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Case Report

Gastric adenocarcinoma due to *H. Pylori* infection on patient with chronic kidney disease on hemodialysis

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ABSTRACT

A 53-year-old Male was admitted to our emergency department presenting with severe anemia (Hb 6.9 gr/dl) with a history of chronic kidney disease on routine hemodialysis twice a week and insulin-dependent type 2 diabetes. He was diagnosed with gastric adenocarcinoma after biopsy sample acquired from endoscopy confirmed by pathologist. The patient also has a positive HPSA test result. He was given rapid insulin 3 times daily and long-acting insulin once daily, continued routine hemodialysis and planned to do partial gastrectomy after recommended serum-creatinine level and minimum hemoglobin level are reached. We will take into consideration the routine use of anti-platelets and anti-coagulants during hemodialysis and how it will affect our decision to do surgery. Currently the patient still discusses the option of surgery even though it has been suggested by the digestive surgeon. In conclusion, we are discussing the difficulties of managing patient with gastric adenocarcinoma caused by *H. pylori* infection with chronic kidney disease and diabetes as the aggravating factors, including the use of routine medication and managing the blood sugar.

Keywords: *H. pylori* infection, Peptic ulcer disease, Gastric adenocarcinoma, MALT

INTRODUCTION

H. pylori infection is a gram negative spiral shaped bacterium that affects up to 50% of the population worldwide, with a higher prevalence in developing countries.[1] It is the most important cause for chronic or atrophic gastritis, peptic ulcer, gastric lymphoma, and gastric carcinoma.[2] High *H. pylori* prevalence has been reported in Russia, Jordan, Iran, China, and Latin American countries as well as in Arctic populations in Canada. Indigenous inhabitants in the Arctic were found to be infected substantially more frequently than non-indigenous inhabitants.[3] In 2015, the Kyoto global consensus reported on *H. pylori* gastritis. According to the consensus report, patients diagnosed with *H. pylori* infection should receive eradication therapy to minimize the risk of long-term sequelae including peptic ulcer disease, gastric adenocarcinoma, and gastric mucosa-associated lymphoid tissue (MALT) lymphoma.[4]

CASE REPORT

A 53-years old Male complained of general weakness and shortness of breath since 2 days ago. The patient has a history of end stage chronic kidney disease with routine hemodialysis since 4 years ago, the patient also has a history of hypertension and type 2 diabetes mellitus controlled with valsartan 160 mg, rapid acting insulin (3×8 IU) and long acting insulin (1×8 IU) and heart failure caused by hypertensive heart disease controlled by spironolactone, clopidogrel, ISDN, and carvendilol.

The patient also complained of experiencing Nausea and vomiting since admission which does not subsides even

after IV omeprazole 2×20 mg daily and ondancetron 2×8 mg IV. He was diagnosed with chronic gastritis. However, because treatment courses failed to alleviate his symptoms, patient are scheduled to undergo esophago-gastroduodenoscopy as a diagnostic procedure. During endoscopy, following results are obtained, (Figure 1).

