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CASE REPORT

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Good's syndrome combined with CMV gastroenteritis: A case report and literature review

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Key Clinical Message

Good's syndrome (GS) presents with thymoma, hypogammaglobulinemia, and recurrent infection. The manifestations of patients diagnosed with GS and *Cytomegalovirus* (CMV) gastroenteritis are rare and non-specific. Early diagnosis and treatment can improve the prognosis of the rare disease.

Abstract

Good's syndrome (GS), a rare acquired immunodeficiency condition, is characterized by thymoma, hypogammaglobulinemia, and low peripheral B-lymphocyte count. GS tends to occur in individuals aged 40–60 years, resulting in increased risk of recurrent infections with various conditional pathogenic bacteria, viruses, and fungi. *Cytomegalovirus* (CMV) can cause pneumonia, retinitis, encephalitis, and enteritis in GS patient, but CMV infection in the alimentary tract is usually underestimated, delayed diagnosed and misdiagnosed. In this study, we reported a female patient with GS and chronic diarrhea due to CMV infection and reviewed the literature to conclude the characteristics of this rare condition to improve the clinical diagnosis and prognosis of CMV gastroenteritis in patients with GS.

KEYWORDS

chronic diarrhea, *Cytomegalovirus*, gastroenteritis, Good's syndrome

1 | INTRODUCTION

Good's syndrome (GS), a rare acquired immunodeficiency condition, first described by Dr. Robert Good in 1954, is characterized by thymoma, hypogammaglobulinemia, and low peripheral B-lymphocyte count. GS tends to occur in individuals aged 40–60 years, resulting in increased risk of recurrent infections due to pathogenic bacteria, viruses, and fungi.[1,2] Almost half of GS patients suffer from chronic diarrhea due to opportunistic infections caused by, for example, *Salmonella* spp., *Campylobacter. jejuni*, *Clostridium difficile*, and *Cytomegalovirus* (CMV).[3–5] CMV, a kind of encapsulated double-stranded DNA β-herpesvirus, is a common virus that can infect human beings. CMV could cause pneumonia, retinitis, encephalitis, and enteritis in patients with GS.[4,6–14] In this study, we reported a female patient with GS and chronic diarrhea due to CMV infection and reviewed literature of related cases to conclude the characteristics of this condition to decrease the misdiagnosis and delayed diagnosis and improve the clinical diagnosis and prognosis of this rare disease.

2 | CASE HISTORY

A 64-year-old female hospitalized in our center complained that she suffered from intermittent diarrhea for more than 11 years, and her stool was watery, 5–6 times

per day. The patient went to the local hospital 9 years before admission to our hospital, and her laboratory examination showed reduced globulin (14.3 g/L), hypokalemia (3.07 mmol/L), and hypocalcemia (2.03 mmol/L). She underwent colonoscopy, and revealed scattered congestion and erosion throughout the colon, and chronic inflammation was found in intestinal mucosa biopsy. Meanwhile, she received chest computed tomography (CT) examination, which showed that there was a mass (about 5×4×4 cm) in the right mediastinum with rich blood supply. Subsequently, she was scheduled for mediastinal tumor resection, and the histopathological report of the tumor was type A thymoma (World Health Organization classification). The symptom of diarrhea could be alleviated with intermittent medication. However, the disease continued to recur.

The patient had severe diarrhea, abdominal distension, fatigue, dizziness, weight loss, and numbness of hands and feet 2 months before coming to hospital. She then attended the local hospital again. Laboratory findings are shown in Table 1.

Gastroscopy and colonoscopy were performed. The results of gastroscopy showed swelling of the gastric mucosa and gastric body with scattered erythema. The biopsy results using immunohistochemistry showed positive for CMV and negative for *Helicobacter pylori*. Meanwhile, colonoscopy revealed rough swelling, hyperemia and erosion of the descending colon mucosa, rough rectal mucosa, and obvious congestion and swelling of other colons mucosa. Immunohistochemistry of biopsy showed CMV+, PCK+, CD4+, CD8+, Ki-67+. Therefore, the final diagnosis of the patient was CMV gastroenteritis. She received cefotaxime and levofloxacin for treating infections, ganciclovir (250 mg Q12h × 14 days) for treating virus, human immunoglobulin (10 g × 5 days), potassium supplementation, spasmolysis, oral montmorillonite powder, and probiotics. However, she still had recurrent symptoms and then came to our hospital.

