Viral Diarrhea Induces Ischemic Colitis

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ABSTRACT

Ischemic colitis is a popular diagnosis suggesting sudden hypo-perfusion of the colon. Pathophysiology of this illness has not been resolved clearly. Most of ischemic colitis is a one time disease and do not need specific treatment and recover self-limitedly under only bowel rest. Ischemic colitis sometimes exist with viral infection, this therefore necessitated the need to analyze the relationship between ischemic colitis and viral infection in this study. A consecutive two hundred and fifty-six (256) acute gastroenteritis cases were recruited and pathogen examined for both the virus and bacteria. All of these cases were sporadic. Viral check was carried out by the commercialized kit and PCR. Colonoscopy was performed as soon as possible to get information about changes occurring in the large intestine. Among the 256 cases, 84 cases had viral infection (Norovirus, Adenovirus, Rotavirus, and Astrovirus) and 26 cases bacterial infection. Bloody diarrhea occurred in 89 and ischemic colitis was diagnosed in 42 cases. Pathogen examination revealed significant increase of viral infection (66.7%) among ischemic colitis, comparing with non-infection (26.8%) and bacterial infection (31.3%). According to our result, not only Norovirus but other viral infection possibly induced ischemic colitis. When ischemic colitis is diagnosed together with some viral infection, we can observe patients without excessive examination. This study suggests enteric viral infection could be related as triggers of ischemic colitis. This thus led us to consider an alternative route to quietly unknown ischemic colitis. Word count of enteric virus is 247 and as such it is important to check enteric virus in the so-called ischemic colitis.

Key words: Ischemic colitis, viral diarrhea, norovirus, rotavirus, bloody diarrhea, acute diarrhea, gastroenteritis.

Abbreviations: IsC: Ischemic colitis; IHB: Index of hemoglobin.

INTRODUCTION

Pathogenesis of ischemic colitis has not been fully resolved. We sometimes experience ischemic colitis in young age without any background, or most of ischemic colitis does not relapse again. There may be many cases of a one time occurrence of ischemic colitis. We analyzed cumulated cases against conventional ischemic colitis theory under other hypothesis.

MATERIALS AND METHODS

Acute gastroenteritis patients were recruited for this study. Among 256 cases, we investigated, furthermore pathogens and colonoscopy or abdominal CT was performed as soon as possible. Virus check was by PCR to Astrovirus, Adenovirus, Norovirus, and ELISA to Rotavirus.
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Figure 1. Infection state in acute gastroenteritis. Viral infection was 84 cases (32.8%), bacterial infection 26 cases (10.2%), and co-infection 10 cases (3.9%). Detected virus cases are Rotavirus 30.9%, Astrovirus 19.6%, Adenovirus 14.0%, and Norovirus 34.6% respectively. Total number of detected viruses was up to 113 including duplicated infection.

Stool culture was carried out for enteric pathogenic bacteria. We had a chance to get supplemental stool sample throughout colonoscopy. A series of colonoscopic procedure and its diagnosis was made by two or more skilled endoscopists. The diagnosis was made comprehensively by clinical course, past medical history, colonoscopy, abdominal CT and laboratory data. Pathogenic bacteria cultures in the feces stool culture for pathogenic bacteria started on the first visit day, including Salmonella, Shigella, Vibrios, Campylobacter, Clostridium difficile, Yersinia, and pathogenic Escherichia coli. Intraluminal pressure and blood flow of the colon mucosa endoscopically by already established method was examined. We aim to obtain the possibility of disturbed blood flow by the increase of pressure. This study is approved by the ethical comity of Tokyo Metropolitan Komagome General Hospital.

RESULTS

Infection was confirmed in 120 out of 256 cases; 84 virus infections, 26 bacterial infections, and 10 co-infections. Bacteria were detected in 36 cases and no pathogens in 136 (51.0%). Enteric viruses were detected in 94 cases out of 256 patients (co-infected viruses included) (Figure 1). Detected viruses are Rotavirus (30.9%),Astrovirus (19.6%), Adenovirus (14.0%), and Norovirus (34.6%). Ischemic colitis (IsC) was observed in 42 cases and 66.7% was viral infection and on the other hand, 25.0% of non-infectious cases (p<0.001) (Figure 2). Among the 22 virus associated with IsC, 9 cases had some predisposing vascular factors, such as vascular disease 45% and abdominal surgery 23%, but there was 32% without any predisposing factor. Figure 3 shows infection state in IsC.

DISCUSSION

Most of enteric viruses which cause viral acute gastroenteritis are thought to infect the villous enterocytes of the duodenum and oral side of the jejunum through the carbohydrate ligands expressed, and especially as to Norovirus which also plays a role as histo-blood group antigens (Ming and Jiang, 2005). Because of the replication of viruses in the enterocytes, a large number of infected enterocytes die and fall into the enteric lumen, thereby resulting in diarrhea and malabsorption. Although, these viruses do not affect the large intestine from their essential activities, we sometimes experienced simultaneous onset of viral infection and IsC. As for the pathogenesis of the
Figure 2. Colonoscopic diagnosis is summarized regarding 108 acute gastroenteritis cases. Ischemic colitis was diagnosed in 42 cases, ischemic colitis occupied 66.7% of virus positive colitis and ischemic colitis in not-infective colitis was 25.0% (chi-sq. test; P<0.001). (No Infect: no infection, Virus Infect: viral infection, Non spe: non-specific inflammation) Ischemic: ischemic colitis, Ulcerative: ulcerative colitis, Dive Bleed: diverticular bleeding).

