or kidney diseases. However, HIV positivity was significantly higher in the hematogenous dissemination group (19/75, 25.3%) than in the non-hematogenous dissemination group (7/103, 6.8%) ($P=0.001$) (Table 1).

Abdominal pain was common in both groups (107/178, 60.1%) without statistical significance. Abdominal distension was significantly more frequent in the non-hematogenous dissemination group (45/103, 43.7%) than in the hematogenous dissemination group (20/75, 26.7%) ($P=0.020$). Fever was the most common systemic symptom (54/178, 30.3%), with no significant difference between groups. Cough and sputum production were more frequent in the hematogenous dissemination group ($P=0.043$). Five cases in the hematogenous dissemination group presented with superficial lymphadenopathy. Seven patients were asymptomatic and diagnosed incidentally. For the remaining symptoms, there was no significant difference between the two groups (Table 2).

Table 2 Clinical symptoms of ATBL in the hematogenous dissemination group and the non- hematogenous dissemination group

Clinical Symptoms	Hematogenous Dissemination group ($n=75$)	Non-hematogenous Dissemination group ($n=103$)	*P*
Asymptomatic	1 (1.3%)	6 (5.8%)	0.241
Abdominal pain	41 (54.7%)	66 (64.1%)	0.205
Abdominal distension	20 (26.7%)	45 (43.7%)	0.020
Diarrhea	5 (6.7%)	14 (13.6%)	0.140
Hematochezia	1 (1.3%)	6 (5.8%)	0.241
Constipation	0 (0.0%)	2 (1.9%)	0.510
Increased frequency of defecation	0 (0.0%)	1 (1.0%)	1.000
Rectal tenesmus	0 (0.0%)	2 (1.9%)	0.510
The changes of character of stool	1 (1.3%)	4 (3.9%)	0.399
Intestinal obstruction	0 (0.0%)	1 (1.0%)	1.000
Fever	26 (34.7%)	28 (27.2%)	0.284
Loss of appetite	18 (24.0%)	16 (15.5%)	0.156
Night sweat	10 (13.3%)	17 (16.5%)	0.560
Weak	12 (16.0%)	8 (7.8%)	0.086
Loss of weight	8 (10.7%)	5 (4.9%)	0.141
Cough and sputum production	21 (28.0%)	16 (15.5%)	0.043
Breathless	6 (8.0%)	2 (1.9%)	0.071
Chest pain	2 (2.7%)	2 (1.9%)	1.000
Tightness in the chest	0 (0.0%)	1 (1.0%)	1.000
Nausea and vomiting	3 (4.0%)	7 (6.8%)	0.522
Headache	2 (2.7%)	0 (0.0%)	0.176
Dizzy	1 (1.3%)	0 (0.0%)	0.421
Superficial lymph node enlargement	5 (6.7%)	0 (0.0%)	0.012
Abdominal mass	1 (1.3%)	1 (1.0%)	1.000

ATBL, abdominal tuberculous lymphadenopathy

Laboratory tests

In the hematogenous dissemination group, the median $CD4^+$ T cell count was 145 cells/μL (IQR, 57–304), which was significantly lower than that in the non-hematogenous dissemination group, where the median count was 269 cells/μL (IQR, 196–362.5) ($P<0.001$). Similarly, the median lymphocyte count in the hematogenous dissemination group was 0.70×10^9 cells/L (IQR, 0.42–1.31), also significantly lower than the 0.92×10^9 cells/L (IQR, 0.78–1.24) observed in the non-hematogenous dissemination group ($P=0.003$) (Table 3). A total of 172 patients underwent the TST. In the hematogenous dissemination group, 37 cases were positive (37/72, 51.4%), while in the non-hematogenous dissemination group, 66 cases were positive (66/100, 66.0%). There was no significant difference between the two groups ($P=0.054$). A total of 169 patients underwent the smear microscopy test for AFB. In the hematogenous dissemination group, 28 cases were positive (28/72, 38.9%), and in the non-hematogenous dissemination group, 13 cases were positive (13/97, 13.4%). The difference between the two groups was significant ($P<0.001$). Positive evidence of AFB was more likely to be obtained in the hematogenous dissemination group (Table 3).