TABLE 1 Laboratory results of the patient.

Items	Results	Reference ranges
White blood cell count (WBC, $\times 10^9$/L)	**14.57**	3.5–9.5
Neutrophil (NEU#, $\times 10^9$/L)	**10.23**	1.8–6.3
Lymphocyte (LYMPH#, $\times 10^9$/L)	**3.36**	1.1–3.2
Monocyte (MON#, $\times 10^9$/L)	**0.95**	0.1–0.6
Eosinophil (EOS#, $\times 10^9$/L)	**0**	0.02–0.52
Hemoglobin (HB, g/L)	**96**	115–150
Platelet (PLT, $\times 10^9$/L)	300	125–350
C-Reactive protein (CRP, mg/L)	**42.35**	<10
Albumin (ALB, g/L)	**31.4**	35–55
Globulin (GLB, g/L)	**12.2**	20–35
Potassium (K, mmol/L)	**2.19**	3.5–5.3
Sodium (Na, mmol/L)	144	135–148
Chlorine (CL, mmol/L)	104.6	96–108
Calcium (Ca, mmol/L)	**1.32**	2.21–2.81
Magnesium (Mg, mmol/L)	**0.15**	0.66–1.07
Immunoglobulin G(IgG, g/L)	**1.49**	6.9–16.2
Immunoglobulin M (IgA, g/L)	**0.06**	0.3–3.7
Immunoglobulin A (IgM, g/L)	**0.02**	0.6–2.6
T lymphocytes (CD3, %)	**91.91**	50–84
T-helper lymphocytes (CD4, %)	32.35	27–51
T-suppressor lymphocytes (CD8, %)	**52.63**	15–44
B lymphocytes (CD3-/CD19+, %)	**0**	5–18
CD4/CD8 ratio	**0.61**	0.71–2.78
NK lymphocytes (CD16 + CD56, %)	7.25	7–40
Serum *Cytomegalovirus* deoxyribonucleic acid (CMV DNA)	Negative	Negative

Note: The bold values signified abnormal parameters.

3 | INVESTIGATIONS AND TREATMENT

The serum CMV DNA level was positive (8.33E+02 copies/mL) in our hospital, and the autoimmunity screening, including antinuclear antibodies, rheumatoid factors, anti-ENA autoantibody profiles, and antineutrophilic cytoplasmic antibodies, was negative. Serological tests revealed negative results for human immunodeficiency virus (HIV), hepatitis C, syphilis antibodies, and hepatitis B surface antigen. Stool analyses consistently demonstrated yellow, loose stools, occasionally accompanied by positive fecal occult blood tests, in the absence of white blood cells (WBCs), pus cells, parasites, or fungal elements. The bacteriococcal ratio was noted at 4–6:1, and stool cultures were negative. The assessment for *Clostridium difficile* antigen and toxins yielded negative results. Repetitive sputum smears and cultures revealed no positive findings. Radiological findings included bilateral pneumonia identified by chest CT and cholecystitis identified by abdominal CT. Additionally, pelvic and abdominal CT scans revealed multiple segments of small intestine and colon dilation with gas and fluid accumulation, exhibiting gas–fluid levels, with segmental colonic narrowing. According to the medical history, symptoms, laboratory and endoscopic examination results, GS combined with CMV gastroenteritis was definitely diagnosed. Subsequently, she received antiviral therapy with foscarnet sodium injection (3 g q8h ivgtt), intermittent administration of human immunoglobulin (5 g/d), cefoxitin (3 g q8h ivgtt) for pneumonia and cholecystitis, and continuous supplement of potassium, calcium, and magnesium. Afterwards, the patient felt better than before. Gastroscopy reexamination showed hyperemia and edema of gastric body mucosa, erythema of antrum mucosa, and a 0.8-cm mucous protrusive lesion near the pylorus in the anterior wall, with rough and red surface, and soft biopsy quality. Biopsy suggested mild chronic inflammation of mucosa with suspicious inclusion body deposition, and further immunohistochemistry indicated negative for CMV. Colonoscopy revealed a 0.3-cm polyp in the transverse colon near hepatic curvature with soft biopsy. Scattered mucosal patches of sigmoid colon were congested. The rectal mucosa was scattered in sheets with hyperemia and erosion (Figure 1). Pathological biopsy results showed severe chronic inflammation of mucosa with activity (+ ++), crypt abscess with erosion, decreased glands, CMV (+), CD68PGM-1(+).

