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Editorial

Checkpoint Inhibitor Induced Colitis

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EDITORIAL NOTE

Cancer immunotherapy causes checkpoint inhibitor mediated colitis, which is an inflammatory disease affecting the colon. Diarrhea, stomach pain, and rectal bleeding are common symptoms. Nausea and vomiting are less common, but they can indicate the presence of gastroenteritis. The frequency of bowel movements and the symptoms of colitis are used to grade the severity of diarrhoea and colitis, respectively.

Colonoscopy with examination of the terminal ileum is the gold standard for diagnosing checkpoint inhibitor mediated colitis. In certain cases, however, a flexible sigmoidoscopy would suffice. Stool tests, including Clostridioides difficile, bacterial culture, ova, and parasites, can be used to rule out infection

The severity of immune checkpoint inhibitor colitis is determined by the severity of the diarrhoea and colitis. Mild cases may be treated with loperamide, dietary alteration and or a brief cessation of immune checkpoint inhibitor therapy. Immune suppression with corticosteroid therapy is needed in more serious cases. Infliximab can be considered if steroids are ineffective. If infliximab fails to improve colitis, vedolizumab may be an option.

T regulatory cells in the intestine require immune checkpoints to grow normally. Mice lacking the CTLA-4 gene (e.g CTLA-4 knockout mice) develop a serious autoimmune disease characterised by diffuse T cell invasion in multiple organs and fatal enterocolitis.

Diffuse mucosal inflammation or focal active colitis with patchy crypt abscesses are common symptoms of immune checkpoint inhibitor colitis. Intraepithelial neutrophilic infiltrates, crypt abscesses, and increased apoptotic cells within crypts are all common symptoms of acute colitis. However, the histologic presentation differs, and signs of persistent inflammation, such as intraepithelial lymphocytes or basal lymphocytes, as well as crypt architecture distortion, can be seen in certain instances. Histologic inflammation may appear as soon as 1-2 weeks after starting immune checkpoint inhibitor therapy, well before symptoms appear.

Those with Faecali bacterium genus and other Firmicutes present in the colonic flora have longer progression-free survival and overall survival when taking immune checkpoint inhibitors. Furthermore, the involvement of Faecali bacterium in the faecal microbiota is linked to a higher risk of checkpoint inhibitor-induced colitis.

The most frequent symptom is diarrhoea which occurs in 92 % of cases, followed by abdominal pain which occurs in 82% of cases and rectal bleeding which occurs in 82 % of cases. Fever is present in 46% of cases, and nausea and vomiting are present in 36%. Nausea and vomiting are less common side effects. It has been confirmed that you have lost weight. After beginning immune checkpoint inhibitor therapy, diarrhoea typically appears 6–7 weeks later.

The gold standard for diagnosing checkpoint inhibitor-induced colitis is colonoscopy with examination of the terminal ileum. In most cases, however, a minimal distal colon examination with versatile sigmoidoscopy is necessary. Loss of vascular pattern, erythema, erosions, ulcers, exudates, granularity, and bleeding are all possible endoscopic findings. There are no tests for checkpoint inhibitor-induced colitis in the stool or blood. Diagnosing diarrhoea and colitis should, however, include ruling out infectious causes. Clostridioides difficile toxin, bacterial culture, ova, and parasites can all be tested in the stool. CMV infection testing should be considered.

Fecal calprotectin can be beneficial, and it is highly responsive and specific for intestine inflammation. Increases in Faecal calprotectin are linked to the severity of intestinal inflammation. Colitis can be detected using computed tomography (CT) imaging, but the sensitivity is limited to 50%. Bowel perforation is indicated by the presence of free air in the peritoneum.

Depending on the seriousness of the condition, different treatments are used. Supportive treatment, such as loperamide and a low residue or bland diet, can be appropriate for mild disease. The immune checkpoint inhibitor should be stopped if the disease is serious. Corticosteroid therapy, at a dosage of about 1–2 mg per kg of body weight per day, is used to reduce inflammation.

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