Good's Syndrome Complicated by Repetitive *Campylobacter Jejuni* Sepsis and Cellulitis

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Abstract

Campylobacter jejuni rarely causes bacteremia or extraintestinal infection. We report a case with Good's syndrome suffering from repetitive cellulitis and sepsis associated with *C. jejuni* that relapsed after a disease-free interval of almost one year. Serological examination demonstrated hypogammaglobulinemia, and analysis of lymphocyte subsets showed marked decreases in proportion of cells bearing both CD20+ and CD4+ cell markers. Pulsed field gel electrophoresis showed that the microorganisms in this patient were the same bacterial strain, and indicated a latent *C. jejuni* infection. The repetitiveness of cellulitis and sepsis was reflected in the clinical properties of this difficult-to-treat infection. (J Intern Med Taiwan 2014; 25: 295-301)

Key Words: *Campylobacter jejuni*, Cellulitis, Good's syndrome, Hypogammaglobulinemia, Sepsis

Introduction

Good's syndrome (GS) was first reported by Dr. Robert Good in 1954[1], and denotes the coexistence of B and T cell immunodeficiency in thymoma patients[2]. It is characterized by hypogammaglobulinemia, low or absent B cells, variable defects in cell-mediated immunity with CD4+ T lymphopenia, an inverted CD4/CD8+ T-cell ratio, and reduced T-cell mitogen proliferative responses[2,3]. The storm of immunity cannot be corrected by thymectomy[3].

Owing to humoral and cell-mediated immune deficiencies, patients with GS have increased risk of recurrent infections, including encapsulated bacteria, viruses, fungi, protozoa, and opportunistic infections[3,4]. According to one 2010 systematic review article, the most commonly reported infectious complication of GS was recurrent upper and lower respiratory tract bacterial infections (76%; 116/152). Only two cases of bacterial skin infections were reported (1%; 2/152)[4]. Here we represent a case complicated by repetitively concurrent *C. jejuni* sepsis and cellulitis around one year before GS was diagnosed. To our knowledge, this is a rare case of recurrent skin infection associated with GS in the medical literature.

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Case report

A 62-year-old male patient with GS was admitted to our hospital for sepsis and right lower leg cellulitis in Nov 2007. His past medical history included invasive thymoma since 2001 post excision surgery and radiotherapy, pure red cell aplasia (PRCA), Bechcet's disease with oral ulcer and genital ulcer, myasthenia gravis (MG), hypogammaglobulinemia (IgG; IgA; IgM), progressive leucopenia, thrombocytopenia, hepatosplenomegaly, bronchiectasis, multiple segments and transfusional hemosiderosis (Figure 1). His infection history from 2002 (Figure 1) included herpes simplex virus infection, *Pneumocystis jiroveci* pneumonia (PJP), upper respiratory tract infections (URI) and cytomegalovirus (CMV) retinitis.

In October 2007, the patient presented to our emergency room with body temperature, 38.5 degrees Celsius; heart rate, 96 beats/min; respiratory rate, 20 breaths/min; blood pressure, 83/43 mmHg. Chief complaints were general weakness, fever and chills intermittently for 2 months and afterwards, painful swelling of the right leg with local heat and erythema for 2 weeks. He took amoxicillin/clavulanic acid initially, but the symptoms were not relieved. At the emergency room, laboratory workup revealed a white blood cell count: 1.7×10^3 cells/μL (normal range (NR): $3.9\text{-}10.6\times10^3$ cells/μL) with 50% lymphocyte cells, CD3+ cells: 94%, CD4/CD8 ratio: 0.8 (NR: 44%/55%), CD20+ cells: 5%, C3: 156.0 mg/dL (NR: 90-180 mg/dL), C4: 63.9 mg/dL (NR: 10-40 mg/dL), negative for antinuclear antibodies (ANA), hemoglobin: 7.4 g/dL (NR: 13.5-17.5 g/dL), platelet count: 114×10^3 cells/μL (NR: $150\text{-}400\times10^3$ cells/μL), albumin: 2.6 g/dL (NR: 3.5-5.0 g/dL), and C-reactive protein: 43.2 mg/L (NR: < 5.0 mg/L). Gammaglobulin levels measured were: IgG: 48.50 mg/dL (NR: 700-1600 mg/dL), IgA: 22.40 mg/dl (NR: 70-400 mg/dL) and IgM < 3.96 mg/dl (NR: 40-230 mg/dL). Serology for HIV was negative. Blood cultures grew *Campylobacter jejuni*, which were susceptible to ceftriaxone but resistant to erythromycin. Under the impression of sepsis and cellulitis, he was admitted and meropenem (1 g q8h) was administered. All symptoms subsided rapidly and the result of blood culture turned to negative on the 11th day of meropenem therapy. Meropenem