4 | OUTCOME AND FOLLOW-UP

The patient was continuously treated with foscarnet for 3 weeks, human immunoglobulin replacement, and electrolyte supplement, and then the serum CMV DNA level decreased to below 50 copies/L. Finally, she almost completely relieved her symptoms and was discharged, and she then took oral ganciclovir for 3 weeks after her diarrhea completely resolved and the serum CMV DNA level became negative during the follow-up. She received intermittent human immunoglobulin replacement therapy for life.

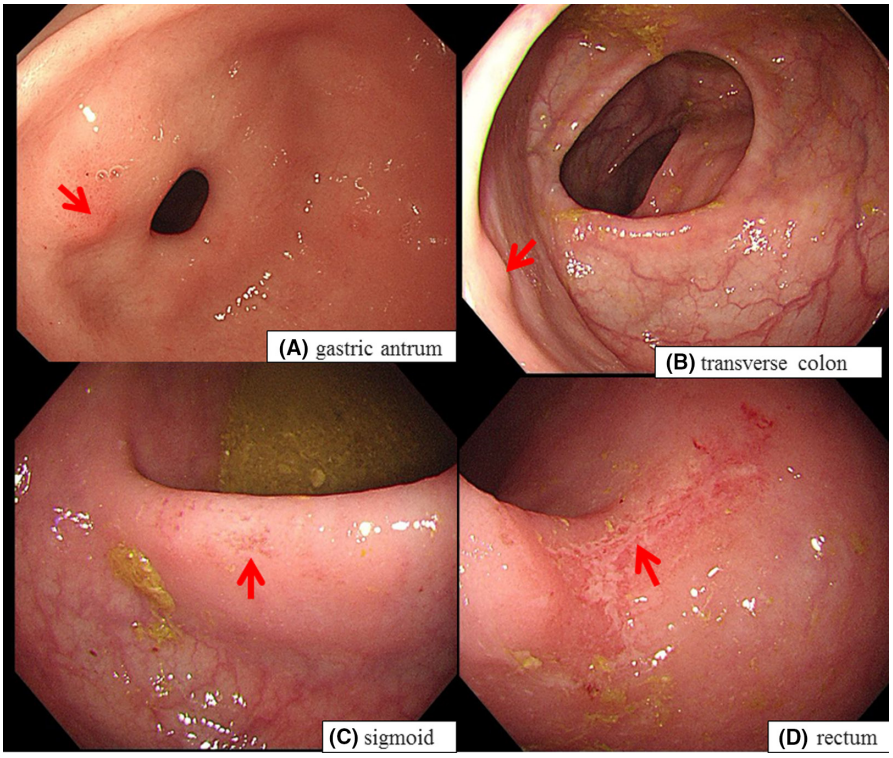

FIGURE 1 The endoscopic features of the patient. Arrows in the four pictures pointed to the following respectively: (A) mucous protrusive lesion near the pylorus; (B) polyp in the transverse colon; (C) congested and scattered mucosal patches of sigmoid colon; (D) scattered rectal mucosa with hyperemia and erosion.

5 | LITERATURE REVIEW

We searched all cases up to October 2022 using China National Knowledge Infrastructure database, Wangfang database and China Science and Technology Journal Database, and PubMed database. Keywords included Good's syndrome, Good syndrome, thymoma, diarrhea, *Cytomegalovirus* infection. According to the literature, 10 patients diagnosed with GS and CMV gastrointestinal infection meet our inclusion criteria (Table 2).

It is reported that eight patients had diarrhea without specific endoscopic manifestation. Although one case showed normal macroscopic appearance of the colon, the random colonic biopsy revealed CMV inclusion bodies.[7] Two cases were manifested as chronic diarrhea due to CMV enteritis and ulcerative colitis.[5,19] All patients received immunoglobulin replacement therapy. Except for two cases (one case of CMV infection confirmed by necropsy and one case did not describe the regime of antiviral agent), eight cases received ganciclovir as the initial treatment for CMV, and four cases switched to foscarnet due to no response or recurrence of diarrhea, and two cases finally switched to cidofovir due to no response or intolerance of foscarnet. Four patients deteriorated and died due to fulminant bronchopneumonia, sepsis, recurrent colitis and colonic perforation, and neurological deterioration with a refractory supraconvulsive state, respectively.