CT findings

In the hematogenous dissemination group, ATBL primarily involved the mesenteric lymph nodes (81.3%), upper para-aortic lymph nodes (81.3%), and lower para-aortic lymph nodes (54.7%). The non-hematogenous dissemination group predominantly affected the mesenteric

Table 3 Laboratory tests of ATBL in the hematogenous dissemination group and non - hematogenous dissemination group

Laboratory tests	Hematogenous Dissemination group ($n=75$)	Non-hematogenous Dissemination group ($n=103$)	*P*
$CD4^+$ T cell counts (cells/μL) *	145 (57– 304)	269 (196–362.5)	< 0.001
Lymphocyte counts ($\times 10^9$ cells/L) #	0.70 (0.42–1.31)	0.92 (0.78–1.24)	0.003
Tuberculin skin test (positive) &	37 (37/72, 51.4%)	66 (66/100 66%)	0.054
Smear microscopy test for AFB (positive)$	28 (28/72, 38.9%)	13 (13/97, 13.4%)	< 0.001

ATBL, abdominal tuberculous lymphadenopathy; AFB, acid-fast bacilli; *, 55 patients were no data; #, 6 patients were no data; &, 6 patients were no data; $, 9 patients were no data

lymph nodes (92.2%). Although both groups were prone to cause mesenteric lymph node TB, it was more common in the non-hematogenous dissemination group ($P=0.029$). The hematogenous dissemination group was more likely to involve the upper and lower para-aortic regions, hepatoduodenal ligament, portocaval space, hepatogastric ligament, and iliac vessels region ($P<0.001$, $P<0.001$, $P=0.003$, $P<0.001$, $P=0.015$ and $P=0.004$, respectively) (Table 4).

Table 4 The anatomical distribution and CT findings of ATBL in the hematogenous dissemination group and the non - hematogenous dissemination group

CT characteristics	Hematogenous Dissemination group ($n=75$)	Non-hematogenous Dissemination group ($n=103$)	*P*
Location			
Mesentery	61 (81.3%)	95 (92.2%)	0.029
Hepatoduodenal ligament	29 (38.7%)	19 (18.4%)	0.003
Portacaval space	26 (34.7%)	12 (11.7%)	< 0.001
Upper para-aortic regions	61 (81.3%)	38 (36.9%)	< 0.001
Lower Para-aortic regions	41 (54.7%)	18 (17.5%)	< 0.001
Hepatogastric ligament	25 (33.3%)	18 (17.5%)	0.015
Iliac vessels region	15 (20.0%)	6 (5.8%)	0.004
Peripancreatic region	12 (16.0%)	16 (15.5%)	0.933
Gastro-splenic ligament	1 (1.3%)	2 (1.9%)	1.000
Inguinal region	1 (1.3%)	1 (1.0%)	1.000
Pelvic cavity	1 (1.3%)	0 (0.0%)	0.421
Greater omentum	4 (5.3%)	9 (8.7%)	0.389
Diameter of ATBL (cm)	2.0 (1.3–2.6)	1.5 (1.1– 2.0)	< 0.001
Multifocal lymph nodes	74 (98.7%)	102 (99.0%)	1.000
Fuse into mass	40 (53.3%)	32 (31.1%)	0.003
Enhancement pattern			
Homogeneous	29 (38.7%)	56 (54.4%)	0.038
Non-homogeneous	3 (4.0%)	5 (4.9%)	1.000
Peripheral rim	6 (8.0%)	11 (10.7%)	0.548
Homogeneous non-enhancement	0 (0.0%)	1 (1.0%)	1.000
Mixed enhancement	37 (49.3%)	30 (29.1%)	0.006
Enhancement degree			
Low density	14 (18.7%)	11 (10.7%)	0.130
Iso-density	23 (30.7%)	36 (35.0%)	0.549
High density	49 (65.3%)	67 (65.0%)	0.969
Lymph node calcification	8 (10.7%)	13 (12.6%)	0.690
Punctate	7(87.5%)	7(53.8%)	0.174
Nodular	6(75.0%)	10(76.9%)	1.000
Patchy	2(25.0%)	2(15.4%)	0.618
Peripheral	1(12.5%)	2(15.4%)	1.000
Pleural effusion	37 (49.3%)	29 (28.2%)	0.004
Ascites	41 (54.7%)	63 (61.2%)	0.385
Intestinal tuberculosis	27 (36.0%)	62 (60.2%)	0.001