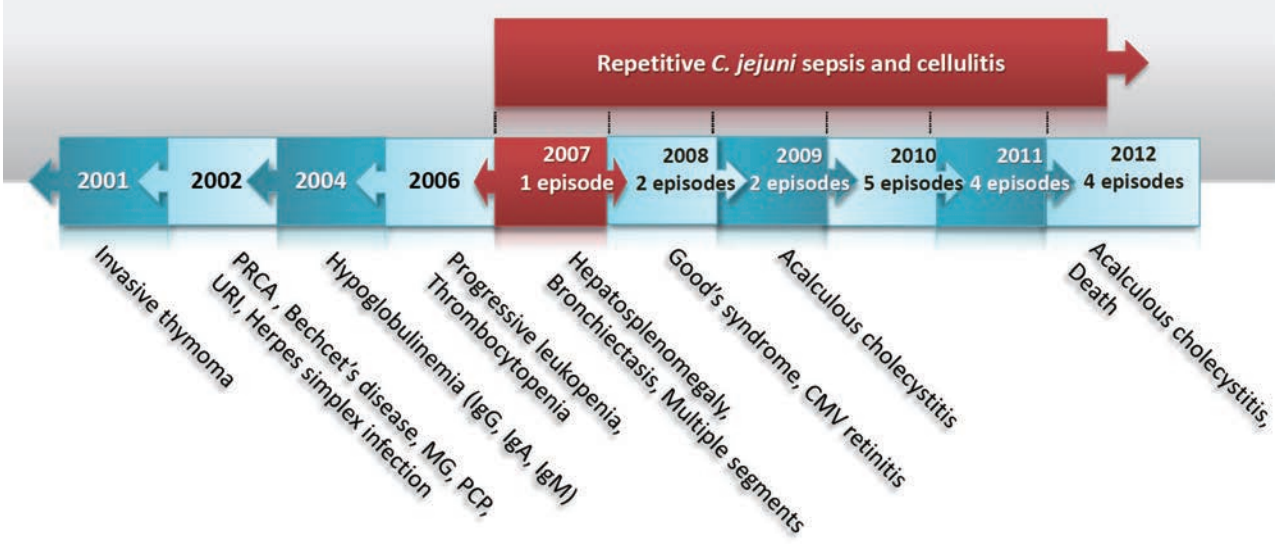

Figure 1. Personal medical history of the presented case. Abbreviations: CMV, cytomegalovirus; MG, myasthenia gravis; PJP, *Pneumocystis jiroveci* pneumonia; PRCA, pure red cell aplasia; URI, upper respiratory tract infection.

was given for a total 13 days and was then switched to oral form ciprofloxacin (500 mg bid for 10 days) on the day he was discharged.

In the next five years, the first episode was followed with 17 separate episodes of community-acquired *C. jejuni* bacteremia, which totally complicated 12 episodes of sepsis and cellulitis and 2 episodes of acalculous cholecystitis. We defined an episode of *Campylobacter* bacteremia as a new positive blood culture detected at least 1 month after the previous episode, considered a recurrent bacteremia. *Campylobacter* bacteremia was defined as community-acquired if the first positive blood culture specimen was draw within the first 48 hours after admission. Cellulitis occurred over one or both legs, right forearm, right hand, or right scapula region. During these episodes, susceptibility tests of blood cultures showed the microorganism was resistant to erythromycin and most times sensitive to but only one time resistant to ceftriaxone. We utilized pulsed field gel electrophoresis (PFGE)[5] to identify genus and species of *C. jejuni* in our patient, which showed that the pathogen isolated in the fifth episode was genetically the same with that in the sixth episode. For detection of infection source, abdominal computerized tomography showed no abscess, enteritis or abdominal aortic aneurysm; transthoracic and transesophageal cardiac echocardiogram revealed no definite intracardiac vegetation, and Gallium scan detected no abnormal lesions either. Skin biopsy and colonoscopy were also suggested, but the patient refused. Besides, the patient did not experience any prolonged diarrhea; several stools specifically cultured were all negative for *C. jejuni*.

