Atypical presentation of acquired tracheo-oesophageal fistula in an adolescent girl with pulmonary tuberculosis

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SUMMARY

We report a case of an adolescent girl presenting with acute respiratory distress syndrome (ARDS) requiring mechanical ventilation. Initial presentation during the ongoing COVID-19 pandemic was compatible with multisystem inflammatory response in children associated with COVID-19 (MIS-C). Subsequently a diagnosis of tuberculosis was made. During ventilation, she developed significant abdominal distension which was not relieved with nasogastric decompression. There was a high index of suspicion of bronchoenteric fistula. Bronchoscopy with adjunct oesophagoscopy demonstrated tracheo-oesophageal fistula (TEF). The classical presentation of TEF has been masked by onset of ARDS. During the pandemic the diagnosis of tuberculosis in high-burden countries decreased for multiple reasons leading to development of complications which are often confused with MIS-C. While diagnosing MIS-C, maintaining a high level of suspicion for concomitant or alternative aetiologies is essential.

BACKGROUND

Development of tracheo-oesophageal fistula TEF) is a very rare complication following primary pulmonary tuberculosis and only few cases have been reported so far.[1–3] After the onset of the COVID-19 pandemic, development of multisystem inflammatory syndrome in children (MIS-C) has not only exaggerated the severity of pre-existing illnesses and masked their typical presentation but also mimicked various endemic infections like dengue, tuberculosis and scrub typhus.[4 5] Here we report a case of complicated tuberculosis which was initially confused with MIS-C. This case emphasised the importance of broad differential diagnoses and well-planned laboratory investigations for the timely diagnosis and management of such cases during the ongoing pandemic.

CASE PRESENTATION

An adolescent girl presented with high-grade continuous fever for 5 days and acute onset of breathlessness. She presented with undocumented low-grade intermittent fever, chronic cough, decreased appetite and undocumented weight loss for the past 2 months. Fever was not associated with chills, rigours, night sweats, sore throat, chest pain, rashes, bleeding, eruptions, joint pain, burning micturition, heaviness in abdomen or abdominal distension. Cough was non-productive, had no diurnal variation and was not associated with any aggravating or relieving factors. The child was fully immunised and had a BCG scar on her left arm. There was no history of significant medical illness or sick contacts. She lived in a dormitory as a student at a religious school. She was the youngest of four siblings. Her siblings were of good health. Her father ran his own business and mother was a homemaker. There was no history of consanguinity.

On examination, she was conscious, oriented and had respiratory distress. Her temperature was 38°C, heart rate 120 beats/min with low pulse volume, respiratory rate 40 breaths/min, blood pressure 80/54 mm Hg and saturation 76% in room air. Her weight was 33 kg with a body mass index of 14.09 kg/m^2.

Respiratory examination showed fine crepitations bilaterally. Her heart sounds were normal, and abdomen was mildly distended with no localised tenderness. There was no generalised lymphadenopathy, rash or oedema.

INVESTIGATIONS

Her initial laboratory parameters showed combined respiratory and metabolic acidosis (pH=7.12, HCO_3 (bicarbonate level)=18 mmol/L, pCO(Partial pressure of carbon dioxide)=47 mm Hg, pO_2(partial pressure of oxygen)=58 mm Hg, lactate=2.1 mmol/L) raised C reactive protein (CRP=1.2 mg/dL), transaminitis (aspartate aminotransferase=163 IU/L) with hypoalbuminaemia (table 1). Chest radiograph revealed bilateral diffuse inhomogeneous opacities (figure 1) suggestive of acute respiratory distress syndrome (ARDS). At admission she was diagnosed with ARDS with probable septic shock. She was given bilevel positive airway pressure (BiPAP) mode of non-invasive ventilation (NIV). Following initial fluid bolus, inotropes and broad-spectrum antibiotics were initiated. Despite NIV her respiratory distress worsened, and she developed gross abdominal distension which increased even after nasogastric tube drainage. She was intubated and mechanically ventilated. Initial ventilatory settings: mode=assisted volume controlled ventilation, tidal volume= 6 mL/kg of body weight, peak end expiratory pressure=9 cm H_2O, FiO_2(fraction of inspired oxygen)=0.6, Inspiratory time was set to provide inverse ratio ventilation (I: E=1:1) and target inspiratory plateau pressure limit of 28 cm of H_2O. Further laboratory investigations showed low platelet counts and increased inflammatory markers with a positive serology test for COVID-19 (table 1). The WHO