Figure 3. Infection state in ischemic colitis cases. Forty two cases of ischemic colitis were pointed out among 84 bloody diarrhea cases. This shows about 50% of bloody diarrhea is possible ischemic colitis. Virus positive cases are 52.3% (22/42) of total ischemic colitis.

Colonic lesion, direct injury of the colonic mucosa by enteric viruses seems unlikely considering the cell affinity of viruses and a report by Salim et al. (1990) revealed that no significant pathological alterations were found in the stomach and colon in patients with viral gastroenteritis (Levy et al. 1976).

Previous concept of IsC has excluded cases where pathogens were detected, but, virus infection has not been discussed and ignored from IsC argument. IsC has been reported to occur in broad spectrum (Cheng, 2012; Zenda et al. 2010; Gandhi et al., 1996; Amott et al. 1999). It is supposed to occur fundamentally related to vascular
changes which can decrease intestinal blood flow. The theory was received specious or excessively subtle as a royal road of pathogenesis of IsC.

According to this theory, all patients must have been in debt of blood flow due to vascular illness or faced with some predisposing factors. However, many of the patients could not necessarily be related to any predisposing medical histories (Holthouse, 2007). If there are no predisposing factors, the illness would be induced by constipation or due to idiopathic reason. We are also of the opinion that the illness could be caused by various conditions. We recognized that for IsC to occur conditions such as vascular changes accompany arteriosclerosis, diabetes mellitus, and vasculitis, but we sometimes experience that young healthy adults are diagnosed of IsC. Previous reports pointed out IsC in young adult are not rare, and more than 50% of its causes and pathogenesis were not clear (Nigel and Grundman, 1990). As they have no past histories, constipation has been supposed to be responsible for the ischemic colitis. Causes of IsC in young adults who have no predisposing conditions have been linked to episodes of constipation and / or unknown (Preventza et al, 2001). We think IsC occurring together with viral infection could occur more frequently than expected. Referring to pathogen detection, viral check at IsC has been unusual except for a specific case (Lin et al. 2004) so we encourage not only stool culture for bacteria, but also viral check to be ordered in cases of bloody diarrhea.

Increase of intraluminal gut pressure causes decrease of blood flow. We speculate severe diarrhea can induce transient ischemia leading to IsC. Thus, this type of IsC seldom leads to gangrenous form or stricturing form (Marston, 1996).

It is more probable that enhanced intestinal peristalsis increased intraluminal gut pressure due to excessive watery stool and gas output, and hypovolemia or hem concentration after diarrhea and vomiting impairs microcirculation at the colonic mucosa, thereby, resulting in IsC.
A case of IsC was reported to be linked with Norovirus infection (Zenda et al. 2010). Moreover, IsC due to bacterial infection has been reported (Preventza et al., 2001). Although, the bacteria give damage to colon mucosa by bacteria toxin, enteric virus does not necessarily cause colonic mucosal damage by itself. This differentiates between these two types of IsC. We could show that bloody diarrhea occurred more frequently in viral infection than bacterial infection or no infection. Besides, IsC occur significantly in viral diarrhea (Figure 2). We are of the opinion that IsC can be induced by enteric virus infection. We suggest this pathway of IsC can explain unknown genesis of IsC, and in our series, fundamental condition was confirmed to be an important factor to induce IsC. As a past medical history, there were 45% of illnesses which led to vascular change.

The importance of predisposing vascular factors was therefore understood, but there remained unknown causes of IsC. As a result, we have gotten viral infection could induce IsC and could accelerate the patients who were loaded with any backgrounds to cause IsC. Colitis which is supposed to be IsC is not simple (Carlson, 2011). We could show the influence of viral infection by virus check in acute gastroenteritis. We strongly proposed enteric viral infection can induce IsC. Acute gastroenteritis are usually accepted as self-limited. Several physicians do not always examine enteric virus infection. We could show here epidemic result that there was concealed viral infection behind so-called IsC. It is important to keep in mind that some healthy people experience IsC only by one chance and we can detect viral infection in those so-called IsC. We should consider also that IsC could be induced by chance of viral acute gastroenteritis. We do not say that to deny any ever predisposing factors inducing IsC, but there are really no predisposing factors among IsC. We advocate the possibility when there is no suggestion to discriminate the clinical factor leading to IsC. Viral acute gastroenteritis should be selected as an inducing factor. We expect this confused IsC pathophysiology is made clear.

CONCLUSIONS

This study reveals enteric viral infection could be related as a trigger of ischemic colitis. Hence, an alternative pathway to quietly unknown ischemic colitis was considered. In conclusion, it is important to check enteric virus in so-called ischemic colitis.

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REFERENCES


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