6 | DISCUSSION

The pathogenesis of GS still remains a mystery, which is defined as the significant reduction or absence of peripheral B cells and impaired T-cell-mediated immunity in adults.[1,2] GS patients are characterized by thymoma and hypogammaglobulinemia. Their initial symptoms tend to occur due to recurrent infection or secondary to thymoma itself, and hypogammaglobulinemia was not corrected following thymectomy.[21–23] Moreover, GS patients might also suffer from autoimmune complications, such as pure red cell aplasia, hypothyroidism, arthritis, myasthenia gravis, systemic lupus erythematosus, Sjögren's syndrome.[2,22] In this case, the female patient was presented with chronic diarrhea, hypogammaglobulinemia, absence of B lymphocytes, and thymoma, and the symptoms were not improved after the tumor removal. Therefore, GS was clinically diagnosed based on her history and blood tests. However, the diagnosis was not confirmed during her initial manifestation 9 years ago, indicating the lacked awareness of GS among clinicians.

Diarrhea was the patient's initial manifestation. It was reported that chronic diarrhea was present in almost up to 50% of GS patients, which was mainly caused by opportunistic infections, such as *Salmonella* spp., *Campylobacter jejuni*, *Clostridium difficile*, and CMV, but in some cases, the pathogen was not identified.[3–5,24] A study on GS in China found that 36% of patients suffered from diarrhea, and it was postulated that this might be related to malabsorption. Moreover, it also revealed that CMV was the most common pathogen among viral infections.[25] Kelesidis et al. systematically summarized the characteristics of 152 patients with GS, and discovered that diarrhea was present in 31.8% of patients, and 35.7% of them were caused by infection, while the pathogenesis of most cases had not been identified. Meanwhile, this study also found that CMV was the most common opportunistic pathogen reported, and the main cause of diarrhea in five cases.[24] Except for infections, the pathogenesis of diarrhea might also be related to immune factors, since some patients could relieve symptom through thymectomy and human immunoglobulin replacement treatment or steroids, as well as villous atrophy of intestines, which led to malabsorption.[5,26–28] It was supposed that intestinal malabsorption and inflammation seemed to cause diarrhea, hypoalbuminemia, and electrolyte imbalance, resulting in fatigue, weight loss, and numbness of hands and feet of this patient.

CMV was a common human viral infection, which could cause various system or organ infections in GS patients.[4,6–14] There might be a high prevalence of undiscovered CMV gastroenteritis in chronic diarrhea of unknown origin.[12] CMV could infect the whole alimentary tract, of which the most common is the colon, and gastric involvement is commonest in the upper gastrointestinal tract.[29,30] Yeh et al. retrospectively investigated 53 cases diagnosed with CMV gastritis in both immunocompromised and immunocompetent patients and concluded that gastric ulcer (88.9%) was the most common endoscopic feature and the gastric antrum was the most commonly affected location.[29] You et al. reviewed the endoscopic manifestations of gastrointestinal CMV diseases, including ulcers, fragile, hyperemic, or erythematous mucosa, edema, and subepithelial hemorrhage.[30] A recent study also reported that the commonest endoscopic feature of CMV disease was ulcers, followed by polypoid lesions and inflammation.[31] The endoscopic manifestations of patients diagnosed with GS and CMV infection with gastrointestinal tract involvement were also non-specific, mainly characterized by intestinal mocosal ulcers, inflammation, and edema of the intestinal mucosa, which might be easily confused with inflammatory bowel disease, and even some cases showed normal manifestations. Therefore, the clinical confirmation depended on biopsy results and further immunohistochemical method.[7,20] In addition, some patients suffered from both CMV gastroenteritis and ulcerative colitis, and they responded well to steroids and/

TABLE 2 Profiles of patients with GS and CMV infection gastrointestinal tract involved.

