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Case Presentation

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Successful Non-Invasive Treatment of Diabetic Ketoacidosis Complicated with Gastric Abscess: A Case Report and Literature Review

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Abstract

Background: Gastric abscessis a entity which main caused by is the immunosuppression or the record of any invasive procedure. Diabetic ketoacidosis complicated with gastric abscess is rare, and the treatment may be challenging.

Case presentation: A 68-year-old male with a ten-year history of type 2 diabetes consulted our clinic for badly nausea and vomiting. He had poor blood glucose control and diabetic ketoacidosis, gastric abscess was found after abdominal computed tomography, gastroscopy and histologic biopsy. Gastric abscess is successfully cured by treatment with intravenous antibiotics and insulin. Meanwhile, we reviewed the therapeutical approaches were employed in the cases of diabetes complicated with gastric abscess.

Conclusion: Gastric abscessis a complication of type 2 diabetes. Effective antibiotics are key to the rehabilitation of this case.

Keywords: Diabetes mellitus; Diabetic ketoacidosis; Gastric abscess; Gastric wall abscess; Phlegmonous gastritis

Abbreviations: CT: Computed Tomography; ESR: Erythrocyte Sedimentation Coefficient; FPG: Fasting Plasma Glucose; HbA1c: Glycosylated Hemoglobin A1c; PCT: Procalcitonin; SARS-CoV-2: Severe Acute Respiratory Syndrome Coronavirus 2

Background

Gastric abscess, known as gastric wall abscess or phlegmonous gastritis is a rare acute suppurative inflammation of the gastric wall. It has a nonspecific presentation in the early stage and is easily misdiagnosed as acute cholecystitis, acute pancreatitis, ulcer perforation, liver abscess and other diseases **[1-3].** In the past, series cases of gastric abscess complicated with diabetes mellitus. Most of gastric abscess requires endoscopic surgery to abscess drainage, but an effective antibiotic is crucial. Here, we present successful antibiotic treatment in a patient with gastric abscess. Meanwhile, we reviewed the 15 case reports of diabetes complicated with gastric abscess from 1980 to the present time.

Case Presentation

A 68-year-old male with a ten-year history of diabetes consulted our clinic for nausea and vomiting. Over the past few years, he has taken metformin and miglitol orally to control glucose. On admission, he vomited, emptied his stomach, and experienced acid reflux and abdominal distension, but no abdominal pain, diarrhoea or melena. There was no history of nonsteroidal anti-inflammatory drugs, immunosuppressive agents, lcohol or endoscopies. About family history, his daughter and cousin had diabetes.The patient had clear consciousness, poor eyesight, and no obvious heart or lung abnormalities? The upper abdomen was mildly tender, with no rebound pain or muscle tension. The superficial sensation in both hands and feet was slightly reduced. Outpatient lab tests showed Fasting Plasma Glucose (FPG) 26.46 mmol/L (normal range, 3.9~6.1 mmol/L), glycosylated hemoglobin A1c (HbA1c) 12.5% (normal range, 4%-6%), urinary ketone bodies (3+) (normal range, negative), blood ketone concentration 4.0 mmol/L (normal range,0.03-0.5mmol),bicarbonate concentration 14.0 mmol/L (normal range, 22-26 mmol/L), venous pH 7.2 (normal range, 7.35-7.45).

One hour after admission, the patient still experienced nausea, vomiting and discomfort, and his body temperature rose to 38.5°C. The results of the laboratory tests show that White Blood Cell (WBC) 25.88 *10^9/L (normal range, 4-10*10^9/L), Percentage of neutrophils (Neu%) 89.7% (normal range, 50.0-70.0%),

Procalcitonin (PCT) 4.28 ng/ml (normal range, 0~0.3 ng/ml), C-Reactive Protein (CRP) 112.71 mg/L (normal range, 0-10 mg/L), Erythrocyte Sedimentation Rate (ESR) 27.32 mm/h (normal range, 0-20 mm/h). The above information suggested severe hyperglycemia and infection. The patient underwent physical cooling and compound amoxicillin barbital injection and was empirically administered intravenous infusion of ceftazidime (2g, q8h) for anti-infective treatment, rehydration, gastric acid inhibitor and gastric mucosa protective treatment. Low-dose insulin (conventional insulin, 0.1 U/kg/h) reduced his blood glucose level to 13.9 mmol/L, at which point the patient was switched to a continuous subcutaneous injection of insulin aspartate. An emergency Computed Tomography (CT) scan of the upper abdomen showed thickening of the gastric wall and esophageal wall and multiple small lymph nodes around the hepatogastric ligament **(Figure 1).** Gastroscopy was recommended to eliminate the possibility of gastrointestinal infection, and shown esophageal ulcers, inflammatory lesions in the gastric fundus and exudative yellow pus **(Figure 2).**

