INFLAMMATORY BOWEL DISEASES: A HIDDEN COMORBIDITY IN PEOPLE LIVING WITH HIV?

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BACKGROUND:

The interplay between HIV infection and Inflammatory Bowel Disease (IBDs) is not clear, although events like cell-mediated immune responses and microbial translocation occur in both conditions. An estimated 2.5–3 million people in Europe are affected by IBD. Prevalence of IBDs in HIV population is currently unknown.
It has been suggested that HIV-related immunosuppression may reduce IBDs activity, but data on HIV patients who have been diagnosed with IBD are still lacking.
The aim of this study was to determine the prevalence of IBDs in our HIV population and describe the characteristics of HIV-positive patients with IBD.

MATERIALS AND METHODS:

We conducted a retrospective study, including all the HIV-positive patients on antiretroviral therapy (ART) with a definitive diagnosis (histologically confirmed) of Ulcerative Colitis (UC) or Crohn's Disease (CD), at our HIV clinic, in Bologna.
Pharmacy records for IBDs drugs were examined for all men and women with HIV infection in the last 10 years to individuate eligible individuals.

Table 1. Characteristics of HIV population with IBD diagnosis.

CHARACTERISTIC OF POPULATION (n 17)	
MALE SEX (n,%)	17 (100%)
AGE (years, median IQR)	44 (37-49.5)
MSM (n,%)	13 (77%)
CDC CLASS C (%)	4 (24%)
CD4 T-CELLS NADIR (/mmc)	396 (209-586)
YEARS SINCE HIV DIAGNOSIS (median IQR)	8 (5.5-18)
CD4 T-CELLS (/mmc)	826 (589-973)
HIV <50 CP/ML (n, %)	17 (100%)
HCV Ab (n, %)	8 (47%)
INI-CONTAINING ART REGIMEN (n, %)	6 (35.3%)
PI-CONTAINING ART REGIMEN (n, %)	7 (41.2%)
YEARS SINCE IBD DIAGNOSIS (median IQR)	7.5 (2.25-10.5)
ULCERATIVE COLITIS (n,%)	17 (100%)
UC EXTENT	
PROCTITIS (n, %)	7 (41%)
DISTAL COLITIS (n, %)	1 (6%)
PANCOLITIS (n, %)	7 (41%)
NA (n, %)	2 (12%)
PHARMACOLOGICAL TREATMENT FOR IBD	
NO TREATMENT (n, %)	6 (35%)
STEROIDS(n, %)	1 (6%)
MESALAZINE (n, %)	8 (47%)
MESALAZINE PLUS STEROIDS (n, %)	2 (12%)
IMMUNOSOPPRESSIVE DRUGS (N, %)	0

RESULTS:

Table 1 shows the characteristics of our study population. We identified 17 HIV-positive men with IBD (0.72 % of the overall HIV population on ART at our clinic), aged 44 years (IQR 44-49.5), with optimal immunological and virological status (undetectable HIV-RNA and good immune response, with median CD4+T cells 826/mmc). 77% were men who have sex with men (MSM). Median CD4+T cells nadir was 369/mmc, and only 4 individuals had CDC class C disease.
In most cases (82%), IBD identification followed (for years) HIV diagnosis. All the included patients had UC (47% proctitis), no CD was observed. Median age at RCU diagnosis was 37 years. Four patients had a mild active disease (partial Mayo score 2) and 6 were not taking drugs for UC, at the time of this study. Three patients had undergone surgery for UC. Median duration of follow-up since IBD diagnosis was 7.5 years. 41% had had at least a UC relapse over time. Median relapse rate was 0.041/year of follow-up. No HIV or ART-related factor was associated to UC extent or relapse rate.

CONCLUSIONS:

Prevalence of IBDs in our HIV population was low. Males and MSM might be at higher risk for UC. Diagnosis of IBD might be often missed or delayed in people living with HIV (PLWH) since intestinal symptoms are usually attributed to HIV itself, ART side effects or opportunisms.
Since IBDs misdiagnosis may represent a risk, new diagnostic tools and algorithms as well as deeper insight in therapeutic options for IBD are needed in PLWH. Main limitation of this study is its retrospective nature.

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