

4. Yaghoobi R, Khazanee A, Bagherani N, Tajalli M. Gastrointestinal tuberculosis with anal and perianal involvement misdiagnosed as Crohn' disease for 15 years. *Acta DermVenereol.* 2010;91:348–9.
5. Oliveira Leonardo Guedes Leite de, PupoNeto João de Aguiar, Vieira Eduardo de Paula, Kim Monika Pereira, Flach Luciana da Costa, Almeida Barbara Cristina Rodrigues de. Proposed tuberculosis investigation and management protocol in complex and recurrent fistula-in-ano. *J. Coloproctol. (Rio J.)* 2015;35(2):113–119.

## Celiac Disease Presenting as Pericardial Effusion

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Celiac disease is a unique enteropathic immune disorder and is now considered a disease entity with protean manifestation and worldwide distribution<sup>1</sup>. Since the immunologic component is paramount in pathogenesis of celiac disease serum IgA antigliadin, antiendomysial and anti tissue transglutaminase antibodies provide a novel, sensitive and specific tool for the diagnosis of celiac disease<sup>2,3</sup>. Deposition of Immune complexes originating in small-bowel could be a possible reason, for extra intestinal autoimmune manifestations of celiac disease<sup>4</sup>. Pericardial effusion though, rare in adults, is probably a result of these autoimmune disorders related to celiac disease<sup>5</sup>.

## Case Report

An 18 year old previously healthy female with no history of systemic illness presented to us with chief complaints of sudden onset progressive breathlessness for eight days, followed by decreased urine output for three days. There was no preceding history of fever, hematuria, oedema, bleeding diathesis, jaundice, chest pain, cough or skin rash. There was no history of weight loss or decreased appetite. On examination patient was conscious but restless. She was afebrile with low volume regular pulse rate of 100/min and blood pressure of 60/40 mmHg. The patient had pallor, tachypnea, her JVP was raised and she had B/L pitting pedal oedema. However there was no icterus, cyanosis, clubbing, or thyromegaly. Systemic examination was suggestive of hepatomegaly of 2 cm below the subcostal margin. On cardiovascular examination the heart sounds were muffled. The respiratory system examination was normal. Laboratory investigations revealed Hb-7.5, TLC of 6600 with differential count of 55% neutrophils, 50% lymphocytes 2% eosinophils and 3% monocytes. ESR was 28 during 1st hour. Peripheral smear and iron studies were suggestive of iron deficiency anaemia. Blood urea was 50 mg/dl, serum creatinine was 1.4 mg/dl. Her serum total bilirubin was 0.8, AST 55IU, ALT 27IU and ALP 120IU. Her total serum protein was 3.2 mg/dl and albumin fraction was 1.6 mg/dl. TSH was 4.7, serum calcium 8.5 mg/dl and phosphorus 4.4 mg/dl. Urine routine and microscopic examination was normal and 24 hour urine protein was 140 mg. X ray chest was suggestive of cardiomegaly. 2D Echocardiography revealed. Large Concentric pericardial effusion of maximum thickness of 2.7 cm with mild TR and Severe PAH with EF of 60%. There was no evidence of cardiac tamponade. A provisional diagnosis of anaemia with pericardial effusion with hypotension and Acute kidney injury was made. After therapeutic pericardiocentesis and inotropic support, clinical condition of the patient improved. Her renal function and blood pressure became normal. Pericardial fluid analysis revealed a cell count of 26 of which 70% were lymphocytes, a sugar level of 140 mg/dl. Protein was absent in pericardial fluid. TB – PCR in pericardial fluid was negative. ANA, RA factor was

negative. Ultrasound of abdomen showed a mild increased in liver echotexture with prominent hepatic veins. CECT Chest and abdomen revealed smooth inter and intralobular septal thickening in bilateral lungs with pericardial effusion and pulmonary hypertension and congestive hepatomegaly. Patient was further evaluated for causes of anaemia and pericardial effusion. Thyroid disorder, Tuberculosis, malignancy and autoimmune causes were scrupulously ruled out. In view of iron deficiency anaemia and altered liver echotexture an upper GI endoscopy was undertaken which revealed normal oesophagus, stomach, and first part of duodenum. Biopsy was taken from D2 segment of small intestine which showed scalloping of folds during the upper GI endoscopy. Biopsy from D2 segment of small intestine showed moderate to severe villous blunting with preservation of overlying mucosal epithelium, along with increased Intraepithelial lymphocyte count and crypt hyperplasia. Lamina propria showed mild lymphoplasmacytic infiltrate. The findings were consistent with celiac disease which was further corroborated by increased tissue transglutaminase levels (>800 IU/ml)

## Discussion

The clinical spectrum of celiac disease is extensive and atypical presentation is commonly seen in paediatric age group, however atypical extra intestinal manifestation are a rarity in adult patients. Pericardial effusion is commonly found in children with celiac disease and often presents with asymptomatic and limited pericardial effusion on echocardiography.<sup>5</sup>

The coexistence of pericardial effusion could be attributed to molecular mimicry by which gliadin or tissue transglutaminase activates T cells that cross-reacts with various self-antigens. Transglutaminase can modify external or self antigens by cross-linking or by deamidation, thus generating neoantigens.<sup>5</sup>

Better diagnostic facilities have led to increase in diagnosis of latent and atypical celiac disease. This case further enhances our knowledge regarding the myriad and varied presentation of celiac disease.

## References

1. Rostom A, Murray JA, Kagnoff MF. American Gastroenterological Association (AGA) Institute technical review on the diagnosis and management of celiac disease. *Gastroenterology*. 2006;131(6):1981–2002.
2. Hunt KA, Zhernakova A, Turner G, et al. Newly identified genetic risk variants for celiac disease related to the immune response. *Nat Genet*. 2008;40(4):395–402.
3. Goldstein NS, Underhill J. Morphologic features suggestive of gluten sensitivity in architecturally normal duodenal biopsy specimens. *Am J Clin Pathol*. 2001;116(1):63–71.
4. C. Grasso, C. Mattia, M. Spina, P. Sciacca: Pericardial effusion in celiac disease. *Journal of Medicine and Medical Sciences* Vol. 3(2) , 2012
5. Fasano A. Clinical presentation of celiac disease in the pediatric population. *Gastroenterology*. 2005;128(4 Suppl 1):S68–S73.

## An Unusual Cause of Abdominal Lump: Omental Torsion with Infarction

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The greater omentum is a long peritoneal fold which is in continuity with the visceral fold of peritoneum of stomach as well as transverse colon. It plays a pivotal role in containing the spread of intraperitoneal inflammation. Infarction of the omentum is a rare etiology