

# A CASE OF YERSINIA ENTEROCOLITICA DIVERTICULITIS MIMICKING INFLAMMATORY BOWEL DISEASE: THE IMPORTANCE OF DIFFERENTIAL DIAGNOSIS

ANA-MARIA BOȚIANU<sup>1</sup>, PETRE BOȚIANU<sup>2</sup>

<sup>1,2</sup>University of Medicine and Pharmacy Țirgu-Mureș

**Keywords:** *Yersinia enterocolitica, serology, culture*

**Abstract:** *We present the case of a 64-year-old male, referred to our ambulatory unit for colonoscopy. The patient complained about repeated mild rectal bleeding and abdominal pain. He was afebrile and had no weight loss. Initial laboratory showed a mildly elevated erythrocyte sedimentation rate, no anemia or leukocytosis. Repeated routine stool culture was negative. Colonoscopy revealed macroscopic changes of the mucosa suggestive of Crohn disease associated with acute diverticulitis. Histopathology showed chronic and acute inflammation, cryptitis and cryptic abscess suggestive for ulcerative colitis. Treatment was started with 5-ASA (aminosalicylate) derivatives and Rifaximin, but bleeding worsened and 5-ASA derivatives were stopped. Serology for Yersinia enterocolitica IgA and IgG was positive. Antibiotherapy with Ciprofloxacin was started, followed by rapid improvement with stool normalization. The case suggests the importance of differential diagnosis and the use of serology for excluding unusual infections, despite the presence of highly suggestive macroscopic changes for inflammatory bowel disease.*

## INTRODUCTION

Lower gastrointestinal bleeding is a common presentation in ambulatory endoscopy setting, as well as in emergency and surgical units. Cases that require hospitalization represent less than 1% of all hospital admissions in the United States. Usual causes of lower gastrointestinal bleedings include: hemorrhoids, tumours, angiodysplasia, postradiotherapy colitis, ischemic colitis, inflammatory bowel disease (usually ulcerative colitis), diverticular disease, infectious colitis. Colonoscopy is mandatory especially for the exclusion of tumoral causes and represents the initial diagnostic method of choice, especially in hemodynamically stable patients.(1)

## CASE REPORT

We present the case of a 64-year-old male, originated from an urban area, referred to our unit for lower gastrointestinal bleeding associated with mild abdominal pain. The onset of symptoms was 2 weeks before current presentation. Medical history included a cerebral meningioma, operated 7 years before current presentation, without any neurological sequelae. Travel history and exposure to raw food or non-pasteurised milk or contact with animals were also negative. Initial laboratory showed only mildly elevated erythrocyte sedimentation rate (32 mm/hour), no anemia or elevated leukocytes. Hepatic and renal profile, as well as coagulation status were within normal range. Initial usual stool culture was negative. Abdominal ultrasound was negative, showing moderate right colon aerocolia, but without enteral or colonic wall thickening, adenopathies or free abdominal fluid. Colonoscopy was performed after adequate preparation. Progression was performed up to the caecum and biopsies were taken from the terminal ileum and the colon at the level where inflammatory changes were present. Colonoscopy showed macroscopic changes highly suggestive of inflammatory bowel disease at different skipping levels: granular pattern of the

mucosa with loss of the vascular appearance, erythema and friability of the mucosa, superficial longitudinal ulcerations present in the rectum and sigmoid colon. Granular aspect, loss of vascular pattern and friability of the mucosa were also present in the ascending colon, caecum and at the level of the ileocaecal valve. A polyp was also present at the level of the ileocaecal valve, which was also biopsied. A few isolated aphthoid ulcerations were present in the transverse colon, surrounded by macroscopically normal mucosa, with preservation of the vascular pattern. A few diverticulas were present in the left colon (sigmoid) as well as the caecum. Obvious inflammatory changes, such as granular pattern of the mucosa and intense hyperemia, were present mostly at the level of caecal diverticulas. Due to the presence of diverticula near the ileocaecal valve and the high risk of perforation, endoscopy progression to the ileum was not performed but biopsies were taken carefully by passage of the biopsy-forceps. Ileal biopsies showed minimal inflammatory changes such as oedema and lympho-plasmocytic infiltrate, epithelial cryptic ectasia and cytoplasmatic mucin. Caecal and rectal biopsies showed a predominant lympho-plasmocytic infiltrate in a diffuse as well as a focal pattern, polymorphonuclear cells, oedema at the level of lamina propria, cryptitis and cryptic abscesses. The polyp was not removed due to inflammatory changes and subsequent higher risk of bleeding. Following colonoscopy, treatment was introduced having a high index of suspicion for either, Crohn disease, ulcerative colitis or indeterminate colitis as well as infectious colitis. Initial treatment consisted of 5-aminosalicylic acid (5 ASA) derivate Mesalazine 3x500 mg per oral, daily and Rifaximin 3X400mg per oral, daily. The third day following treatment introduction, the patient presented the worsening of the diarrhea and massive rectorrhagia. Mesalazine was stopped but Rifaximine was continued. The patient remained afebrile, without leukocytosis. Usual coproculture was repeated and was

<sup>1</sup>Corresponding author: Ana-Maria Boțianu, Str. Gheorghe Marinescu, Nr. 66/1, Țirgu-Mureș, România, E-mail: anamariabotianu@yahoo.com, Phone: +40741 016626.