ATBL, abdominal tuberculous lymphadenopathy

The median short-axis diameter of lymph nodes was 2.0 cm (IQR, 1.3–2.6) in the hematogenous dissemination group and 1.5 cm (IQR, 1.1–2.0) in the non-hematogenous dissemination group, with significantly larger lymph nodes in the former ($P<0.001$). ATBL exhibited five enhancement patterns (peripheral rim, homogeneous, non-homogeneous, homogeneous non-enhancement or mixed enhancement). Both groups commonly showed mixed and homogeneous enhancement, but homogeneous enhancement was more common in the non-hematogenous dissemination group ($P=0.038$) (Fig. 1). Irregular mass-like fusion of TB lymph nodes was more common in the hematogenous dissemination group ($P=0.003$) (Fig. 2). There was no significant difference between the two groups in terms of the degree of lymph node enhancement (low, iso-, or high enhancement) (Table 4).

Both groups often had concomitant pleural effusion and ascites, with pleural effusion being more common in the hematogenous dissemination group ($P=0.004$), while intestinal TB was more frequently associated with the non-hematogenous dissemination group ($P=0.001$). Lymph node calcification was observed in 21 cases, with no significant difference between the two groups. Lymph node calcifications in both groups appeared as multiple lesions, with specific patterns including punctate, nodular, patchy, and peripheral calcifications. Both groups predominantly exhibited punctate and nodular calcifications, no significant difference in calcification patterns between the two groups ($P>0.05$) (Table 4).

Discussion

In this study, we found that the clinical and CT findings of ATBL can differ between patients with hematogenous dissemination and those with non-hematogenous dissemination. These findings aid in the early diagnosis of ATBL.

Our results showed a significant tendency for non-hematogenous dissemination in males and younger individuals. This may be due to more frequent social activities and exposure to risk factors in young males, leading to increased risk of non-hematogenous dissemination of tubercle bacilli. This is consistent with the research of Kang W. et al., with a male-to-female ratio of 1.51, and the highest proportions of EPTB are found in the15-24 and 25–34 age groups [18]. However, some studies report a higher prevalence of EPTB in females, still predominantly in younger individuals [3, 19]. Gender differences may be related to sample selection and regional variations.

Abdominal pain was the most common symptom in ATBL patients (60.1%), followed by abdominal distension

