

## A case report of cytomegalovirus associated gastritis

Sitomegalovirüse bağlı gastritis; Olgu sunumu

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*Cytomegalovirus is one of the most common viral pathogens, which, in some cases, leads to organ specific infections such as gastrointestinal manifestations that have a significant risk of morbidity and mortality. However, cytomegalovirus infection is rare in an immunocompetent host. Herein, we report a case of cytomegalovirus gastritis in a 54-year-old immunocompetent patient, diagnosed through upper gastrointestinal endoscopy with biopsies and serology.*

**Key words:** CMV, gastritis

### INTRODUCTION

The infection caused by cytomegalovirus (CMV) is diverse and depends greatly on the host. It is a common infection in immunocompetent hosts but is generally asymptomatic, or may present with a mononucleosis-like syndrome of fever, lymphadenopathy and pharyngitis. The prevalence of CMV infection ranges from 40 to 100% of the adult population (1). An uncommon manifestation of CMV is organ specific, which can involve the gastrointestinal system. CMV infections in immunocompromised hosts such as those with human immunodeficiency virus (HIV), malignancy, or organ transplantation, can lead to substantial morbidity and mortality. Literature reports on the occurrence of organ specific CMV infection is limited however, to small series and case reports. (2) CMV infection in the stomach can lead to gastric ulcers that are difficult to differentiate from *Helicobacter pylori*-or non-steroidal anti-inflammatory drug-related ulcers; characteristic morphological features specific to CMV, immunohistological detection of CMV and polymerase chain reaction (PCR) amplification detection of CMV DNA may help differentiate between the two. (3) Further, it is further unclear if and when antiviral therapy is indicated. A recent study suggest that antiviral therapy such as ganciclovir should be considered in immune-incompetent patients and in immuno-

*Sitomegalovirüs, bazı durumlarda morbidite ve mortalite ile önemli gastrointestinal manifestasyonlar gibi bazı organ spesifik enfeksiyonlara yol açan yaygın bir viral patojendir. Immün sistemi sağlam bir konakçıda sitomegalovirüs enfeksiyonu nadirdir. Biz burada biyopsiler ve seroloji ile üst gastrointestinal endoskopide tanı konulan 54 yaşındaki bir hastada sitomegalovirüs gastrit olgusunu sunuyoruz.*

**Anahtar kelimeler:** CMV, gastritis

competent patients who are male, age 55 years or older, and those with chronic disease. (4)

In this case report we describe an immunocompetent adult who presented with epigastric pain and weight loss. An upper gastrointestinal endoscopic examination revealed gastritis. Biopsies showed viral inclusion bodies characteristic of CMV.

### CASE

A 54-year-old Turkish female who was previously healthy consulted a physician for a second opinion after being diagnosed with nonspecific gastritis. She presented with complaints of increasing epigastric pain, nausea, vomiting and weight loss during a two month period. These symptoms suddenly occurred. Her medical history included hypothyroidism following treatment with iodine 131 for hyperthyroidism. The patient had no history of organ transplantation, or malignancy, and was HIV negative. She neither took medications such as non-steroidal anti-inflammatory drugs (NSAIDs) nor had undergone immunosuppressive therapy. She had no history of alcohol abuse.

On physical examination her abdomen was soft, painful - specifically in epigastrio, with normal bowel sounds. Super-