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Table 1 Relevant laboratory investigations

Laboratory parameters	On admission	Day 3, Day 4	Day 8
Haemoglobin(g/dL)	12.7	12.2	13.4
TLC (1000/mm^3)	8.2×10^3	6.9×10^3	4.55×10^3
DLC (polymorphs (P), lymphocytes (L) Monocytes(M) in %)	P75 L22 M3	P79 L18	P64 L21
Platelet counts (100 000/mm^3)	1.2	0.45	1.52
Blood urea (mg/dL); normal range :17–43 mg/dL	32.1	33	13.1
Creatinine (mg/dL); normal range :0.67–1.17	0.24	0.23	0.08
AST (IU/L); normal range: <50 IU/L	163		96
ALT (IU/L) Normal range: <50 IU/L	40		40
Albumin (g/dl); normal range: 3.5–5.2 g/dL	2.3	1.8	2.4
CRP (mg/dl); normal; <0.6 mg/dL	1.2	2.4, 4.8	<0.6
Serum ferritin (ng/ml); normal range:12–140 ng/mL	1275	1601	–
D-dimer (FEU/L); normal range: <0.5 FEU/L	3.32	20.92	–
Prothrombin time (PT) (seconds) Reference range : PT control=13 s	16.9		–
INR	1.26		–
Blood culture	–	Sterile	–
COVID-19 RT-PCR	Negative	Negative	–
SARS-CoV-2 antibody (AU/ml) (reference range: <12.0: negative, 12.0–15.0: equivocal, >15.0: positive)	–	38.0	–

ALT, alanine aminotransferase; AST, aspartate aminotransferase; AU/ml, absorbance units per millilitre; CRP, C reactive protein; DLC, differential leucocyte count; FEU/L, fibrinogen equivalent unit per litre; INR, international normalised ratio; RT-PCR, reverse transcriptase PCR; TLC, total leucocyte count.

and CDC criteria for MIS-C was fulfilled: fever>3 days, hypotension, raised inflammatory markers (CRP, D-dimer, ferritin), hypoalbuminaemia along with positive SARS-CoV-2 serology.[6 7] A working diagnosis of MIS-C was made, and intravenous dexamethasone was started accordingly. Her nasopharyngeal swab for COVID-19 reverse transcription PCR (RT-PCR) was negative on the first day; the next two consecutive tracheal aspirate samples were also negative for COVID-19. Blood and urine cultures were sterile. Her echocardiography was normal. As per the National Tuberculosis Elimination Programme (NTEP), tuberculosis workup was carried out.[8] Cartridge-based nucleic acid amplification test (CBNAAT) demonstrated rifampicin-sensitive *Mycobacterium tuberculosis* in tracheal aspirates. As per NTEP guidelines, nucleic acid amplification test (NAAT) is performed in every patient where a biological specimen can be procured. If a specimen is positive it is labelled as microbiologically confirmed tuberculosis (TB) and definitive diagnosis of

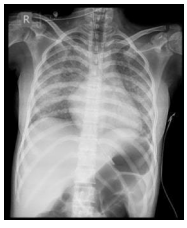


Figure 1 Chest X-ray showing bilateral diffuse inhomogeneous opacities.

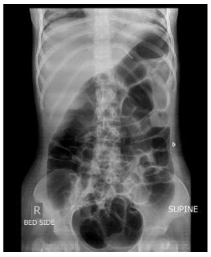


Figure 2 X-ray abdomen showing gaseous distension of bowel loops.

tuberculosis is made. Whereas in cases of negative NAAT with strong clinical suspicion, one aliquot of the sample is sent for liquid culture.

Therefore further samples were not sent for liquid culture. Her tracheal aspirate culture was negative for bacterial and fungal growth.

