abdominal ultrasound was normal. *C. difficile* and *C. Tropicalis* were found in stool cultures. 2 consecutive sweat tests were performed, showing borderline values: 61,25 mmol/l; 67,7 mmol/l. There was no polyuria or excessive chloride loss in urine, which are typical features of Bartter's syndrome. Diarrhea was not present at birth, no watery stools were observed and the failure to thrive was not significant prior to admission, making the diagnosis of congenital chloride diarrhea unlikely. Genetic testing for CF showed that the infant was a delF508 mutation and G178r mutation compound heterozygote.

The child showed no clinical symptoms of hypokalemia. No hyponatremia seizures were observed. Diarrhea was persistent. Rehydration therapy and antibiotic treatment significantly improved the condition.

Patients who are compound heterozygous for the milder CFTR mutations (classes IV and V) experience partial CFTR function, resulting in atypical features such as borderline sweat chloride concentrations, unlike our patient who is a compound heterozygous with class II and III mutation, where high amount of chloride in sweat is more commonly seen. Such patients are more likely to experience severe CF. Therefore making early diagnosis is crucial to further normal growth and development as therapy could be started early in life.

P82 IN PURSUIT OF A WANDERING SPLEEN

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Introduction Acute abdominal pain in the paediatric population is a common and generally benign, self-limiting condition. The challenge is to identify a minority of life threatening presentations. We describe young female presenting with diffuse abdominal pain, fever and vomiting.

Case presentation A previously well 4 year old female presented with sudden onset of severe central abdominal pain, vomiting and fever. Pain was colicky and responded to analgesia. Clinical examination did not yield an aetiology. Initial working diagnoses of acute viral illness with mesenteric adenitis and subsequently acute appendicitis following second presentation. C-reactive protein (CRP) was moderately raised, WBC 15×10^9 and haemoglobin low at 10.5 g/dL.

Representation prompted diagnostic laparoscopy.

Laparoscopic examination revealed an enlarged spleen, displaced across the midline. The patient was discharged home well on day 2 postoperatively with resolving symptoms. CT abdomen imaging subsequently showed torsion of a grossly enlarged spleen with widespread necrosis. The patient underwent splenectomy.

Discussion Splenic torsion is rare in children and is described in a condition referred to as wandering spleen (1). Wandering spleen results from the abnormal development and absence of the normal splenic suspensory ligaments, resulting in an untethered organ predisposing to rotation and torsion of a large organ on a small vascular pedicle (1,2).

Sometimes asymptomatic, the symptom complex may be one of recurrent acute episodes of pain relating to spontaneous twisting and untwisting of the mobile spleen.

Wandering spleen has two peaks of incidence the first in infancy with a male female ratio 2:1. Medical conditions complicated by splenic enlargement, including infectious mononucleosis and metabolic storage disorders predispose individual to splenic torsion in at risk groups (1,2).

Conclusion Although rare, splenic torsion is an important differential diagnosis in patients presenting with acute abdomen of unclear aetiology. Prompt consideration should be given to this Diagnosis on account of the associated life threatening complication. Conservative management with preservation of the organ and splenoplexy is the treatment approach of choice but splenectomy is frequently necessary (2). Mortality risk relates chiefly to post-operative infection. Patients who undergo splenectomy should be offered vaccination against encapsulated organisms (*Pneumococcus*, *H.Influenzae* and *Meningococcus*), and oral penicillin prophylaxis.

REFERENCES

1. Alimoglu O, Sahin M, Akdag M. Torsion of a wandering spleen presenting with acute abdomen: a case report. *Acta Chir Belg* 2004;104:221-23.
2. Yakan S, Telciler KE, Denedi AG. Acute torsion of a wandering spleen causing acute abdomen. *Hong Kong J Emerg Med* 2011;18:34-36.

P83 EVANS SYNDROME WITH ABDOMINAL DISCOMFORT: A CASE REPORT

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Background A 15 year old boy, a known case of Evans Syndrome with a feeling of discomfort in his abdomen.

Case report 15 year old boy was presented to emergency department with a feeling of discomfort in his abdomen from last 3 weeks. He also felt a lump on right side of his abdomen. There was no associated history of fever, weight loss, poor appetite, constipation, vomiting or night sweats. On examination boy had a firm rounded mass of about 10x15 cm extending from umbilicus to right hypochondrium with irregular surface and no tenderness. Serum LDH was raised 2200 IU/ml at admission. Abdominal Ultrasound revealed large lobulated heterogeneous hyper vascular mass in right iliac fossa. CT was requested that demonstrated the presence of large lobulated heterogeneous mass involving caecum, ascending colon and associated hepatic flexure confirming the diagnosis of proximal large bowel lymphoma with accompanying extensive intra peritoneal and retro peritoneal lymph nodes. It also showed lobulated lymph node mass of 8.5x6.5 cm in retro vesicular recess. Mediastinal and axillary lymph nodes were noted on CT thorax. Histopathological examination confirmed the diagnosis of high grade B-cell lymphoma. Regarding past history he was diagnosed as Evans syndrome at the age of 3 years, resistant to treatment and splenectomy was done at the age of 7 years. No significant family history of note. He was treated with 4 courses of chemotherapeutic agents and disease was cured.

Discussion Evans syndrome is a rare autoimmune disorder with no known genetic basis. It usually presents with coombs positive hemolytic anemia with thrombocytopenia. Because of its autoimmune nature it exhibits signs of lymphoid hyperplasia with increased risk of lymphoreticular malignancies. Prognosis for B cell lymphoma is usually good as it was in our case.