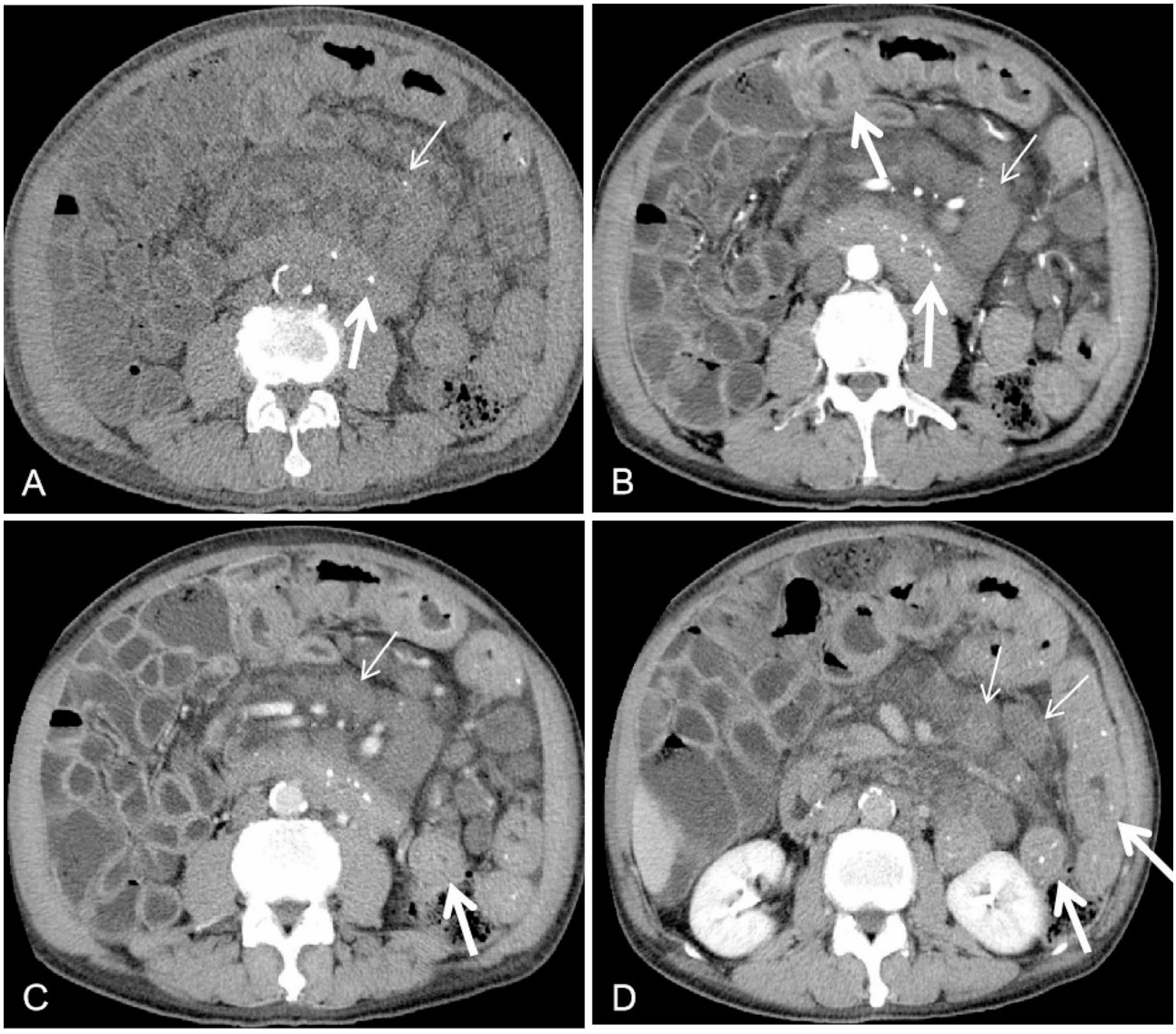

Fig. 1 A patient with non-hematogenous disseminated ATBL combined with small bowel tuberculosis had a 6 - month history of abdominal distension. Axial CT plain scan images showed multiple enlarged lymph nodes in the mesentery, some of which were calcified (thin white arrow), and the wall of the small intestine was thickened and calcified (thick white arrow) (**A**). Axial contrast - enhanced images showed that multiple enlarged mesenteric lymph nodes had homogeneous low enhancement (thin white arrows), and the thickened small intestinal wall had homogeneous enhancement (thick white arrows) (**B, C, D**)

(36.5%), fever (30.3%), as well as cough and sputum production (20.8%). These findings are consistent with previous studies, indicating that abdominal pain, abdominal distension and fever are common clinical manifestations of ATBL [9, 10, 20, 21]. Tubercle bacilli directly affect gastrointestinal function after gastrointestinal infection, leading to more frequent abdominal distension in the non-hematogenous group. The lungs, being the origin or main organ affected in hematogenous dissemination, result in more frequent cough and sputum symptoms in this group. Furthermore, both groups had asymptomatic patients, totaling 7 cases.