During ventilation she had persistent abdominal distension despite continuous nasogastric tube drainage (figure 2). X-ray abdomen revealed gaseous distension of the stomach and small bowel loops. On initiating tube feeds air bubbles were observed in the syringe synchronous with the positive pressure breaths (figure 3, video 1). A high index of suspicion of bronchoenteric fistula led to further investigations. Contrast-enhanced CT (CECT) chest showed diffuse ground glass haziness with interposed smooth interlobular septal thickening involving bilateral lung fields with mild bilateral pleural effusion with no significant mediastinal and hilar lymphadenopathy suggestive of ARDS (figure 4); a possible communication between the right bronchus and oesophagus was also suspected. Upper gastrointestinal endoscopy (UGIE) showed no fistula or any evidence of oesophageal tuberculosis.

Bronchoscopy was not done as it was not available at our centre. She was gradually weaned off and extubated on day 5 of ventilation and given BiPAP mode of NIV for the next 2 days. She responded well to a treatment that included antitubercular antibiotics (see the Treatment section). The level of her inflammatory markers improved (table 1). She developed paroxysmal cough following ingestion of solids and liquids. Bronchoscopy with a repeat UGIE was performed at another centre. A fistulous opening just right to the carina communicating with oesophagus with air bubbles was seen (figure 5 (still from video), video 2). Histopathology report of biopsy samples showed granulomatous inflammation consistent with tuberculosis. A final diagnosis of pulmonary tuberculosis with acquired TEF was made though the exact underlying mechanism remain unanswered in the absence

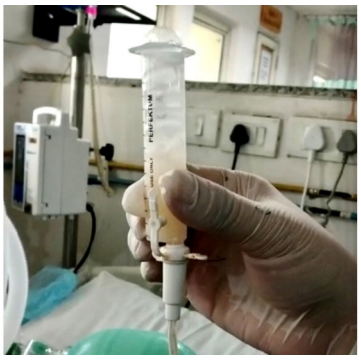
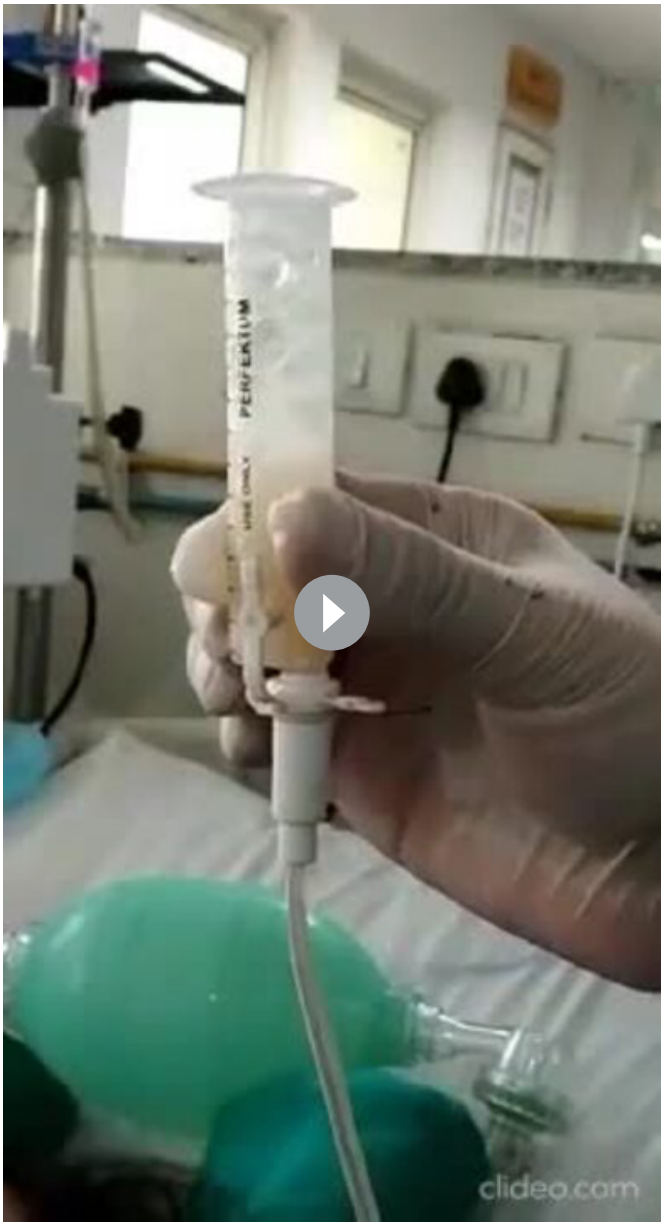


Figure 3 Bubbles observed in nasogastric tube with delivery of positive pressure breaths.