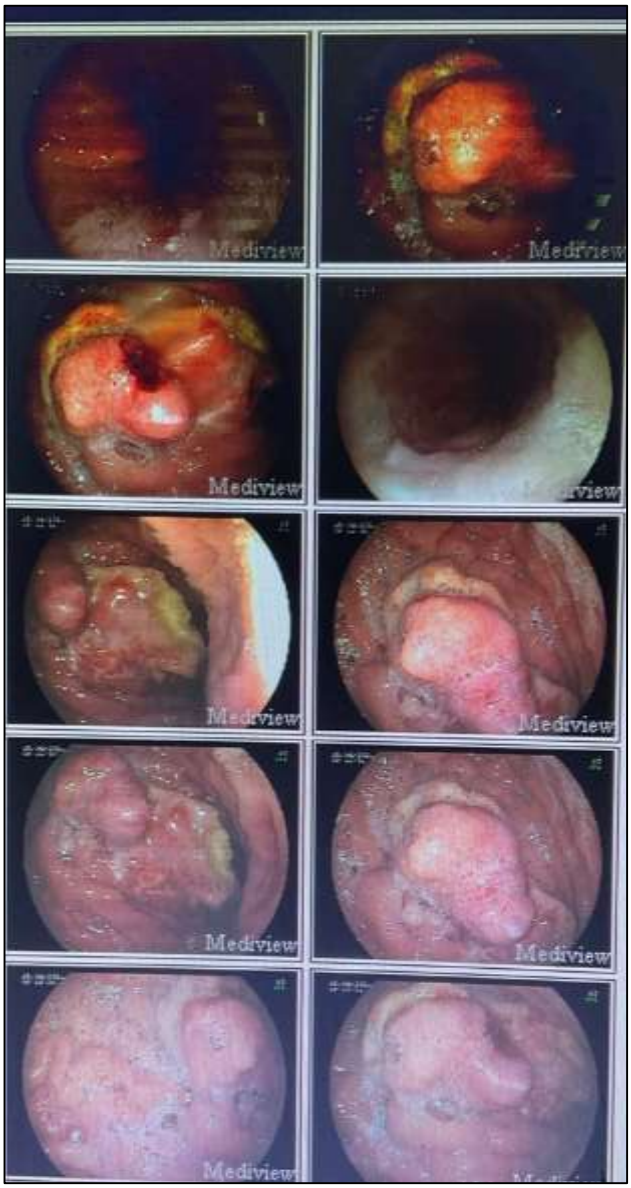

Figure 1: Camera images obtained during endoscopy.

With irregular mucosal pattern, growth abnormality, and easily bleeding gastric mucosa, gastric adenocarcinoma is expected. To confirm the diagnosis of gastric adenocarcinoma, some histological samples are obtained to be checked by pathologist, and the following results are obtained in Figure 2.

Figure 2 revealed adenocarcinoma gastric. The tissue section preparation contains a tumor mass with proliferation of neoplastic cells forming an infiltrative glandular pattern in the stroma. The cells are cuboidal to columnar in shape, the N/C ratio is increased, nuclear pleomorphia is moderate, hyperchromatic part of the vesicular nuclei with prominent nuclei. Mitosis can be found. There were also lymphoplasmacytic inflammatory cells in the surrounding stroma. Conclusion Adenocarcinoma, moderately differentiated.

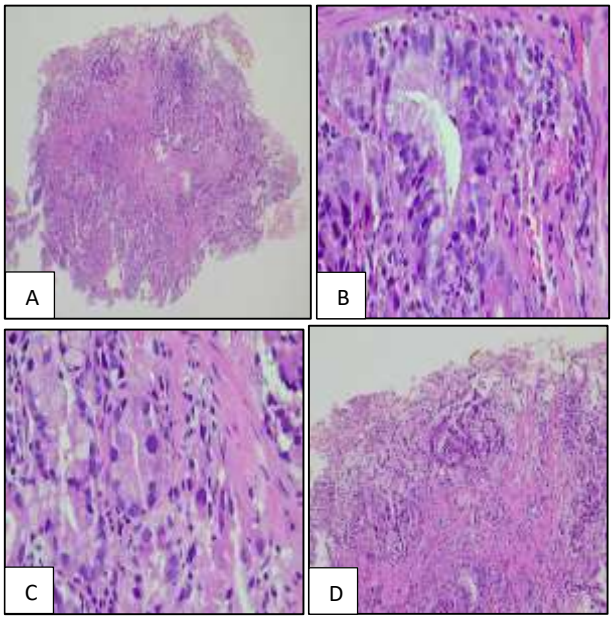

Figure 2 (A-D): Histological samples from biopsy during endoscopy.