Although the patient's cutaneous manifestations responded well to both meropenem and ceftriaxone, meropenem was chosen for therapy during hospitalization as morbilliform skin rashes appeared after ceftriaxone was administered in the second episode. Furthermore, intravenous immunoglobulin (IVIG) 400 mg/kg was also added as a part of therapy since the second episode. After discharge, either ciprofloxacin or ceftibuten was given as suppressive therapy. However, all the therapy courses failed to eradicate this microorganism in that clinical and bacteriologic relapse followed. The possibility that asymptomatic bacteremia existed in the patient arose as during the third episode, one set of blood culture for *C. jejuni* was performed before the patient self-discharged against advice. The culture result unexpectedly showed *C. jejuni* growth 6 days later when the patient went to the outpatient clinic without any clinical symptoms. Due to no fever and other infection signs except for the blood culture result, no antibiotic but only IVIG was given then.

The last time when the patient presented to the emergency department was with head injury due to falling down. During hospitalization, traumatic diffuse subarachnoid hemorrhage and right fronto-temporal lobe parenchymal hemorrhage with rupture into ventricle and leftward midline shift was noted; septic shock with multiple organ failure developed. After one month, according to his wife's request, he was discharged due to shock with cardiopulmonary failure and shortly expired thereafter.

Discussion

The pathogenesis of the immunodeficiency in GS remains elusive for 56 years after Dr. Good's first observation on thymoma and hypogammaglobulinemia patients. A bone marrow defect is suggested by B and T cell lymphopenia[3]. It has a worldwide distribution and 22.8% of cases were reported in Asia[4]. Male and female patients are equally affected and the mean age at presentation is 59.1 years[4]. Approximately, 40%, 6-11%, and 5% of thymoma patients have related parathymic syndromes of MG, hypogammaglobulinemia, and PRCA respectively[6]. Nevertheless, patients with concurrent thymoma, PRCA, and hypogammaglobulinemia are very rare. In the present article, we discuss a 60-year-old male with thymoma, who had developed MG, PRCA and

hypogammaglobulinemia after thymectomy and radiotherapy, and unfortunately, the immunologic abnormalities didn't response to cyclosporin, azathioprine, corticosteroids therapy, or thymectomy.

Previous reports have estimated that autoantibodies that play a role in the pathogenesis of hypogammaglobulinemia can present in up to 30% of causes of GS[4]. The most common autoimmune antibodies, antinuclear antibodies (ANA), present in more than half of the cases[4]. However, they were not detectable in our case, and complements 3 and 4 were detected as normal. His IgA, IgG and IgM were low even after extended thymectomy. The presence of both CD20+ B and CD4+ T lymphocytes was very few with the proportions of only 5% and 44% of total leukocytes respectively. Besides, an inverted CD4+/CD8+ T-cell ratio of 0.8 was also noted. Immunological defects in thymoma-associated immunodeficiency affect humoral (CD20+ B-cell lymphopenia, hypogammaglobulinemia) as well as cellular immunity (CD4+ T-cell lymphopenia, inversion of CD4+/CD8+ T-cell ratio). Combined defects of humoral and cellar immunity account for frequent occurrence of opportunistic infections[7].

C. jejuni has become the predominant pathogen of food-borne gastroenteritis both in developing and industrialized countries[8,9]. It has been reported that when causing gastroenteritis, the mean duration of excretion of *C. jejuni* in the feces is two to three weeks in most patients compared to two years in patients with common variable immunodeficiency[10]. *Campylobacter* bacteremia is a rare disease, occurring mainly in patients with immune deficiency (51.6%), including HIV infection (23.4%) and hypogammaglobulinemia (15.6%)[11]. Of the bacteremic episodes in GS patients, 38.5% were caused by *Campylobacter* species[12]. *C. jejuni* was recovered in 66% of *Campylobacter* bloodstream infection cases[11].

Although *C. jejuni* can stimulate phagocytic activity of polymorphonuclear neutrophil granulocytes, the role of the nonspecific immune responses as a defense mechanism is secondary, and the humoral responses have the primary role against infection[13]. Human monocytes ingest *C. jejuni*, and antibody-mediated immunity plays a major role in patients' recovery from infection[10]. The intracellular survival of *C. jejuni* is around 6-7 days, whereas in the absence of phagocytes, the survival of the microorganism is only 3-4 days[13]. Therefore, monocytes may be a reservoir of *C. jejuni* in immunocompromised hosts. It may also play an important role in the translocation of *C. jejuni* to the blood stream and persistence of infection in patients like AIDS or CD4+ T lymphopenia[13]. This evidence reflected the 40-fold higher incidence of *Campylobacter* diseases in AIDS patients than the general population[14], although HIV infection is generally associated with hypergammaglobulinemia[3]. Patients with GS have severe defects in cell-mediated immunity due to abnormalities in T lymphocyte proliferation and/or IL-2 production[3], and based on the above opinions, *C. jejuni* might an important cause of bloodstream infection in such patients.