Article received on 23.12.2014 and accepted for publication on 24.02.2015  
ACTA MEDICA TRANSILVANICA March 2015;20(1):52-54

## CLINICAL ASPECTS

negative. Serology for *Yersinia enterocolitica* was performed and showed high levels of IgA (3.2 IU/ml, negative <0.8, inconclusive: 0.8-1.1, positive > 1.1) and IgG (2.3 IU/ml) levels of antibodies. After serology suggesting *Yersinia enterocolitica* infection, antibiotherapy with Ciprofloxacin orally, 2X 400 mg per day was introduced for a course of 10 days. Clinical course improved with disappearance of diarrhea and rectal bleeding. Colonoscopy was repeated 6 months later, showing resolution of inflammatory changes. The polyp was endoscopically removed. Histopathology showed a hyperplastic polyp with no signs of dysplasia. Clinical course was favourable.

**Figure no. 1. Endoscopic appearance of the caecum: inflamed mucosa and a diverticulum, a polyp near the ileo-caecal valve**



**Figure no. 2. Inflamed mucosa of the caecum: appendix and a diverticulum**



**Figure no. 3. Isolated aphthoid ulceration within macroscopic appearing normal mucosa, with preserved vascular pattern**



**Figure no. 4. Rectal mucosa with friability, granular aspect and loss of vascular pattern**



## DISCUSSIONS

The presented case reveals the importance of differential diagnosis in any case of suspected idiopathic inflammatory disease, especially indeterminate colitis, mandatory before considering any aggressive immunosuppressive therapy.

In the presented case, colonoscopy was mandatory for excluding colonic cancer as the cause of lower gastrointestinal bleeding. The absence of fever, severe abdominal pain, diarrhea or leukocytosis was not highly suggestive for an infectious cause. Macroscopic changes at colonoscopy were initially inconclusive: rectal changes suggested ulcerative colitis but the presence of caecal and ascending colon inflammatory changes as well as isolated presence of aphthoid ulcerations also suggested the possibility of Crohn disease. After histopathology, the initial differential diagnosis was with idiopathic inflammatory bowel disease: the absence of granulomas did not support a possible diagnosis of Crohn disease. The absence of granulomas as well as the general clinical picture (normal respiratory system, lack of tuberculosis in the medical history, lack of exposure to animals or infected humans, immunocompetence) also made intestinal tuberculosis very less probable. The next step in differential diagnosis regarded other infectious causes of colitis, though the patient had no fever or leukocytosis and neutrophilia. Usual coproculture excluded etiologies such as *Shigella*, *Salmonella* and *Escherichia Coli*. Stool examination for ova and parasites was also negative. Endoscopic aspect as well as anamnesis was not supportive for *Clostridium difficile* colitis (lack of antibiotherapy).

Medical history was not supportive for ischemic colitis: the patient had no significant cardiovascular disease risk factors, except age, and presented only mild intermittent abdominal pain. IgG and IgA positive serology for *Yersinia enterocolitica* suggested infectious colitis associated with diverticulitis. Though false positive results are also possible, diagnosis was supported by the rapid resolution of symptoms and a good clinical outcome following antibiotherapy with Ciprofloxacin.

According to the literature (2), *Yersinia enterocolitica* is not sensible to Rifaximin, previously used in the treatment. Besides fluoroquinolones, other sensible antibiotics for *Y. enterocolitica* include: aminoglycosides, doxycycline or trimethoprim-sulfamethoxazole.(2)

Laboratory diagnosis of *Yersinia enterocolitica* infection is not routinely performed by most microbiology laboratories.(3) Routine coproculture does not include testing for *Yersinia enterocolitica*, as well as other bacteria such as *Campylobacter jejuni* or *Clostridium difficile*. Viral etiologies (such as Norovirus, Rotavirus, Adenovirus, Norwalk-like virus, Calicivirus, Enteric adenovirus) are also not routinely tested, but are more frequent in children.(4)

*Yersinia enterocolitica* is a Gram-negative motile aerobic bacterium which belongs to the Enterobacteriaceae family.(3) It was first identified as a distinct organism isolated from the stool of human cases of diarrhea by Coleman and Schleifstein in 1939.(5) Only 3 of eleven species of the genus *Yersinia* are associated with the disease: *Yersinia pestis* is the causative agent of plague, while *Yersinia pseudotuberculosis* and several pathogenic bio-serovars of the species *Yersinia enterocolitica* cause yersiniosis.(6)