Among the 178 ATBL patients, 26 cases (14.6%) were HIV positive. Further analysis revealed that the HIV positivity rate was 25.3% in the hematogenous dissemination group and 6.8% in the non-hematogenous dissemination group, suggesting that HIV infection may increase the risk of developing ATBL, with HIV-positive patients being more likely to have hematogenous dissemination. Previous studies have strongly supported this finding, suggesting that HIV infection severely damages the immune system, which greatly increases the risk of opportunistic infections, including ATBL [20, 22–24]. Compared to the general population, individuals with

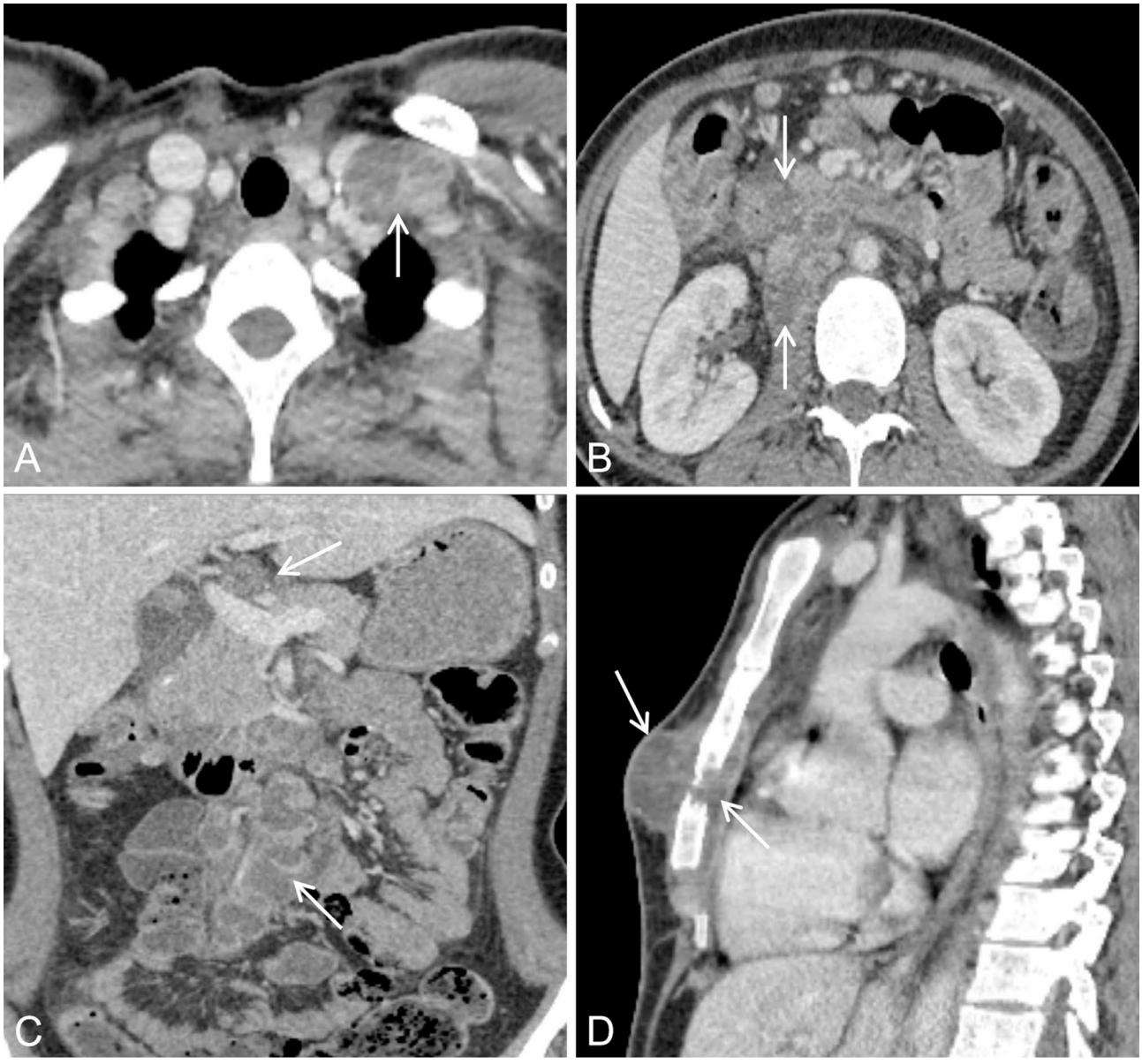

Fig. 2 A patient with hematogenous disseminated ATBL suffered from upper abdominal pain and weight loss for 1 month. Axial contrast - enhanced CT demonstrated that the enlarged lymph nodes in the left neck presented with peripheral rim enhancement (white arrow) (**A**). Axial and sagittal contrast - enhanced CT images revealed multiple enlarged lymph nodes in the mesentery, para - aortic area, and hepatogastric ligament. These lymph nodes showed ring - shaped enhancement and were fused together (white arrow) (**B, C**). The sagittal contrast - enhanced CT image showed destruction of the sternum's bone, accompanied by the formation of a cold abscess in the chest wall (white arrow) (**D**)

HIV have a much higher incidence of EPTB, and their conditions are often more complicated and severe.

The non-hematogenous group had higher $CD4^+$ T cell counts and lymphocyte counts, likely due to the higher proportion of HIV-positive patients in the hematogenous group. HIV infection leads to immune system damage, particularly a reduction in $CD4^+$ T cell counts, affecting overall immune function. The hematogenous group exhibited a higher rate of positive smear microscopy for AFB and pleural effusion, which may be related to the frequent presence of active PTB in this group. The active proliferation and dissemination of MTB in the lungs may lead to a higher detection rate of AFB in sputum smears and increase the risk of pleural effusion, which is consistent with the pathophysiology of hematogenous dissemination TB.