Case number	Publish year	Gender/age	Gastrointestinal presentation	Endoscopic features	Biopsy results	Treatment	Outcome (cause of death)
1	1985[15]	Male/69	Watery diarrhea	/	Widespread cytomegalovirus infection with duodenal and ileal ulceration, subtotal villous atrophy, marked nonspecific inflammation of the small intestine at necropsy	Infusions of gamma-globulin	Died (fulminant bronchopneumonia)
2	1999[16]	Male/59	Weakness, fever, and diarrhea	/	CMV enterocolitis	Antiviral therapy and replacement intravenous immunoglobulin (IVIG) therapy	Improved
3	2000[17]	Male/47	Severe anorexia, dysphagia, and vomiting	Severe pan-gastritis with a normal esophagus and duodenum	Granulation tissue, but no gastric epithelium, specific immunostaining for CMV was strongly positive.	Intravenous immunoglobulin (0.4 g/kg/month); Repeated courses of anti-CMV therapy in the form of ganciclovir, then forscarnet, combination therapy with both, and cidofovir	Died (sepsis)
4	2001[18]	Male/54	Diarrhea, fever, and weight loss	Multiple mucosal ulcers	Acute and chronic inflammation, with giant cells, intranuclear, and cytoplasmic inclusions; acute necrotizing inflammation and CMV inclusion bodies	Ganciclovir (2.4 mg/kg every 8 h for 14 days), following a second course of ganciclovir, combined with high-dose IVIG (500 mg/kg every other day); foscarnet (43 mg/kg every 8 h) was then given for 21 days, then foscarnet was continued at 75 mg/kg every 24 h	Died (recurrent colitis and colonic perforation)
5	2001[18]	Male/59	Epigastric pain and melena	Severe ulcerative gastritis	Acute ulcerative gastritis with extensive CMV-like intracellular inclusions bodies and strongly positive CMV-immunofluorescent staining	Ganciclovir (5 mg/kg every 12 h) for 14 days, then foscarnet (60 mg/kg every 8 h) was added to the ganciclovir for 24 days. Two doses of IVIG and 1 infusion of CMV-immune globulin were given. Ganciclovir was stopped after 12 weeks, after follow-up endoscopy showed improved gastritis with no inclusions and negative CMV immuno-stain.	Improved
6	2004[12]	Female/64	Watery diarrhea, abdominal distention	Gastritis, a duodenal polyp, and mucosal edema in duodenum	Intracellular inclusion bodies in the epithelial cytosol that were strongly positive to mouse anti-CMV antibody	Ganciclovir and CMV-immune globulin	Improved
7	2009[19]	Female/55	Dysphagia, watery diarrhea, a weight loss o, abdominal pain	Several ulcers in the descendens duodeni; edematous swelling of the mucosa with contact vulnerability, multiple ulcers in the rectum and sigmoid colon as part of ulcerative colitis	Duodenitis; positive immunohistochemical reactivity against CMV protein of the endothelium and fibroblasts	Steroid (1 mg/kg body weight) and immunosuppression with azathioprine (1.5 mg/kg body weight) and mesalazine and topical therapy in the form of clysters; a 3-week therapy with ganciclovir; then subsequent therapy with foscarnet had to be discontinued due to gastrointestinal side effects and was replaced with cidofovir and CMV-specific immunoglobulins; azathioprine switched to mycophenolate mofetil	Improved
8	2010[7]	Male/68	Watery diarrhea, reduced appetite	Normal	CMV inclusion bodies	Intravenous ganciclovir 5 mg/kg 12 hourly for 2 weeks, followed by lifelong oral maintenance with valganciclovir 900 mg daily for CMV retinitis; Monthly immunoglobulin infusion	Improved
9	2013[5]	Female/80	Watery, non-bloody diarrhea	Left-sided colitis suggestive of ulcerative colitis	Diffuse active chronic inflammation and atypical cells with inclusion bodies that stained positive for CMV	Prednisone; intravenous ganciclovir 5 mg/kg every 12 h for 2 weeks for disseminated CMV infection; intravenous immunoglobulin (IVIG) followed by monthly maintenance infusions	Improved
10	2022[20]	Undescribed /44	Fever, weight loss of 30 kg and chronic diarrhea	Ulcers in the sigmoid colon	Acute colitis with cryptic apoptosis without evidence of chronicity, compatible with CMV colitis	Ganciclovir and nitazoxanide; gamma-globulin	Died(neurological deterioration with a refractory supraconvulsive state)

Abbreviations: CMV, Cytomegalovirus; GS, Good's syndrome.

or immunosuppressive treatment, indicating that autoimmune factors might also play a significant role in the inflammation of gastrointestinal mucous membrane.[5,19] In the present case, the patient presented with mucosal inflammation and polypoid lesion under gastrointestinal endoscopy.