Video 1 Bubbles observed in nasogastric tube with delivery of positive pressure breaths.

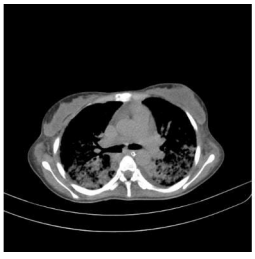


Figure 4 Contrast-enhanced CT (CECT) chest showed diffuse ground glass haziness with interposed smooth interlobular septal thickening involving bilateral lung fields with mild bilateral pleural effusion with no significant mediastinal and hilar lymphadenopathy suggestive of acute respiratory distress syndrome (ARDS).

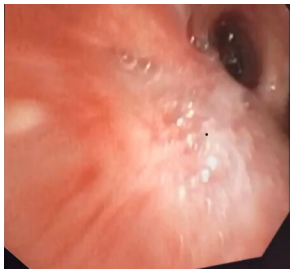
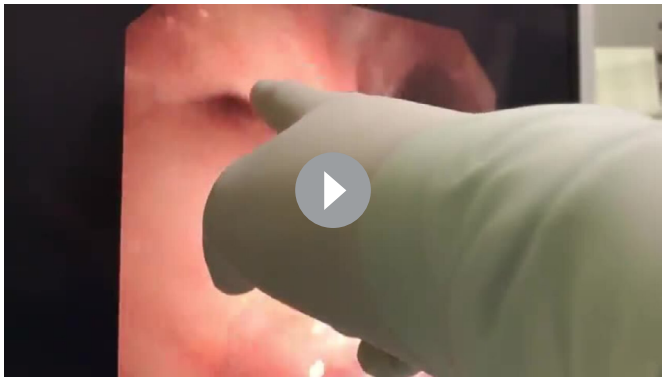


Figure 5 Bronchoscopy with adjunctive oesophagoscopy showing a fistulous opening just right to the carina communicating with oesophagus, with air bubbles.

of any evidence of oesophageal tuberculosis on UGIE and mediastinal lymphadenopathy in CECT.

DIFFERENTIAL DIAGNOSIS

The initial impression was ARDS with haemodynamic shock of probable septic origin. Her workup for SARS-CoV-2 infection was significant. In the presence of fever, hypotension, respiratory system involvement, raised inflammatory markers with positive COVID-19 −19 serology and negative RT-PCR, the patient was also suspected to have MIS-C. Though undocumented, there was a history of intermittent fever for 2 months. This raises the suspicion of tuberculosis in an endemic country. With a positive tracheal aspirate CBNAAT, tuberculosis was confirmed and four-drug antituberculosis therapy (ATT) was started. Further investigations did not support influenza or mycoplasma infection. She responded well to the four-drug antitubercular drugs. Her respiratory parameters improved on ventilation but abdominal distension and the presence of air bubbles in the nasogastric tube during enteral feeding persisted. X-ray abdomen ruled out digestive tract perforation. Her abdominal distension decreased after extubation, and initial endoscopy failed to identify any bronchoenteric fistula. Post extubation when allowed orally, she developed bouts of cough following oral ingestion of fluids. On asking direct questions she recalled having episodes of cough while drinking for the past 1 month which was denied earlier by the attendants. Subsequently bronchoscopy with simultaneous oesophagoscopy clinched the diagnosis of TEF. A review of the history ruled out congenital TEF, trauma, ingestion of caustic materials and foreign body inhalation. She did not have any relevant medical or surgical history since her neonatal period.



Video 2 Bronchoscopy with adjunctive oesophagoscopy showing a fistulous opening just right of the carina communicating with the oesophagus.

