

Unraveling the Complexities of Wilkie's Syndrome in the Context of Pulmonary Tuberculosis. A Case Report

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Abstract

Case Report

This case report describes a 35-year-old female undergoing treatment for pulmonary tuberculosis who developed persistent bilious vomiting and abdominal pain. Despite the temporary pause of anti-tuberculosis therapy due to hepatotoxicity, her symptoms persisted. Imaging exams revealed aorto-mesenteric compression syndrome (AMCS), a rare cause of duodenal obstruction resulting from compression of the duodenum between the aorta and the superior mesenteric artery, commonly associated with rapid weight loss. This case underscores the importance of considering AMCS in patients with refractory gastrointestinal symptoms and significant weight loss.

Keywords: Aorto-mesenteric compression syndrome, Superior mesenteric artery, Duodenal obstruction, Rapid weight loss, Bilious vomiting.

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1. INTRODUCTION

Vomiting can be a side effect of taking anti-tuberculosis treatments, but it may also be associated with aorto-mesenteric compression syndrome (AMCS), or Wilkie's syndrome, which is a rare cause of duodenal obstruction (Merrett ND, 2009). This syndrome results from the compression of the third segment of the duodenum between the aorta and the superior mesenteric artery (SMA) (Shukla RC, 2009).

The symptoms related to this duodenal obstruction are consistent with high intestinal obstruction. The most common cause is rapid weight loss, leading to a reduction in the thickness of the adipose tissue in the aorto-mesenteric space (Wilkie DPD 1927, Shukla RC 2009). Other contributing factors may also be involved.

The initial treatment is medical, but if unsuccessful, surgical intervention becomes necessary (Singal AK, 2001).

2. CASE REPORT

We report the case of a 35-year-old female patient with a history of chronic smoking and occasional alcohol use, who was being treated for bacteriologically confirmed pulmonary tuberculosis and had been started on anti-tuberculosis treatment.

The course of the disease was marked by the onset of bilious vomiting, abdominal pain, and general deterioration one month after the start of the treatment. The temporary pause of anti-tuberculosis treatment was decided due to severe hepatic toxicity. After normalization of liver function tests, the anti-tuberculosis treatment was reintroduced. However, despite the gradual reintroduction of the treatment, uncontrollable vomiting persisted, leading to the decision to stop the treatment and conduct further etiological investigations due to the persistence of vomiting.

Clinical examination revealed an oxygen saturation of 95% in ambient air, a WHO performance status of 4, cachexia (35 kg, 167 cm, BMI 12.5), and hepatomegaly.

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A thoraco-abdominal CT scan showed a miliary pattern with no abnormalities in the abdominal region. An enteroscopy revealed inflammatory thickening of the

cecal base. An esophagogastroduodenoscopy revealed erythematous antritis, erosive bulbitis, and thinning of the duodenal folds with signs of external compression.

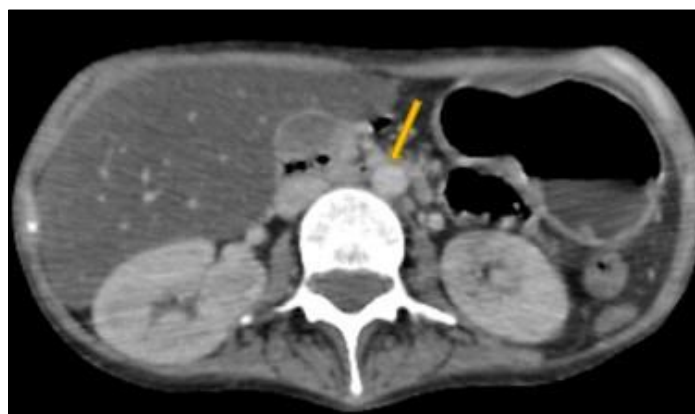


Figure 1: Abdominal CT showing the aorto-mesenteric compression (yellow arrow)

Biological tests showed cholestasis, electrolyte imbalance with hypokalemia and hyponatremia, and a biological inflammatory syndrome.

The progression was marked by the persistence of digestive symptoms despite the cessation of anti-tuberculosis treatment and administration of Ondansetron. Consequently, a second contrast-enhanced abdominal CT scan, coupled with water ingestion, revealed an aorto-SMA distance of 5 mm and an aorto-SMA angle of 11° , indicating aorto-mesenteric compression responsible for external compression of the third part of the duodenum. Duodenal bulb thickening was observed, which was regular, circumferential, and enhanced after contrast injection, measuring 4 mm in thickness over 19 mm. Additionally, there was thickening of the adjacent peritoneal layer and a comb-like appearance of the mesentery.