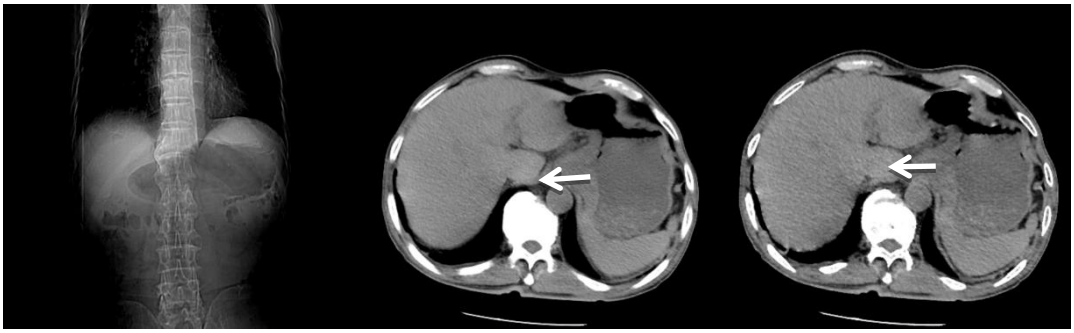

Figure 1: The wall of the stomach is thickened, and there are many small lymph nodes around the hepatogastric ligament.

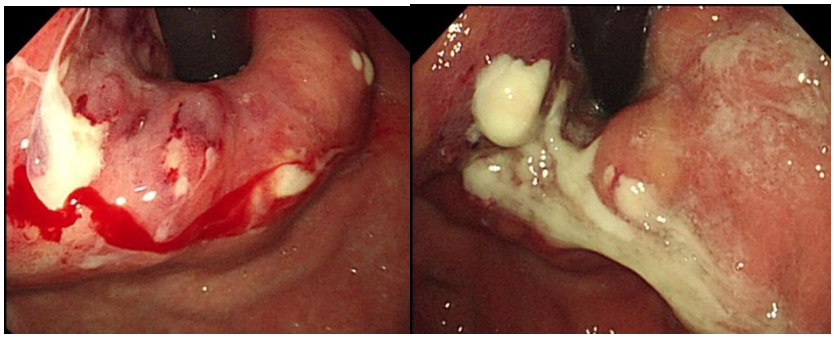

Figure 2: The back wall of the upper part of the cardia is slightly curved, the stomach wall is swollen, the surface is congested, and there are many ulcers approximately 0.3-0.6 cm in diameter on the surface. Yellow and white viscous purulent secretions are seen flowing out, which remain after repeated washing, and the biopsy is soft.

Four days after admission, his nausea, vomiting and fever were relieved, appetite and mental state improved, and blood glucose control was stable. Blood tests demonstrated WBC 15.54*10^9/L, Neu% 76.4%, and PCT 2.77 ng/ml.The inflammation was improved and the treatment was effective. Nine days after admission, blood tests revealed that the infection parameters of the patient were obviously improved (WBC 4.33*10^9/L, Neu%55.1%, CRP 5.8 mg/L). Enhanced CT scans at the twelfth day after admission, showed that the wall of the thoracic segment of the esophagus, gastric cardia and gastric fundus was thickened, with even enhancement.The surrounding fat space was blurred. There were inflammatory lesions and multiple small lymph nodes around the mediastinum and hepatogastric ligament **(Figure 3).** Repeated gastroscopy showed chronic nonatrophic gastritis and gastric mucosal infectious lesions, suggesting partial absorption of the gastric wall abscess **(Figure 4).**

