Author's response to reviews

Title: Acute gastric dilatation due to a superior mesenteric artery syndrome : an autopsy case

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Version: 2 Date: 30 September 2013

Author's response to reviews: see over
Dear the Editor
We enclosed revised manuscript (Manuscript ID: 1106104689286886) for consideration as a publication in BioMed Central gastroenterology. We appreciate your review of this manuscript again.

Sincerely,

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The reviewer’s comments (bold font) and our replies (normal font) are provided below. Revised text is indicated in the manuscript by bold fonts.

Referee 1

Major

1. Please comment that there was no other possible cause of death in the autopsy. When she was found dead, a large amount of dark brown vomit was found on the floor around her. As a clinician, I suspect the possibility of asphyxia by vomitus. Is there any evidence of asphyxia in the autopsy?

Accordingly, we added the sentence: “No vomitus was observed in the larynx, trachea, bronchi, or alveolus. Asphyxia by vomitus was ruled out as the cause of death.” (Page 5, line 5. ~line 6.)
2. Authors described that “For the cause of the gastric dilation, intestinal occlusion by the SMA at the third portion of duodenum was suspected.” However, she had never complained symptoms suggesting SMA syndrome. And duodenum distal to SMA seemed to somewhat dilated in figure 2. So I think that excessive eating of large food was main cause of gastric dilation and the narrowing duodenum (not occlusion) could be a aggravation factor for gastric dilation, not leading cause. I agree with author’s summary “an excessively large mass of food in the narrowed duodenal lumen also promoted the development of acute gastric dilation in the case of SMA syndrome”.

We revised the two sentences as follows: “The cause of death was diagnosed as SMA syndrome with intra-duodenal aggregation of an excessively large mass of food in the narrowed duodenal lumen” (Page 2, line 16. ~ line 18.), and “This is an atypical case of SMA syndrome associated with intrinsic peritoneal degeneration, through an excessively large mass of food triggering the SMA syndrome” (Abstract, Page 3, line 1. ~line 3.)

3. Authors described that “The root of the mesentery was thickened and adhered to the duodenum, which also reduced the duodenal motility (Fig.2). In addition to these findings, a reduction of peristalsis in the duodenum might have enhanced gastric dilation in this case.” However, it cannot be assumed whether “motility” or “peristalsis” was really impaired by thickened mesentery by only autopsy findings in this patient. In addition, she had no previous gastrointestinal symptoms and was well nourished. Autopsy finding just showed narrowing of duodenum by thickened mesentery and SMA.

According to these comments, we added the histopathological findings showing necrotic duodenal mucosa and thickened mesentery in Figures 2B, and added the: “Dense fibrotic degeneration was found in the thickened peritoneum around the duodenum (Fig.2B)” (Page 5, line 15. ~ line 16.) in the Case Presentation section, and “Histopathology of lesion site in the duodenum (hematoxylin and eosin staining). Mucosal necrosis was apparent at the dilated proximal duodenum and there was dense fibrotic degeneration in the thickened peritoneum around the duodenum” (Page 13, line 17. ~ Page 14 ,line 2.) in the legend for Figure 2.

Referee 2
Major Compulsory Revisions
1. First paragraph of case presentation mentions that “the patient was found dead lying on her back with large amount of dark brown vomit on the floor around her.” This description suggests choking due to aspiration of large amount of vomitus that could be a
possible cause of death in this case. However autopsy finding of the lung including trachea-bronchial tree are not mentioned.

Accordingly, we added the sentence: “No vomitus was observed in the larynx, trachea, bronchi, or alveolus. Asphyxia by vomitus was ruled out as the cause of death.” (Page 5, line 5. ~line 6.)

2. Second paragraph of case presentation mentions that “left proximal part of greater curvature was gray colored and necrotic (Fig. 1)” However, the greater curvature appears normal in the photograph. The author should include closer view of greater curvature, cut section of stomach and histopathological findings showing transmural gastric necrosis to prove the point.

On histopathological examination, we could not confirm necrotic degeneration in the gray-colored part of gastric mucosa, because of postmortem autolysis. We therefore revised the sentences to: “The surface of the stomach showed pallor and the left proximal part of the greater curvature was gray colored (indicated by an arrow in Fig.1), which was suggestive of gastric necrosis” (Page 5, line 9. ~ line 11) in the Case Presentation section, and “The surface of the whole stomach showed pallor, and the left proximal part of the greater curvature was gray colored (indicated by arrow), which suggested the possibility of gastric necrosis.” in the legend for Figure 1 (Page 13, line 4. ~line 7.)