Unfortunately, the patient passed away before receiving a digestive bypass.

3. DISCUSSION

The aorto-mesenteric compression syndrome (Wilkie's syndrome) is a rare condition, with an incidence ranging from 0.013% to 0.3% in the general population (Welsch T, 2007). This condition primarily affects young adults, with a predominance in women, often due to rapid weight loss or eating disorders (Neri S, 2005). The sex ratio is estimated to be 3:1 (Lee TH, 2003). Risk factors include extreme thinness, history of abdominal surgeries, or rapid weight loss, as observed in our patient suffering from tuberculosis (Wilkie DPD, 1927).

The clinical manifestations of Wilkie's syndrome are non-specific and include: postprandial bile vomiting (80-90% of cases) (Merrett ND, 2009), localized abdominal pain especially in the epigastrium, exacerbated by meals (Morimoto N, 2018), and

significant weight loss, present in more than 90% of cases, as observed in our patient (Cazelles JL, 2002). More rarely, symptoms may include early satiety, belching, and sometimes signs of dehydration (Lippl FJ, 2002).

The syndrome is often associated with rapid weight loss, leading to a decrease in the fatty mass surrounding the mesentery, resulting in a reduction of the aorto-mesenteric angle (Shukla RC, 2009). In our case, uncontrollable vomiting and severe cachexia (BMI 12.5) led to a late diagnosis.

The positive diagnosis mainly relies on imaging exams:

- Abdominal CT with water ingestion: This is the gold standard for assessing the aorto-mesenteric distance (< 8 mm) and the aorto-AMS angle ($< 22^\circ$) (Unal B, 2005). In our patient, the angle was 11° with a distance of 5 mm, which is pathological and confirms the diagnosis of Wilkie's syndrome.
- Esophagogastroduodenoscopy: Useful for excluding other causes of obstruction. The signs of erythematous antritis and extrinsic compression observed in our patient are compatible with duodenal obstruction (Shariat N, 2017).

Biological tests revealed cholestasis and an electrolyte imbalance (hypokalemia, hyponatremia), common in Wilkie's syndrome due to prolonged vomiting and malnutrition (Daniel WW, 2016).

Management depends on the severity of symptoms:

- Conservative treatment: Includes rehydration, correction of electrolyte imbalances, and high-calorie feeding (Sarathchandra V, 2006). Positioning in left lateral decubitus or knee-chest position is recommended to reduce compression (Shariat N, 2017). In our patient,

severe cachectic status limited the efficacy of these measures.

- Medical treatment: Use of prokinetics and antiemetics such as Ondansetron. These medications had limited effect in our case, reflecting the severity of the obstruction (Morimoto N, 2015).
- Surgical treatment: Duodenojejunostomy is indicated in cases of conservative treatment failure (Singal AK, 2001). It allows bypassing the compressed area. Delays in surgical intervention and severe hypokalemia contributed to the fatal outcome in our case.

Studies show that conservative treatment is effective in about 70% of patients. In cases of failure, duodenojejunostomy improves symptoms in more than 80% of cases (Cazelles JL, 2002).

The prognosis of Wilkie's syndrome depends on the speed of diagnosis and management. Complications include severe malnutrition, hypovolemia, and secondary infections (Lippl FJ, 2002). The mortality rate is generally low (< 10%) with appropriate management but increases in the presence of comorbidities or diagnostic delays (Lippl FJ, 2002).

In the context of our patient, active pulmonary tuberculosis and severe cachexia contributed to rapid progression and deterioration of general condition, which limited therapeutic options and led to death. Previous studies have shown that patients with rapid weight loss and low physiological reserve, such as tuberculosis patients, have a higher risk of complications (Unal B, 2005).

4. CONCLUSION

Aorto-mesenteric compression syndrome remains a diagnostic challenge, particularly in complex clinical contexts. Our case highlights the need for increased vigilance in patients presenting with rapid weight loss and unexplained gastrointestinal symptoms. Early and multidisciplinary management is essential to improve prognosis. Further studies are needed to guide treatment in contexts of severe comorbidities such as tuberculosis.

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