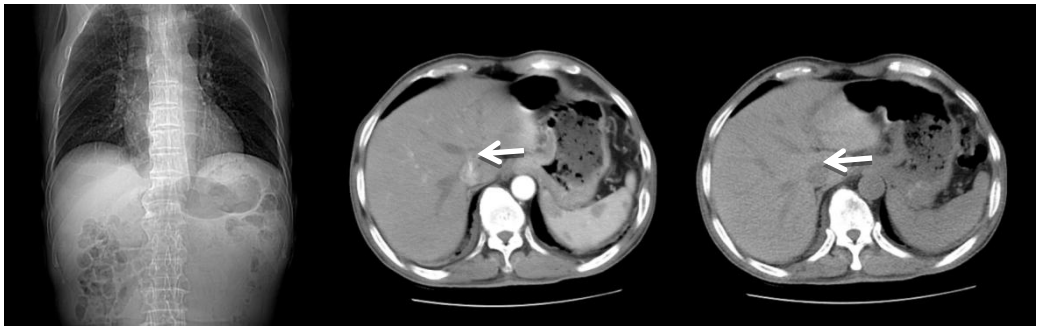

Figure 3: The wall of the gastric cardia and fundus was obviously thickened, the enhancement was uniform, the surrounding fat space was slightly blurred, and there were many small lymph nodes around the mediastinum and hepatogastric ligament.

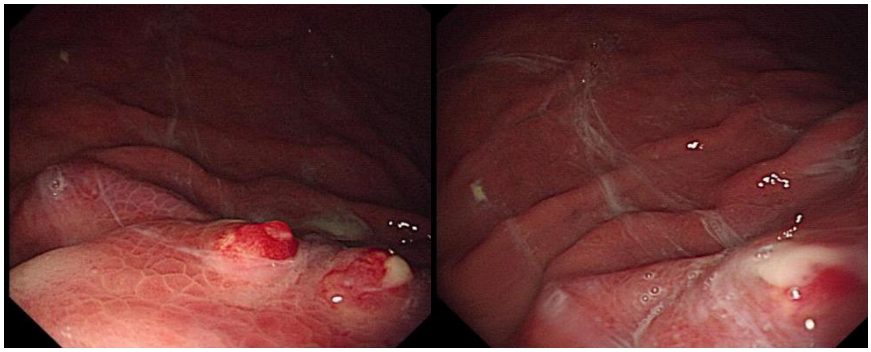

Figure 4: In the upper part of the stomach, along the posterior wall and near the cardia, the mucosa was prominent. The top was hyperaemic, and covered with a small amount of purulent secretions. The mucosa of the fundus, horn and antrum was smooth.

After 22 days of continuous treatment, gastroscopy was performed again and showed chronic nonatrophic gastritis with complete absorption of the gastric abscess **(Figure 5),** the patient recovered and was discharged.

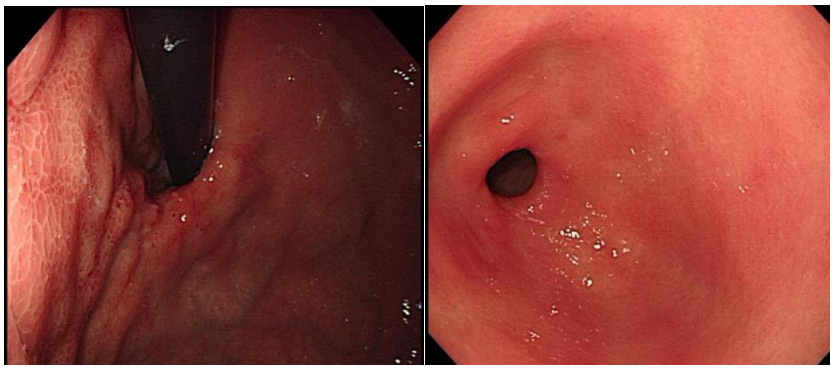

Figure 5: The mucosa of the posterior wall of the upper part of the lower body of the cardia was congested and slightly reddened. There was no obvious uplifting of the gastric wall, and there was a small amount of white secretions on the surface.

The culture results of the abscess secretion of the patient were Streptococcus. He is diagnosed with gastric wall abscess. Pathological examination showed moderate chronic inflammation of the mucosa with erosion and negativity for HP **(Figure 6).** After one month of follow-up, the patient had no obvious discomfort.