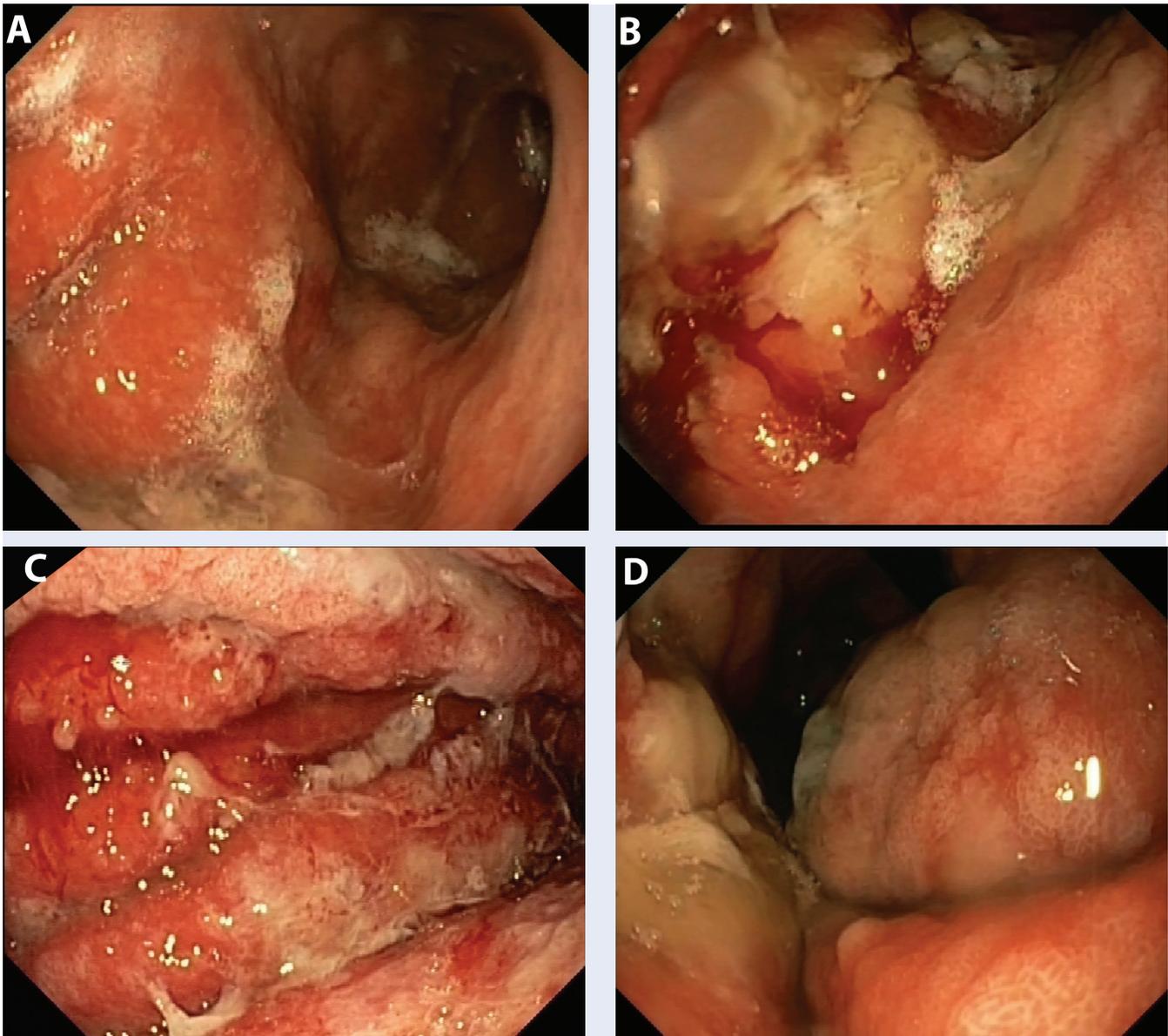


Figure 1. A,B,C, and D. Diffuse erosive gastritis of the corpus

ficial cervical, axillary and inguinal lymph nodes were not enlarged or palpable. Laboratory examination on admission revealed hemoglobin 8.8 mmol/L, leukocytes  $8.2 \times 10^9/L$ , C-reactive protein (CRP) 9mg/L. Further analyses of liver and renal function showed no abnormalities.

An abdominal computed tomography (CT) scan revealed a diffuse thickened stomach wall with several enlarged lymph nodes. We needed to distinguish the gastric lesions from linitis plastica or primary gastric lymphoma because of the thickened gastric wall seen on the CT abdomen scans. The patient underwent an upper gastrointestinal endoscopy, which revealed a normal oesophagus but diffuse erosive gastritis of the stomach, no signs of linitis plastica. Mucosal biopsies were obtained and submitted for histological examination. A duodenal gavage was placed during endoscopy, over which the

patient was fed for nutritional support. The initial treatment did not include antiviral therapy.

Multiple biopsies of the stomach mucosa revealed chronic active inflammation with intranuclear eosinophilic inclusion bodies within epithelial cells, consistent with a CMV infection. The biopsies further showed intestinal metaplasia, no dysplasia. No *Helicobacter pylori*, parasites or atypical mycobacteria were objectified and the Ziehl Nielsen colouring was negative. Screening for tuberculosis (TBC) was negative. Serology test for HIV was negative, CMV immunoglobuline M (IgM) negative, immunoglobuline G (IgG) positive and PCR was moderately positive for CMV. Immunological examination did not show signs of lymphoma. Following the careful examination of the multiple mucosal biopsies, the patient was diagnosed with CMV gastritis.

