balloon size of 12 mm every 2 weeks (Fig. 1b). Oral proton pump inhibitors and sucralfate were continued for 2 months. He required a total of three dilatations and the last dilatation was performed with an 18 mm size balloon. After three dilatations, his pyloric channel opened well (Fig. 1c). After the third dilatation, the patient became symptom free. On follow-up after 3 months of the last dilatation, the patient was asymptomatic and upper gastrointestinal endoscopy showed completely opened pylorus without any ulcerations or scarring. This is the first case of GOO caused by common salt binge, which was managed successfully with endotherapy. The mechanism of GOO because of common salt is not known. The possible hypothesis is that excessive salt load, when delivered to the stomach near the antropyloric region, without an adequate amount of water, may have resulted in an osmotic shift. This osmotic shift from gastric cells might have been further precipitated by recurrent vomiting and fluid loss, leading to cell shrinkage, dysfunction, and death. This might have activated the inflammatory cascade, resulting in mucosal inflammation and edema, leading to mechanical GOO. Earlier, surgery was considered the conventional treatment modality for benign GOO, but it was associated with morbidity and long-term complications such as anemia and malabsorption [4,5]. Currently, endotherapy in the form of endoscopic balloon dilatation of the obstructed gastric outflow tract is the well-accepted nonsurgical management of benign GOO, although the numbers of dilatations required for corrosive are more compared with other etiology [3]. In conclusion, common salt binge can lead to benign GOO, which can be managed successfully with endoscopic balloon dilatation and supportive treatment.

## Informed consent

This case report study has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its subsequent amendments.

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### *Conflicts of interest*

There are no conflicts of interest.

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OPEN

# Nodular regenerative hyperplasia in inflammatory bowel disease patients with allopurinol–thiopurine cotherapy

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Nodular regenerative hyperplasia (NRH) of the liver is an important cause of noncirrhotic portal hypertension (NCPH) [1]. The onset of NRH has been associated with various disorders and drugs [2]. Especially azathioprine and thioguanine have been associated with NRH, presumably related to the high levels of generated end-metabolites, 6-thioguanine nucleotides (6-TGNs) [3]. The occurrence of complicated NRH in patients with allopurinol and thiopurine cotherapy for inflammatory bowel disease (IBD), a strategy to optimize therapeutic 6-TGN levels, has not been reported before. Here, we report on two NRH patients with NCPH and allopurinol–mercaptopurine combination therapy for IBD.

A 37-year-old male patient with stricturing Crohn's disease (CD) for 7 years presented with persisting fatigue and thrombocytopenia. He was treated with allopurinol and mercaptopurine (50 mg/day) cotherapy for over 5 years and his CD remained well controlled during this period. At the time of presentation, physical examination and laboratory tests showed no abnormalities apart from the thrombocytopenia. An abdominal ultrasound showed splenomegaly and heterogeneous liver parenchyma resembling liver cirrhosis. In a liver biopsy, histopathological characteristics of NRH and mild portal sclerosis were observed. There were no varices or other abnormalities during esophagogastroduodenoscopy; therefore, prophylaxis with β blockers was not initiated. Cotherapy with allopurinol and mercaptopurine was discontinued and both the CD and manifestations of NCPH have remained stable during follow-up of 1 year.

A 33-year-old male patient with fistulizing CD for 12 years presented with thrombocytopenia and elevated liver tests. He was treated successfully with allopurinol and mercaptopurine (25 mg/day) cotherapy for almost 4 years until his presentation with biochemical abnormalities. Cotherapy was discontinued, but hepatotoxicity (grade 1) persisted. Abdominal imaging showed a heterogeneous liver parenchyma, but no other abnormalities suggestive of portal hypertension. In a liver biopsy, NRH and mild phlebosclerosis, and during an esophagogastroduodenoscopy, large esophageal varices were

observed. Prophylaxis with β blocker therapy was initiated. Within 3 years, the patient showed a splenomegaly and variceal bleeding, managed with endoscopic variceal ligation. Variceal bleeding recurred once during 5 years of follow-up.

NRH is a rare liver condition with an uncertain etiology and prognosis. NRH has been particularly associated with thioguanine exposure [4]. The pharmacokinetic conversion of thioguanine leads to relatively high 6-TGN concentrations, presumably attributed to NRH development. The addition of allopurinol to thiopurine therapy favorably alters thiopurine metabolism, leading to increased 6-TGN levels as well. In such a manner, NRH-like abnormalities may also occur in IBD patients with allopurinol–thiopurine cotherapy, as reported in our two cases. Interestingly, both cases were young men with complicated CD, which are determinants that have been considered as risk factors [5]. Complications of NCPH remained stable in one and progressed further in the other patient. Our reports illustrate the importance of suspecting NRH in IBD patients with allopurinol–thiopurine cotherapy and its uncertain prognosis.

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Melek Simsek and Margien L. Seinen drafted the manuscript. Nanne K.H. de Boer revised the manuscript critically. All authors commented on drafts of the paper. All authors have approved the final draft of the article.

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There are no conflicts of interest.

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