After gastric adenocarcinoma is confirmed, the patient is planned to do *H. pylori* stool antigen, after the stool sample is obtained, which shows reactive (+) result after test. The patients are then consulted to digestive surgery department and planned to do partial gastrectomy if possible. The difficulties appeared when preparing the patient for surgery including to stop the use of anticoagulant and antiplatelet while the patient is on hemodialysis, to control the blood sugar, to do blood transfusion to an acceptable level, and all while keeping the patient vitals' stable for surgery. The patient's family still weigh the risk and benefit for the operation and no decision has been made.

DISCUSSION

Interest in *H. pylori* as a cause of cancer began after the pioneering discoveries of Marshall and Warren in the 1980s.[5] Before the organism was discovered, it was understood that gastric adenocarcinomas usually developed in areas affected by gastritis. Once the connection between *H. pylori* and chronic gastritis was identified, researchers started to explore *H. pylori*'s potential role in causing gastric cancer. The initial research efforts investigating the link between *H. pylori* and gastric cancer were ecological in nature, assessing the correlation between regional *H. pylori* prevalence and the occurrence of gastric cancer.[6] Unique among bacteria, it finds a niche in both the antral and fundic mucosa of the stomach under the mucus gel. The presence of infection is universally associated with chronic and acute inflammation and, more

variably, with other gastric lesions, including lymphoid follicles, atrophic gastritis and intestinal metaplasia. Treatment with antimicrobial agents causes inflammation to regress over time.[7]

The most compelling observational evidence of an association between *H. pylori* infection and gastric cancer comes from longitudinal cohort studies. In a large prospective trial conducted in Japan, 36 out of 1246 infected individuals developed gastric cancer compared to none of 280 uninfected participants (infinite OR).[8] A prospective study of 1225 Taiwanese patients confirmed this 'infinite' OR (p=0.015).[9] Based on the amalgam of observational and experiment studies, the attributable risk of gastric cancer in the population (i.e. the proportion of gastric cancer in the population that would not occur were the *H. pylori* not to exist) has been estimated to be 75%.[10] If this is accurate, *H. pylori* would be responsible for as many as 5.5% of all cancers, making it the leading infectious cause of cancer worldwide and second only to smoking as a defined cause of malignancy

Although mechanisms of *H. pylori*-induced carcinogenesis are only beginning to be understood, inflammation is the most commonly cited factor in the carcinogenic process. Inflammation is thought to induce cancer by increasing production of free radicals, increasing apoptotic and necrotic epithelial cell death and augmenting cell proliferation.[11-13] To compound these pro-carcinogenic processes, *H. pylori* has been noted to reduce DNA repair *in vivo* and *in vitro*.[14]

Although half of the world's population is infected with *H. pylori*, only a minority of individuals (estimated 1-2%) progress to gastric cancer over a lifetime. This percentage is deceptively low, however, because children and young adults are included in the lifetime risk assessment. When only middle-aged adults are considered, the risk of cancer appears more substantial. For example, in prospective studies conducted in Asia, between 3% and 6% of *H. pylori*-infected subjects developed gastric cancer within a decade.[15] Yet, worldwide, the great majority of adults infected with *H. pylori* survive without ever suffering from malignancy. Unfortunately, the reason some develop cancer and others do not is incompletely understood.

H. pylori is responsible for the majority of gastric cancers worldwide. Understanding this gives us the potential to control and perhaps eradicate this deadly disease. However, to date, there is no consensus on whether to completely eliminate this bacterium from the human body. The appropriate response might differ among various populations based on their risk levels for the disease. Without human intervention, it is fortunate that *H. pylori* is naturally declining in many populations. This ongoing natural phenomenon provides an excellent opportunity to gather more data, which will undoubtedly help us determine the most effective global strategy for managing this infection and its associated diseases.[16]

While the treatment for this particular disease outweighs the risk, because of the complication brought by the patient itself (including Kidney failure and diabetes) which worsens the chance of surviving such major surgery, even after explanation from multiple healthcare professionals, it is understandable that the family is reluctant to undergo such major surgery.

CONCLUSION

This case report further acknowledges the correlation between *H. pylori* infection and gastric adenocarcinoma, further study may be required to exact the correlation between the infection and malignancy.

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