# SESSION 5
# INFLAMMATORY BOWEL DISEASE MIMICKERS

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### Learning Objectives

- Review checkpoint inhibitor colitis, NSAID colopathy and infectious colidites;
- Differentiate IBD mimickers from real IBD; and
- Discuss treatment of checkpoint-inhibitor colopathy

IBD diagnosis is not always straightforward. Five years after the diagnosis of IBD has been made, it is refuted in approximately 7% of patients.[1] This proportion is as high as 50% in patients in whom the diagnosis of *possible* IBD had been made.[1]

Main mimickers of IBD are infections (bacterial [including tuberculosis[2]], viral, fungal, parasitic), drug-induced enterocolitis (non-steroidal anti-inflammatory drugs [NSAIDs], gold compounds, angiotensin-converting-enzyme inhibitors), ischemia, chronic diverticular disease, immune deficiencies, intestinal neoplasia, endometriosis, systemic diseases (lupus, microscopic polyangiitis, Henoch-Schönlein purpura, Wegener disease, Behcet's disease) and others.

Proctitis should be differentiated from rectal solitary ulcer syndrome, endometriosis and sexually-transmitted infection. In patients with a confirmed diagnosis of IBD, diarrhea and abdominal pain can be due to IBD activity, but may also be related to infection (*Clostridioides difficile*, cytomegalovirus, other bacterial, viral, parasitic enteropathogens), drug toxicity and/or cancer.

New drugs such as immune checkpoint inhibitors (ICIs, e.g., anti CTLA-4 and anti PD-1) and phosphatidylinositol-3-kinase inhibitors (e.g., idelalisib) can also lead to presentations mimicking IBD. ICIs have improved the treatment of several cancers. These drugs enhance T-cell activity, and therefore stimulate anti-cancer immunity, but also cause immune-related adverse effects (irAE), including those affecting the gastrointestinal (GI) tract.[3] GI-irAE are observed in 7 to 30% of patients treated with ICI therapy. Enterocolitis due to anti-CTLA-4 therapy is frequent and often severe. GI irAE associated with PD-1 blockade is less frequent and clinically more diverse. Current management of patients with GI IrAE should include refutation of differential diagnoses, assessment of severity, use of corticosteroids and rapid introduction of infliximab in non-responders.

### Key References

1. Henriksen M, Jahnsen J, Lygren I, et al. Change of diagnosis during the first five years after onset of inflammatory bowel disease: results of a prospective follow-up study (the IBSEN study). *Scand J Gastroenterol*. 2006;41:1037–43.
2. Limsrivilai J, Shreiner AB, Pongpaibul A, et al. Meta-Analytic Bayesian Model For Differentiating Intestinal Tuberculosis from Crohn's Disease. *Am J Gastroenterol*. 2017;112:415–27.
3. Soularue E, Lepage P, Colombel JF, et al. Enterocolitis due to immune checkpoint inhibitors: a systematic review. *Gut*. 2018;67:2056–206.