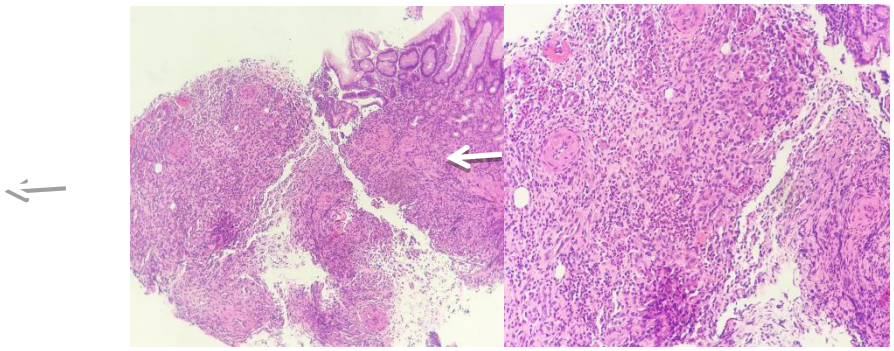

Figure 6: (CARDIA) Biopsy tissue shows moderate chronic inflammation of the mucosa with erosion and negativity for HP.

Literature review

From 1980 to the present, a total of 15 cases of diabetic gastric abscess were reported, including 2 cases with esophageal abscess, the youngest age was 30 years old, the maximum age was 77 years old, 11 (73.3%) cases

were male, and 4 (26.7%) cases were female. Only 6 (40.0%) patients reported glycosylated hemoglobin values, with a median of 10%. Most of the symptoms are fever, abdominal pain, nausea, vomiting, and shock. Pathogenic bacteria were cultured in most cases, mainly Klebsiella pneumoniae and Streptococcus, and multiple pathogenic bacteria were cultured in some cases. Most patients were treated with antibiotics combined with surgery or endoscopy on the basis of blood glucose control, and only 3 (20.0%) patients were treated with medical treatment alone. The prognosis of most patients was good, and 5 (33.3%) patients died, but the hospital stay was long, with the shortest being 10 days (improved) and the longest being112 days **(Table 1).**

Table 1: Summary of clinical data of cases of gastric abscess with diabetes reported between 1980 and 2022.

Authors	Year	Type	Age /sex	Underlying Medical Condition	HbA1c	Sypto ms Bacte ria	Culture	Treatment Method	Result/ days
Bracco E et al [4]	1987	phlegmo nous gastritis	51 y/M	DM + alcoholis m	No ne	Naus ea, vomit ing	*None*	ATB	Death/ None
O'Toole et al [5]	1988	phlegmo nous gastritis	30 y/M	DM + alcoholis m	No ne	Naus ea, vomit ing, abdo minal pain	*S. pyogenes*	No	Death/ None
Hsu CY et al [6]	1996	phlegmo nous gastritis	42 y/M	DM + alcoholis m	No ne	Fever , chest pain, short	*K. pneumoniae*	ATB, esofagecto my and gastrectom y	Cure / 90d

						ness of breath			
Kim GY et al [7]	2005	phlegmonous gastritis	60y/F	DM + cirrhosis	None	Nausea, vomiting	*Clostrium seticum*	ATB	Death/None
Iwashima Y et al. [8]	2005	wallabscess gastric	67y/F	DM	None	Fever	*None*	ATB	Cure/None
Hironari Ajibe et al. [9]	2008	Phlegmonous gastritis	74y/M	DM + CKD, HD	None	Fever, epigastric pain	*Citrobacter freundii, Enterobacter cloacae, oc-Streptococcus*	ATB, total gastrectomy	Cure/27d
Guisado P et al [10]	2010	phlegmonous gastritis	50y/M	DM	None	nausea, vomiting, epigastric pain	*None*	ATB	Cure/None
Kim HS, et al. [11]	2010	phlegmonous esophagogastritis	48y/M	DM	None	Chest pain, abdominal pain,	*Klebsiella pneumoniae*	ATB, Surgery	Cure/73d