An iatrogenic cause was ruled out because the patient had a history of cough while drinking before admission; she also developed significant abdominal distension while on NIV mode, and a nasogastric tube was inserted later. The history of fever, chronic cough, coughing while drinking, weight loss, overcrowding, detection of *M tuberculosis* in tracheal aspirates, and histopathological evidence of granulomatous lesion along with good response to antituberculosis drug therapy with closure of fistula in repeat endoscopy make tuberculosis a confirmatory diagnosis. But underlying mechanisms leading to fistula formation could not be explained in absence of mediastinal lymphadenopathy, mediastinal air and oesophageal tuberculosis.

A long-standing tuberculosis infection of 2 months or more could lead to an acquired TEF. She was a boarding student and along with the pandemic restrictions, early diagnosis of tuberculosis was missed. The positive COVID-19 serology may be anticipated based on the high community prevalence, and is not necessarily indicative of MIS-C, particularly as an alternative cause for the presentation has been identified.

TREATMENT

On admission she required two doses of fluid resuscitation with 20 mL/kg of 0.9% normal saline. Inotrope support (epinephrine infusion at 0.3 µcg/kg/min) was started. Broad spectrum antibiotics meropenem (60 mg/kg/day) and vancomycin (40 mg/kg/day) were administered She required ventilatory support for 7 days (5 days of mechanical ventilation followed by 2 days of NIV). With the main differential diagnosis of MIS-C she was given intravenous methyl prednisolone (30 mg/kg). Intravenous immunoglobulin could not be given due to financial constraints. She received subcutaneous enoxaparin (0.5 mg/kg) twice daily. Four-drug ATT including isoniazid, rifampicin, pyrazinamide and ethambutol was started. She remained on nasogastric feeding throughout her hospital stay. Paediatric surgeons advised conservative management for TEF and close monitoring with a follow-up UGIE. She was discharged on weaning dose of prednisolone, ATT and continued nasogastric feeding

OUTCOME AND FOLLOW-UP

She was discharged from hospital after 2 months of initial presentation. She continued her ATT as per the NTEP regimen and remained on nasogastric feeding. She showed signs of improvement and well-being. Follow-up UGIE 5 months after treatment revealed a healed TEF. She was then given oral feeds, which she tolerated well.

DISCUSSION

Healthcare systems worldwide are challenged by COVID-19 and essential healthcare delivery has been restricted. The healthcare infrastructure is pivoted towards COVID-19 and away from illnesses like tuberculosis. In countries with high tuberculosis burden like India the diagnosis and treatment has decreased. Access to healthcare is constrained due to disruption in transport during lockdowns, reduced opening hours, fear and stigma. The vulnerable population of children and adolescents with tuberculosis tend to be neglected in the COVID-19 responses around the globe.[9] With well-documented difficulties in diagnosing paediatric tuberculosis, the effect of tuberculosis during the COVID-19 pandemic can be severe. In a systemic review and meta-analysis by Gao *et al* tuberculosis was associated with a 2.10-fold increase in risk of severe COVID-19 disease, though not statistically significant.[10] A tubercular infection impairs the lung function increasing susceptibility to SARS- CoV-2 infection and can lead to ARDS. MIS-C is a postinfective delayed antibody-mediated dysregulated immune response occurring 4–6 weeks after initial infection.[11] To understand the exact interaction between these two infections, a global study coordinated by Global Tuberculosis Network and supported by WHO is ongoing.[12] Preliminary results cannot rule out or confirm the hypothesis that COVID-19 contributes to the pathogenesis of tuberculosis.[13] It is now known that dysregulated immune response by both the infections can augment the severity of each other.[14] In our patient coexistence of both infections may have increased the inflammation and TB progression resulting in TEF but positive COVID-19 serology may also be an incidental finding and nothing confirmatory can be extrapolated with the available evidence.

