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Case report: Colon ischemia and perforation as a result of Norovirus infection

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ABSTRACT

INTRODUCTION: Norovirus (NoV) gastroenteritis has been documented as the worldwide leading cause of the majority of acute cases of viral gastroenteritis. Here, we present a Case of NoV that progressed into colon perforation.
PRESENTATION OF CASE: A 47-year-old woman was admitted via the emergency unit with diarrhoea, lower abdominal pain, vomiting and fever. The virological testing of her stool revealed a NoV infection. The abdominal CT scan showed massive pneumatosis intestinalis. Following the scan findings, the patient was admitted for a diagnostic laparotomy the same day. A side-to-side ileosigmoidostomy was performed. We performed two clinical re-evaluations of the patient, the first one took place 2 weeks after we discharged the patient and another one-year later. The patient is in perfect health.
DISCUSSION: To the best of our knowledge and following a thorough bibliographical search, this is the first case report in Germany and the first case report of colon perforation due to NoV infection in adults in the European Union.
CONCLUSION: A NoV infection could, along with the typical symptoms, indicate a life-threatening bowel ischemia and/or necrosis.

1. Introduction

Norovirus (NoV) gastroenteritis has been documented as the worldwide leading cause of the majority of acute cases of viral gastroenteritis [1,2]. Originally named Norwalk virus after its identification in the town of Norwalk in Ohio, USA, [1,2] NoV belongs to the Caliciviridae family of viruses. Although it does not show the characteristic Caliciviridae morphology, it is assigned to this family due to its genomic structure [1]. There are 5 genogroups (GI–V) and multiple genotypes, of which only genogroups I, II, and IV are relevant for humans [1,3,4]. According to the Robert Koch Institute in Germany, in 2017 the genotype GII.4 was responsible for 70%–80% of acute cases of gastroenteritis worldwide [4]. The majority of the cases occur in the winter period, usually in outbreaks [3,4].

The spread of the virus can be via contaminated food and water, fecal-oral, or airborne via contaminated aerosols. It is usually mild and self-limiting [1–4]. It has a short incubation period (24–60 hrs.) and an infection duration of about 12–60 hrs. [1,2]. It usually affects small children and the elderly with acute onset of frequent vomiting and watery diarrhoea, although bloody diarrhoea has also been reported [1,2,6]. It may also present with muscle aches, abdominal cramps and fever [1].

2. Case report

A 47-year-old woman was admitted via the emergency unit with reduced general condition, diarrhoea, lower abdominal pain, vomiting and fever. She had no significant past medical history of note as well as family genetic disorders. The Patient only took birth control pills. Patient is married and has 2 sons. She was a non-smoker and only drank alcohol sporadically. The physical examination was typical for gastroenteritis with mild lower abdominal tenderness, lively bowel sounds and no abdominal guarding whatsoever. The virological testing of the stool revealed a NoV infection. The inpatient's admission was carried out by the internal medicine unit.

The following day, the patient showed no improvement in spite of conservative treatment. She complained of increasing abdominal pain. Clinically the patient had developed an extraordinary painful bloated abdomen with diminished bowel sounds. The laboratory results did not show any unusual results.

It was decided to perform a computer tomography for further clarification. The abdominal CT scan showed massive pneumatosis intestinalis in the distal ascending colon and caecum with air in the intrahepatic portal vein and suspicion of colonic ischemia (Figs. 1 and 2). Following the scan findings, the patient was admitted for a diagnostic laparotomy the same day.

Intraoperatively a segmental colon ischemia was found, specifically in the ascending colon and colon transversum. The small intestine was not affected. The liver and gallbladder were unre-

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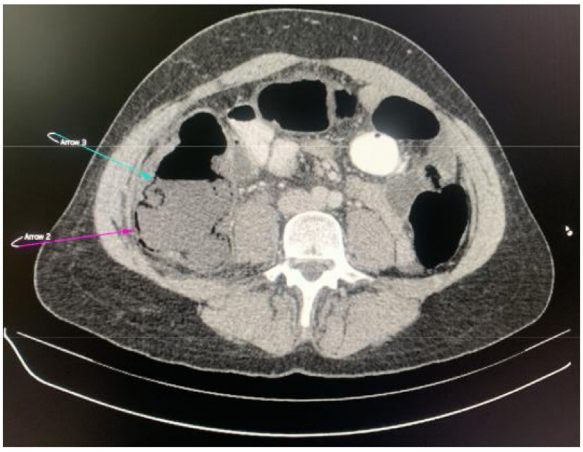

Fig. 1. Pneumoatosis intestinalis.