The bacteria can be detected via several methods, though none performed routinely.(3) Diagnosis of *Yersinia* infection is difficult without specific culture and/or serology. *Yersinia* is not routinely tested in the United States. Isolation of the organism by culture may be difficult with standard

## CLINICAL ASPECTS

media.(3,4) According to the literature, culture diagnosis with cold enrichment may be improved reaching a 56% sensitivity, with selective media such as CIN (Cefsulodin-Irgasan-Novobiocin) agar being more sensitive. Serology reaches a sensitivity of 84% after 1 week of symptoms. Combined culture and serology is 88% sensitive for serology.(3,4,7,8)

Clinical picture varies to more severe forms, which could include elements such as sepsis, vomiting, diarrhea, abdominal pain, pseudo-appendicitis like picture, arthritis, extraintestinal features such as cellulitis, pyomyositis, osteomyelitis, pneumonia, lung abscess, meningitis or glomerulonephritis.(3) Serious and unusual complications like endocarditis, (9) necrotizing pneumonia (10) or sepsis (11) are also possible, but mostly described in immunosuppressed patients. Postinfectious complications include reactive arthropathy and erythema nodosum.(12)

Symptomatic *Y. enterocolitica* infection is more common in children than adults.(13) This could explain the lack of several elements, especially fever or leukocytosis in our adult patient. Spontaneous resolution of the symptoms, without any antibiotic treatment, is also possible in uncomplicated forms.(3,12)

### CONCLUSIONS

Our case suggests the importance of *Yersinia enterocolitica* testing, either through culture but mostly through serology titers when evaluating patients suspected from inflammatory bowel disease. This should be mandatory especially before considering corticotherapy or even more aggressive immunosuppressive therapy (Azathioprine or biologic therapy) in patients suspected of inflammatory bowel disease.

The case underlines the importance of laboratory testing, especially of serology titers, in successfully completing endoscopic and histopathology data. Though important, endoscopy and histopathology may not be enough for establishing a correct diagnosis and thus, a correct treatment.

### REFERENCES

1. The American Society for Gastrointestinal Endoscopy. Guideline-The role of endoscopy in the patient with lower gastrointestinal bleeding. *Gastrointestinal Endoscopy*. 2014;79(6):875-885.
2. Centers for Disease Control and Prevention. Diagnosis and management of foodborne illnesses: a primer for physicians and other health care professionals. *MMWR Recomm Rep* 2004;53:1-33.
3. Centers for Disease Control and Prevention. *Yersinia enterocolitica*. CDC. Available at [http://www.cdc.gov/ncidod/dbmd/diseaseinfo/yersinia\\_g.htm](http://www.cdc.gov/ncidod/dbmd/diseaseinfo/yersinia_g.htm). Accessed at 23<sup>rd</sup> december 2014.
4. Zheng H, Sun Y, Lin S, Mao Z, Jiang B. *Yersinia enterocolitica* infection in diarrheal patients. *Eur J Clin Microbiol Infect Dis*. Aug 2008;27(8):741-52.
5. Schleifstein J, Coleman, MB. An unidentified microorganism resembling *B. lignieresii* and *Pasteurella pseudotuberculosis*, pathogenic for man. *New York State Journal of Medicine*. 1939;39:1749-53.
6. Bottone EJ. *Yersinia enterocolitica*: the charisma continues. *Clin Microbiol Rev*. 1997;10(2):257-76.
7. Zheng H, Wang J, Sun Y, Jiang B. Clinical isolation and characterization of *Yersinia enterocolitica* in China using real-time PCR and culture method. *Digestion*. 2007;75(4):199-204.
8. Zheng HX, Sun Y, Jiang B. Evaluation of 4 culture

methods of *Yersinia enterocolitica*. *Nan Fang Yi Ke Da Xue Xue Bao*. 2007;27(9):1438-40.

9. Krajinovic V, Tambic Andrasevic A, Barsic B. Tricuspidal valve endocarditis due to *Yersinia enterocolitica*. *Infection*. 2007;35(3):203-5.
10. Girszyn N, Kerleau JM, Robaday S, Lefebvre S, Marie I, Levesque H. Pneumonia with bacteremia due to *Yersinia enterocolitica* in a diabetic patient carrying HLA-B27. *Rev Med Interne*. 2007;28(12):882-4.
11. Lenz T, Schulte KL, Meyer-Sabellek W. *Yersinia enterocolitica* septicemia during long-term immunosuppressive treatment. *J Infect Dis*. 1984;150(6):963.
12. Rosner BM, Werber D, Höhle M, Stark K. Clinical aspects and self-reported symptoms of sequelae of *Yersinia enterocolitica* infections in a population-based study, Germany 2009-2010. *BMC Infect Dis*. 2013;13:236. doi: 10.1186/1471-2334-13-236.