Ganciclovir was considered as the first-line therapy for CMV disease. The recommended dose for ganciclovir was 5 mg/kg intravenously twice daily (the dose should be adjusted according to the glomerular filtration rate, and the infusion time should last for over 1 hour).The course of treatment was at least 2–3 weeks, or even 3–6 weeks. However, in immunocompromised individuals, the potential adverse effects of ganciclovir were common, including headaches, elevated transaminases, fever, rash, and myelosuppression. On this basis, foscarnet, 90 mg/kg twice daily, could be considered as the initial therapy or in patients with ganciclovir nonresponse or intolerance. In addition, combined therapy might be effective after failure of monotherapy.[30] Pierce et al. showed that foscarnet was effective in the treatment of most ganciclovir-resistant CMV documented in the literature, and the incidence of nephrotoxicity in patients with solid organ transplantation was lower.[32] Wang suggested in his report that the treatment of disseminated CMV infection in GS could be similar to that of solid organ transplant recipients due to severe immunosuppression condition.[33] The patient, Tavakol reported, diagnosed with unilateral CMV retinitis with GS switched to oral valganciclovir maintenance after 6 months of intravenous ganciclovir treatment, and subsequent follow-up examination of the involved retina showed partial response.[3]

There was insufficient evidence or consensus on standard treatment strategies for CMV gastroenteritis with GS, and reference to other systems for the prevention and treatment of CMV infection might be reasonable. According to the literature review, ganciclovir could be used as the first line of initial treatment for GS and CMV gastroenteritis, and foscarnet or combined with both could be used for ganciclovir resistance or symptom recurrence, and cidofovir could be used for those who were not responsive or intolerant to foscarnet, which was currently the accepted treatment option. Although our patient was not tested for resistance, there was no significant clinical improvement after 2 weeks of ganciclovir and human immunoglobulin replacement therapy. Considering that ganciclovir might be resistant or the course of treatment might be inadequate, foscarnet sodium was adjusted for antiviral treatment with a dose according to the drug instruction. After 3 weeks of foscarnet treatment, serum CMV DNA level decreased and the symptom of diarrhea improved almost completely.

Despite active treatment, four patients still deteriorated and died, and the main cause of death was refractory severe infection and complications according to the literature review. Therefore, it is significant to prevent the infection in GS patients. Because the immunodeficiency persisted after thymectomy, patients with GS should receive regular lifelong human immunoglobulin replacement therapy (IGRT), every 3 months or once a month, so as to maintain IgG levels of above 500 mg/dL in case of low IgG, and furthermore, if a patient had recurrent infection regardless of IgG levels, IGRT could reduce the incidence of further severe infections.[4,23,33] Wang reported that the GS patient who was recurrently admitted to the hospital for pneumonia was administered with human immunoglobulin to maintain serum IgG levels of at least 500 mg/dL, while monitoring for signs of infection, and further infections had been prevented in the 1-year follow-up.[33] Most reports had come to similar conclusions. Therefore, it was necessary for our patient to undertake periodic human IGRT and monitor immunity status in order to prevent and timely identify opportunistic infection.

7 | CONCLUSION

It is of paramount importance for clinicians to consider the possibility of GS when adult-onset patients present with thymoma, hypogammaglobulinemia, and recurrent infection. Chronic diarrhea was present in almost up to 50% of GS patients. Additionally, CMV infection is a common opportunistic pathogen and the main cause of diarrhea. The endoscopic manifestations of patients diagnosed with GS and CMV gastroenteritis are also nonspecific, mainly characterized by ulcers, inflammation, and edema of intestinal mucosa. Therefore, clinical confirmation depends on biopsy results and further immunohistochemical methods. The recognized regime suggests that ganciclovir can be used as the initial therapy, and can be switched to foscarnet, or combined with both for ganciclovir resistance or symptom recurrence. Finally, it is necessary for GS patients to undertake periodic human IGRT and monitor their immune status, to prevent and timely identify opportunistic infection.

AUTHOR CONTRIBUTIONS

Xiaoran Li: Data curation; formal analysis; methodology; resources; writing – original draft. **Yanbin Liu:** Conceptualization; investigation; project administration; supervision; validation; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

Authors state no conflict of interest.

DATA AVAILABILITY STATEMENT

All data are included in this published article. The authors confirm that the data supporting the findings of this study are available within the article and its supplementary materials.

ETHICS STATEMENT

The need of ethics approval was waived because the data were obtained from previous clinical records and the subject provided informed consent to participate in this study.

CONSENT

Written informed consent for publication of the clinical details and/or clinical images was obtained from the patient. Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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