						dyspnea			
Masaya Morimoto et al. [12]	2014	Phlegmonous gastritis	77y/M	DM	6.40%	Fever, nausea, vomiting,	*Group A streptococcus*	ATB, ICU	Death/None
Kim KH.et al. [13]	2016	Gastritis Phlegmonous	74y/M	DM+hypertension, alcoholic liver cirrhosis, Early Gastric Cancer	None	None	*None*	ATB, endoscopic ablation	Improve/14d
Huang YC et al. [14]	2017	Phlegmonous Esophagogastritis	60y/F	DM	11.30%	Fever, fatigue, vague chest pain	*Pseudomonas aeruginosa, Klebsiella pneumoniae, Pseudomonas aeruginosa*	ATB, video-assisted thoracic, surgery	Cure/56d
Matsuura et al [15]	2018	Phlegmonous gastritis	76y/F	DM + MDS	7.00%	Fever	*Klebsiella pneumoniae, Pseudomonas aeruginosa, Candida*	ATB	Cure/112d

							albicans		
Miyaguchi et al [16]	2020	Perigastric abscess	70y/M	DM	10.00 %	Fever, abdominal pain	*Klebsiella pneumoniae*	ATB, Laparoscopic removal	Cure/27d
Mana Modares et al [17]	2021	Phlegmonous gastritis	67y/M	DM	None	Fever, nausea, vomiting, epigastric pain	*Group A streptococci*	ATB, Roux-en-Y, esophagojejunostomy and jejunojejunostomy reconstruction	Cure/90d
Andrea DeCino. et al [18]	2021	Phlegmonous gastritis	47y/M	DM+ hypertension	11.90 %	fevers, abdominal pain, vomiting	beta-hemolytic streptococcus	ATB	Cure/10d

M: Male; F: Female; DM: Diabetes mellitus; ATB: Antibiotics; ICU: Intensive care unit; MDS: Myelodysplastic syndrome; CKD: Chronic kidney disease; HD: Hemodialysis

Discussion and Conclusions

We present a rare case of diabetic ketoacidosis complicated with gastric abscess. Our literature review shows that the mortality rate of gastric abscesses with diabetes is 33.3%, which is higher than the 27% mortality rate of general gastric abscesses **[15].** Moreover, the most common precipitating causes of diabetic ketoacidosisis infections **[19].** The main mechanism is hyperglycemia leading to decreased chemotaxis, migration, phagocytosis and bactericidal activity of neutrophils, lower immune function, decreased oxygen dependent bactericidal action of leucocytes, and enhanced bacterial growth and reproduction **[20,21].**

The pathogenesis of gastric wall abscesses includes direct invasion by microorganisms and their hematogenous dissemination to generate distant lesions. The causes may include gastric lesions, gastric wall injury, infection of adjacent tissues, low immune function, drinking, and eating raw and cold food **[22].** Theoretically, diabetes mellitus is a risk factor for gastric abscess, but it has rarely been reported in clinical practice **(Table 1).** The possible reason is that the blood flow in the gastric wall is abundant, vascular involvement is not severe, and gastric acid has an antibacterial effect. This patient was elderly and had poor blood glucose control for more than 10 years. He suffered severe microvascular complications and hyperglycemia. His gastrointestinal adverse reactions were severely related to metformin and miglitol. The above factors may be the causes of gastric abscess in this patient. Gastric wall abscess is a serious and rare suppurative condition that develops rapidly. The conventional treatment of gastric abscesses is intravenous antibiotics, endoscopic abscess puncture or incision and drainage, gastric surgery and so on. The mortality rate was as high as 66.7% in the past, which was significantly higher than the current mortality rate **[15-27].** This may be related to the current widespread application of endoscopy and CT, the ability to achieve a diagnosis in a timely manner, the application of appropriate treatment, the rational use of antibiotics, and adequate experience treating this condition. In the treatment of this case, we administered intravenous insulin and a large amount of fluids in a timely manner to improve his high sugar level and then used an insulin pump to continuously inject subcutaneous insulin to control his blood glucose level. After achieving blood glucose control, we provided empirical antibiotic treatment, acid suppression, stomach protection and other treatments in a timely manner; performed abdominal CT and gastroscopy to support an accurate diagnosis; and formulated a reasonable treatment plan through multidisciplinary consultation. These methods were effective, and the patient was discharged after comprehensive treatment. Although the treatment of gastric abscesses often requires surgical intervention, it is not necessary. Previous literature reviews also suggested that diabetes mellitus complicated with gastric abscess could be cured only by medication **[4,7,8,10,15,18].** From this case, we can see that even in the extreme

condition of diabetes ketoacidosis, gastric abscess can be obtained through nonoperative intervention with timely diagnosis and early effective antibiotics and blood glucose control.

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