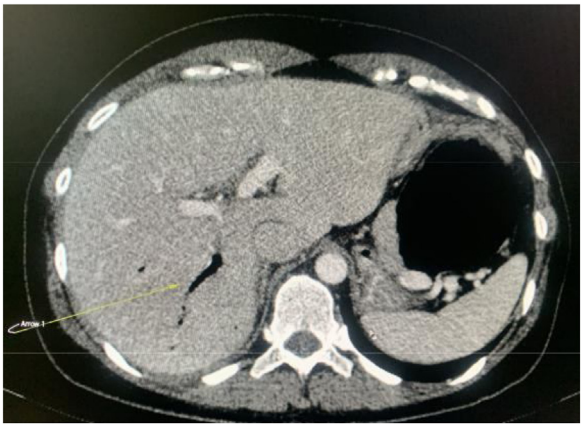

Fig. 2. Air in the intrahepatic portal vein.

markable. Fibrinous purulent peritonitis was described. There was no clinical evidence of embolism. A resection from the caecum, colon ascendens and transversum with left colonic flexure was performed. A side-to-side ileosigmoidostomy was performed. The procedure was performed by the chief of the department. Antibiotic therapy with Piperacillin and Tazobactam 3 g. i.v was administered postoperatively. The patient spent one night in the intensive care unit for post-op monitoring with no further complications.

The following day the laboratory results were alarming, with a CRP mg/l value of 201 g/l and procalcitonin of 13.25; the leukocytes were not elevated. The patient was clinically diagnosed with a paralytic ileus. The next day the lab results showed an improvement of the CRP (93.6 mg/l) and procalcitonin (4.48 g/l). The leukocytes were still stable. Unfortunately, in spite of the measures taken to try to resolve the paralytic ileus, the patient's condition continued to worsen and it was decided to perform a second-look relaparotomy on the third day post-op.

Intraoperatively, signs of a small and large intestine paralytic ileus became apparent. Around 100 mL of murky secretion were also observed. The anastomosis was almost completely healed, with no signs of insufficiency. A size 10 Easy Flow Drain was placed. With no other apparent pathological problems, the operation was terminated.

The pathology report showed ischemic colitis with extensive mucosal necrosis, partly with transmural inflammatory diffusion, accompanied by purulent peritonitis.

As there was no apparent reason for the colonic ischemia, we performed more tests to try to identify possible embolic sources such as a transoesophageal echocardiography and long-term ECG monitoring without any striking results. Blood tests showed no relevant changes.

Since we could not explain the colonic ischemia and our other attempts at trying to find a cause of the disease pattern brought us no results, we requested a pathological reevaluation. This time we contacted the pathologists and conveyed that we had a strong suspicion that norovirus was the culprit. The re-evaluation showed big subserosal as well as submucosal thrombosed blood vessels. Although these thrombosed vessels were found in extensively altered inflamed tissue, a thromboembolic source could not be diagnosed, because the origin of the thromboembolic events could also be a secondary thromboembolic event due to the ischemic mucosal necrosis.

6 days after the second operation, under close medical supervision, we were able to discharge the patient. At the time of the dismissal, the patient was in perfect medical condition without any infection or anastomosis insufficiency signs.

We performed two clinical re-evaluations of the patient, the first one took place 2 weeks after we discharged the patient and another one year later. The first re-evaluation showed that the patient was in perfect health; bowel movements and stool consistency were satisfactory. The healing process was unremarkable. We ordered coagulation testing as a last attempt to try to explain the embolic source. A coagulopathy was ruled out, reinforcing our assumption that the clinical picture was due to norovirus infection.

The second re-evaluation showed that the patient was completely healthy, able to work without complaints and leading a normal life.

3. Discussion

To the best of our knowledge and after a thorough bibliographical search, this is the first case report in Germany and the first case report of colon perforation due to NoV infection in adults in the European Union. A Cochrane search for colon perforation in adults one year later yielded no results. Isolated reports of associated perforation/ischemia and NoV in England, Italy and Japan were found [2,6,7].

To date there is no way to predict whether a NoV infection will develop to such a life-threatening point as in our case. It is also difficult to say whether external factors, like gene-specific alterations or a especially aggressive NoV strain, play a role.

It is known that, following a NoV infection, disruption of the gut microbiota develops [8], but if such alterations can lead to the development of pneumoatosis intestinalis [5] and in turn to ischemia is yet to be proved.

It was also unclear why the colon was mainly affected, leaving the small intestine intact, especially because it is thought that NoV mainly affects the villous enterocytes of the duodenum and jejunum [6], which result in the typical clinical manifestations mentioned above.

If a patient with NoV infection admitted to hospital shows no signs of improvement, we strongly recommend imaging diagnostics to rule out ischemia and/or signs of perforation.

4. Conclusion

NoV gastroenteritis could, along with the typical symptoms, elicit life-threatening bowel ischemia and/or perforation. This should particularly be taken into account in the case of abnormally intense abdominal pain. It was demonstrated that the early

computer tomography imaging diagnostics clearly showed pathological changes, even before altered laboratory parameters became apparent. In individual cases, this can lead to quicker surgical intervention.

Declaration of Competing Interest

There is no conflict of interests.

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Ethical approval

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Consent

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Author contribution

Carlos M. Beredjiklian: Writing original draft, review & editing, investigation, data curation
Marc Olf: Review & editing
Ahmat Abusaman: Review & editing
Richard Becker: Review & editing
Jens O. Jonescheit: Review & editing

Registration of research studies

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