LETTER TO THE EDITOR

Superficial abdominal thrombophlebitis (Mondor’s disease) presenting as loss of response to adalimumab in a Crohn’s disease patient☆

Dear Editor,

Multiple etiologies should be considered in Crohn’s disease (CD) patients experiencing worsening symptoms while treated with anti-TNFs.1 We describe an unusual case of seeming loss of response to adalimumab.

A 39 year-old man was hospitalized due to worsening right lower abdominal pain lasting for three days prior to presentation. The patient was inflicted by fibro-stenotic Crohn’s ileo-colitis for the last three years. He has been treated during the last two years by adalimumab 40 mg bi-weekly, and 6-mercaptopurine at 1.3 mg/kg. The patient underwent four endoscopic balloon dilations of 2 colonic strictures during the course of his disease, but has enjoyed near complete remission in the prior eleven months with a Harvey–Bradshaw index of 4 at his last clinic visit. Upon his admission to the surgical department he was in pain but afebrile and abdominal palpation revealed moderate tenderness in his right lower quadrant. C-reactive protein level was normal. Antibiotic treatment was initiated intravenously, and abdominal sonographic examination was performed revealing normal appendix and no evidence of fluid collection or thickened small bowel loops. Adalimumab drug level was adequate at 5.6 mcg/ml and no antibodies-to-adalimumab were detected. The patient was discharged from the hospital three days later with presumed diagnosis of CD exacerbation and referred to one of us (SBH) for gastroenterology consultation. Upon his clinic visit he was still in pain and there was tenderness at the right lower quadrant. However, a palpable superficial cord-like structure was also appreciated by gentle palpation and by squeezing the abdominal wall (Fig. 1A). He was referred for abdominal wall-focused sonography, which revealed superficial subcutaneous vein thrombo-phlebitis in the right lower abdominal wall, corresponding to Mondor’s disease (Fig. 1B & C). Analgesics and low-molecular-weight-heparin for six weeks were prescribed and the patient’s symptoms gradually resolved within the next two weeks. Hyper-coagulability work-up was negative.

Patients treated with anti-TNFs can experience loss of response to the drug due to re-emergent inflammation. However, it is pertinent to exclude other causes unrelated to relapsing CD inflammatory activity, before designating the patient as experiencing anti-TNF drug failure. Mondor’s disease is a rare disorder of superficial vein thrombosis (thrombophlebitis) of the thoraco-abdominal wall, which can be either idiopathic or associated with diverse underlying disorders.2 To the best of our knowledge, this is the first description of Mondor’s disease in CD. This is somewhat striking given the predisposition of CD patients to thrombotic events.3 However, the absence of prior reports in CD may echo the unsettled controversy pertaining to the question of whether Mondor’s disease constitutes a genuine thrombotic event associated with classical risk-factors for thrombosis. Indeed, it is also still not established if anti-coagulants are mandatory in these cases and the symptoms may often abate spontaneously.2 Nonetheless, physicians caring for IBD patients should be cognizant of this rare entity, either in isolation or as another possible etiology in the growing list of disorders masquerading as loss of response to anti-TNF.

Conflict of interests

Shomron Ben-Horin received consultancy fees from Abbott, Janssen, Takeda & Schering-Plough. None of the other authors has any conflicts to declare.

References


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Fig. 1 A). Superficial gentle squeezing of the abdominal wall to palpate the band-like sub-cutaneous structure. B) Gray scale ultrasound image of the left lower abdominal wall demonstrating superficial vein with thrombus, compatible with superficial vein thrombosis. C) Sonographic Doppler image of the sub-cutaneous thrombotic vein.