3. Second paragraph of conclusion mentions that “We suspected that past intrinsic peritonitis may have been the cause of the pathological changes.” The author has highlighted it as first case report of SMA syndrome due to intrinsic peritonitis. However neither there is any term like “intrinsic peritonitis” in the literature nor any cross reference has been provided in the text for this term.

According to the comment, we corrected this portion of Discussion as follows: “Some of the factors that have been reported to precipitate SMA syndrome are as follows: external compression of the abdomen induced by wearing tight belts, body spica, or body cast; anatomic anomalies such as lumbar lordosis [7] and high insertion of the ligament of Treitz; and surgical alterations of anatomy such as spine surgery and ileoanal pouch anastomosis [7,9,10]. None of these factors were identified in the present case, whereas a dense fibrotic and thickened degenerated area arose from the posterior parietal peritoneum around the duodenum (Fig.2B) and stretched to the root of the mesentery where the SMA was involved (Fig.2A). We could not confirm the subject’s past history of peritonitis, but the present pathological findings of
circumscribed peritoneal dense fibrosis provided definitive evidence of past intrinsic peritonitis. Therefore, we suspected that past intrinsic peritonitis may have been the cause of the pathological changes.” (Page 8, line 9. ~Page 9, line 2.)

Minor Essential Revisions
1. The heading of conclusion should be replaced as discussion.
According to the comments, we replaced the heading “Conclusion” with “Discussion”.

Referee 3

Major comments
What characteristics caused the lethal gastric dilation through SMA syndrome? Please refer previous lethal case reports about acute gastric dilation through SMA syndrome and author should discuss more.

In response to this comment, we have corrected the paragraph in the Discussion section as follows: “Acute gastric dilatation raises the intragastric pressure resulting in vascular insufficiency [6]. The pressure within the dilated stomach lumen must be greater than 20 cm H$_2$O, which exceeds the gastric venous pressure, before mucosal ischemia will occur [6]. The gastric circulatory deterioration causes fragility of the gastric wall, resulting in life-threatening complications such as dehydration, metabolic alkalosis, and gastric necrosis [2-5]. In addition, acute gastric dilatation increases intra-abdominal pressure and leads to systemic circulatory failure following collapse of the inferior vena cava [1]. The medical literature confirms gastrointestinal necrosis and intra-abdominal high pressure from marked distention of the stomach as features in lethal cases of SMA syndrome [11]. In the present case, the significant dilatation of the stomach (shown in Fig.1) indicated a large increase in abdominal pressure, which could have induced systemic circulatory failure. Taking these findings into consideration, we surmised that acute gastric dilatation was the cause of death in the present case.” (Page 6, line 11. ~Page 7, line 7.)

Minor comments
1) The author should show the microscopic findings of gastric necrosis in more detail.

On histopathological examination, we could not confirm necrotic degenerations in the gray-colored part of the gastric mucosa because of postmortem autolysis. We revised these sentences accordingly as follows: “The surface of the stomach showed pallor and the left proximal part of
the greater curvature was gray colored (indicated by an arrow in Fig.1), which was suggestive of
gastric necrosis” (Page 5, line 9. ~ line 11) in the Case Presentation section, and “The surface of
the whole stomach showed pallor, and the left proximal part of the greater curvature was gray
colored (indicated by arrow), which suggested the possibility of gastric necrosis.” in the legend
for Figure 1 (Page 13, line 4. ~line 7.)

2) The authors should comment whether there were focal necrotic change in the
duodenum or jejunum and thrombosis in the SMA and Celiac artery in the case
presentation.

According to the comments, we revised the text as follows: “the mucosal necrosis was
apparent at the dilated proximal duodenum (Fig. 2B)” (written on Page 5, line 11. ~line 12.),
and “No pathological abnormalities such as arterial thrombosis, arteriosclerosis, aneurysm, or
arteritis were observed at the SMA and celiac artery.” (Page 5, line 16. ~ line 18.) in the Case
Presentation section.