A high index of suspicion is required for a diagnosis of acquired TEF. Paroxysmal attacks of coughing on ingestion of fluids is known as Ono's sign and is considered pathognomic and the most common presentation of acquired TEF. Other symptoms which may be seen are dysphagia and recurrent pneumonia which may progress to life-threatening conditions. In our case, a similar history was not retrieved from her attendants and the patient presented with severe respiratory distress and was mechanically ventilated the same day. The clue to diagnosis was development of significant abdominal distension on BiPAP and ventilator with intermittent bubbling observed through nasogastric tube on delivery of positive pressures. Most of the acquired TEF in children are benign and are caused by infectious processes or trauma.[15–19] Among infectious causes, granulomatous inflammation of the mediastinal lymph node is the the most common and majority of them are tuberculous. They are more commonly seen in immunocompromised patients.[20 21] Devarbhavi *et al* have suggested that oesophageal tuberculosis is associated with mediastinal lymphadenopathy in all the cases, along with oesophagotracheal fistula or oesophagomediastinal sinus in half of them.[22] Oesophagoscopy didn't reveal any evidence of oesophageal tuberculosis in our patient and therefore oesophageal tuberculosis as a cause of TEF has been ruled out. Also, the location of fistula and development of abdominal distension on NIV ruled out iatrogenic causes as neither nasogastric tube nor nasotracheal/endotracheal tube was inserted till the patient developed abdominal distension on NIV. Various mechanisms have been suggested to explain the formation of TEFin pulmonary tuberculosis. One is mediastinal lymphadenopathy with inflammation of the surrounding structure leading to formation of abscess whose rupture results in formation of fistula. Another possible mechanism is formation of fibrous scar with traction diverticulum of oesophagus and subsequent fistula formation at the tip.[23 24] Barium swallow and multislice CT with increased resolution are very useful for the diagnosis.

Erlank *et al* studied the radioimaging findings of acquired oesophageal perforation secondary to primary pulmonary tuberculous lymphadenopathy in children. They showed the leakage of contrast medium on contrast swallow, large low-density lymph nodes on CT and mediastinal air as the most common finding whereas Im *et al* described the presence of perioesophageal gas in patients with tuberculous mediastinal lymphadenitis suggestive of oesophageal fistula.[25 26] The CT findings in our patient with and without contrast swallow failed to demonstrate any of the abovementioned features which led us to proceed for bronchoscopy which is pivotal in identifying the fistulas with oesophagoscopy as an adjunct.

The treatment of acquired non-malignant TEF depends on multiple factors like aetiology, extent of tissue damage and involvement of surrounding structures. TEFs caused as a complication to endotracheal tubes or tracheostomy tubes require surgical repair in a majority of the patients whereas tuberculous fistulas have been treated successfully with ATT.[27–29] Our patient who was critically ill with a tormentous course during her illness also responded gradually to ATT.

Patient's perspective

Patient: When my condition became very bad, my father and brothers arranged an ambulance to take me to the hospital. After visiting about four hospitals, I was finally admitted. Due to the corona pandemic, it was difficult to get hospital admission. The doctors and nurses in the hospital took very good care of me. I later came to know that I was unconscious for 6 days and was on a ventilator. My brother took a photograph of my unconscious state and after seeing that I feel thankful to be alive. I had tuberculosis along with Corona virus infection. I remember one evening when I was asked to take oral feeds and I coughed continuously after that. I could not sleep the entire night, I felt scared that I might be put on the ventilator again. I had immense faith in the doctors and the Almighty. I prayed a lot during my hospital stay. I was told that there is a connection between my food pipe and windpipe, and I will have to continue feeding through the tube. I was very depressed at the thought of not having to taste my biryani and kheer. But I kept praying to God and took all the medicines prescribed. I never missed any dose. I was discharged from hospital after 2 months.

My family and extended family supported and encouraged me. I did not feel awkward with a tube stuck in my nose. After 5 months I was very happy when the doctor permitted me to eat orally. I am thankful to the Almighty and the medical staff for saving my life.

Learning points

- The symptoms and findings of multisystem inflammatory syndrome in children (MIS-C) can overlap with other non-SARS-CoV-2 infections. Clinicians should be aware of this and have a broad differential diagnosis while treating children with MIS-C.
- While treating COVID-19, primarily during the pandemic, one should keep in mind one of the 'great mimickers' tuberculosis as the differential diagnosis. The case demonstrates that delay in diagnosis of tuberculosis during the pandemic restrictions can increase the severity of the disease and thereby the morbidity.
- Possibilities of rare complications of tuberculosis, such as acquired TEF in our case, should be thoroughly investigated.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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