Hematogenous dissemination ATBL lesions were more common in the mesenteric, upper and lower para-aortic regions, hepatoduodenal ligament, portocaval space, hepatogastric ligament, and iliac vessels region, which is consistent with previous research [12]. The hematogenous dissemination of MTB can lead to involvement

of multiple systems and sites throughout the body, thus resulting in a wide distribution. Non-hematogenous disseminated ATBL typically stems from gastrointestinal infections. MTB often invades through the gastrointestinal mucosa, commonly in the ileocecal region [25]. Subsequently, the bacteria spread to the mesenteric lymph nodes (the first station of intestinal lymphatic drainage) along the lymphatic drainage pathway. Therefore, non-hematogenous disseminated ATBL is more concentrated in the mesenteric region, which is closely related to the lymphatic drainage pathway and consistent with previous research [9]. ATBL commonly affects multiple lymph nodes, with only two cases being solitary in our study, which can mimic tumor-like changes, making clinical diagnosis very difficult [6, 11].

The larger lymph nodes and irregular mass-like fusion observed in the hematogenous dissemination group could be due to the massive entry of MTB into the bloodstream during hematogenous dissemination. These bacteria affect multiple sites simultaneously, triggering a severe inflammatory response. HIV infection severely compromises the immune system, particularly by reducing $CD4^+$ T cell counts, which are crucial for maintaining effective immune responses against MTB [26]. After MTB breached the lymphatic barrier, it proliferated rapidly. All these factors contributed to the rapid enlargement and fusion of lymph nodes, which is consistent with the findings of Li Y et al. [12]. In contrast, lymph nodes in the non-hematogenous dissemination group were relatively smaller and more homogeneously enhanced, possibly due to the more localized lymphatic drainage pathway after gastrointestinal infection with TB, with a milder and more concentrated inflammatory response. Intestinal-associated lymphoid tissues (such as macrophages) rapidly activate the immune response, forming compact granulomas that encapsulate MTB and limit its spread.