The initial symptoms of epigastric pain, nausea and weight loss increased over the next couple of weeks. The patient was treated with valganciclovir for three weeks and the symptoms of pain and nausea improved. Follow up endoscopy examination was performed after two months and showed improvement of the mucosal ulceration, and the histological examination showed less inflammation. Inclusion bodies had disappeared in the gastric biopsies.

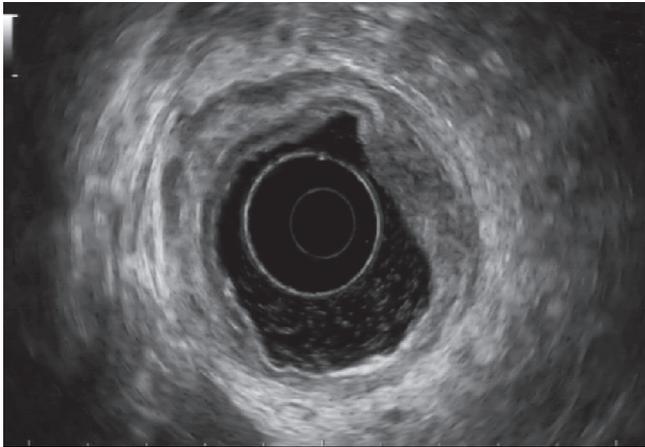


Figure 2. EUS picture of the stomach.

## DISCUSSION

Although CMV infections in the gastrointestinal tract of immunocompromised individuals have been reported with increasing frequency in recent years, there have been only a few reports of CMV infections in the gastrointestinal tract of immunocompetent individuals. We report a case of CMV gastritis in an immunocompetent patient. We continue to evaluate the patient's immuno-status; thus far no abnormalities have been found. The relationship between symptomatic ulcer di-

sease with the finding of CMV inclusions in mucosal biopsies of ulcers is unclear. It is unknown whether CMV is a primary cause of gastrointestinal lesions or whether CMV colonizes pre-existing lesions.

We needed to distinguish the gastric lesions in the present case from linitis plastica and primary gastric lymphoma, as it needed further evaluation and treatment. We diagnosed the gastric lesions as CMV gastritis due to the specific histological findings, serological markers and PCR. The endoscopic findings of CMV gastritis are not different from other etiologies such as NSAID's and *Helicobacter pylori*. Therefore, the diagnosis of CMV gastritis is primarily based on the histological finding of inclusion bodies characteristic for CMV infection. Furthermore, the number of inclusion bodies found does not correlate with the severity of inflammation in the gastrointestinal tract (5).

It is sometimes speculated that the CMV infection that is found has no direct effect on the lesions, and is defined as a co-localized. However, in this case, the patient's symptoms disappeared after treatment and follow-up endoscopy showed healing of the ulceration. Subsequently the stomach biopsies did not show inclusion bodies after treatment. From these findings, we reached the conclusion that CMV infection itself contributed to the formation of gastric ulcerations.

CMV is usually asymptomatic or seen as a mononucleosis syndrome and self-limiting. CMV is a unique disease that rarely causes gastrointestinal involvement in immunocompetent patients. This case demonstrates that CMV should be taken into account when diagnosing patients with gastritis. Since it is uncommon in immunocompetent hosts, an attempt should be made to exclude immunodeficiency. Furthermore, a conservative approach, avoiding anti-viral medications, can be effective.

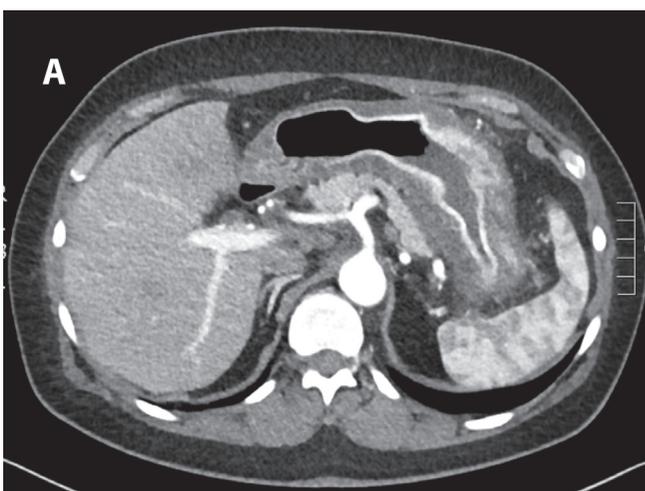


Figure 3. A,B CT abdomen

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