Overall, most ATBL lymph nodes are less than 4 cm in diameter, consistent with previous literature [12, 14]. Regarding lymph node calcification, some studies report no calcification [10, 12], while Deshpande SS, et al. [9] reported calcification in 26.6% (16/60) of cases. In our study, 11.8% (21/178) had multiple calcifications, indicating different stages of the disease process, with calcification being a strong indicator of TB in lymph nodes. No significant differences were found between the two groups in terms of the incidence and patterns of calcification. Therefore, we consider the calcification features to be similar in both groups. Interestingly, our study predominantly showed homogeneous enhancement, especially in the non-hematogenous group, followed by mixed enhancement, which differs from previous findings of peripheral enhancement being more common [11, 14, 15, 21]. Further validation with larger samples is needed.

Limitations

This study has several limitations. Firstly, this study is retrospective, relying on historical medical records, which may have missing information. For example, there were data missing in terms of $CD4^+$ T cell counts, lymphocyte counts, TST, and smear microscopy test for AFB. The most significant data loss was observed in $CD4^+$ T cell counts, with a total of 55 cases missing (55/178, 30.90%). This data gap may lead to biases in the conclusions regarding immune status differences between the two groups. Future studies should consider prospective designs. Secondly, the sample size was relatively small, which restricts the generalizability of the conclusions. Additionally, 26 HIV-positive patients were included, which may have influenced the results. Thirdly, accurately determining the route of infection in clinical practice can be challenging, and there may be complex interactions or co-existence between different routes of infection. Finally, the lack of long-term follow-up data prevented us from gaining deeper insights into the natural course of the disease, treatment outcomes, and potential long-term complications.

Conclusions

In summary, significant differences were observed between the two groups in demographic and clinical information, clinical manifestations, laboratory findings, and CT features of ATBL. For young male patients presenting with abdominal pain and distension, and CT showing multiple enlarged lymph nodes in the mesenteric region with homogeneous or mixed enhancement, take into account the possibility of TB. Further evaluation of intestinal involvement can be beneficial for confirming the diagnosis. If a patient presents with not only abdominal pain and distension but also cough and sputum production or superficial lymph node enlargement, and CT reveals multiple enlarged lymph nodes in various abdominal regions that are fused into mass with homogeneous or mixed enhancement, the possibility of hematogenous disseminated TB is likely. Timely screening for PTB foci is advisable. It is worth noting that once multiple calcifications are found in abdominal lymph nodes on CT images, the possibility of ATBL is highly suspected. This sign has important implications for clinical diagnosis. Understanding these differences in ATBL caused by hematogenous and non-hematogenous dissemination can aid in early diagnosis and treatment, reducing complications and mortality, and better achieving TB control and prevention goals.

Abbreviations

TB	Tuberculosis
MTB	Mycobacterium TB
PTB	Pulmonary TB
EPTB	Extrapulmonary TB
ATBL	Abdominal tuberculous lymphadenopathy
AFB	Acid-fast bacilli
TST	Tuberculin skin test

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Author contributions
GW and SL: conceptualization. XZ, XL and LW: data curation. XL and RL: investigation. GW and SL: methodology. XZ: writing-original draft.

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Data availability
Data is provided within the manuscript or supplementary information files. The dataset generated and analyzed during the current study is not publicly available as it contains protected health information, but the de-identified dataset is available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The studies involving human participants were reviewed and approved by Medical Ethics Committee of Banan Hospital of Chongqing Medical University (BNLL-KY-2023-036), Chongqing Public Health Medical Center (GWZX-2022-122603) and Xinqiao Hospital of Third Military Medical University (2023011401). The committees waived informed consent because the study was retrospective, there was no risk of harm to subjects, and all patients were anonymous. All methods were performed in accordance with relevant guidelines and regulations, and the study was conducted in accordance with the Declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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