Cutaneous involvement is a less well-recognized complication of *Campylobacter* bacteremia (5.9%), and vice versa (4.8%)[11]. An article reported two cases of cellulitis associated with *Campylobacter* bacteremia, which pointed out that possible localized sites of infection, such as the skin, could cause persistent or intermittent bacteremia[15]. Our patient experienced up to 18 episodes of community-acquired sepsis/bacteremia, cellulitis or both, which occurred months or almost one year following the first one. The microorganisms, although all specimens were cultured from blood samples, was isolated simultaneously when the patient had cellulitis, and was therefore likely the cause of the cellulitis. Due to no penetrating injury of skin, the negative results of stool culture, computerized tomography for enteritis, cardiac echo for vegetation, and Gallium scan for inflammation survey, and latent infection revealed from PFGE, intracellular

survival of *C. jejuni* which led to repetitive septic metastasis with cellulitis was considered a possible pathogenesis of this microorganism in our patient.

A retrospective study included twenty patients of *Campylobacter*-associated cellulitis. Nine were infected with *C. jejuni* and eleven with *C. fetus*; nineteen were immunocompromised and one was immunocompetent. The most common site of cellulitis was the lower extremities. More than one-half of the cases had negative stool culture[9]. In our case, sites of recurrent cellulitis were legs, right forearm, scapular region and right hand. Similar to the literature documented, a protracted course of infection with recurrence and negative stool culture also occurred in our patient.

Six GS patients complicated with *Campylobacter* bacteremia including ours were identified from the literature and are summarized in Table 1. Their mean age was 60.2 years. Two were infected with *C. jejuni*, two were *C. fetus* and the other two were unidentified *Campylobacter* species. Sepsis, which is mainly caused by B cell immunity defect[13], is the most common clinical feature, whereas diarrhea, which reflects the T cell specific immunity defect[16], was observed in only one patient. Our patient suffered from sepsis and cellulitis but no diarrhea either. It is implied that neither diarrhea nor positive stool culture is an absolute clinical manifestation or a necessity in *Campylobacter*-induced sepsis and cellulitis.

The most appropriate protocol of antimicrobial therapy for *Campylobacter* extraintestinal infection in immunocompromised patients has not been established, and the widespread emergence of multidrug resistance among *C. jejuni* is of great concern. Even though macrolides and quinolones are considered the choice of antibiotic for the empirical treatment of *C. jejuni* infection[17], resistance has been reported increasingly[11,18,19]. It is speculated that the widespread use of erythromycin and ciprofloxacin in veterinary medicine has accelerated this resistance trend[11,18,19]. In Taiwan, the prevalence of erythromycin-resistant strains was estimated at 18% and 60% among isolates from pediatric patients and chicken isolates, respectively[20]. The large uses of quinolones in poultry have led to the emergence of even higher resistant rates. For example, the use of ciprofloxacin is not recommended for pediatric patients in Taiwan. Nevertheless, as high as 57% and 91%, respectively, of the strains derived from these patients and chicken were resistant to ciprofloxacin[20]. The *C. jejuni* isolated from our patient also displayed resistance to erythromycin and failure of ciprofloxacin suppressive therapy. Third-generation

Table 1. Characteristics of Good's syndrome patients with *Campylobacter bacteremia*. Abbreviations: CET, cefotiam; CIP, ciprofloxacin; CRO, ceftriaxone; ERY, erythromycin; IVIG, intravenous immune globulin; MEM, meropenem; NA, not available; PAN, panipenem; PR, present report

Case No.	Year	Age/ Sex	*Campyloacter* species	Clinical feature(s)	Site of isolation	Antibiotic therapy	IVIG therapy	No. of relapses	Outcome	Country	Reference
1	1994	63/M	*C. fetus*	Sepsis	Blood	CET	No	0	Resolved	Japan	[27]
2	2001	54/M	*C. jejuni*	Diarrhea	Blood	Antibiotics	No	3	Resolved	USA	[3]
3	2002	63/M	*C. fetus*	Sepsis	Blood	Antibiotics	Yes	>1	Resolved	Japan	[28]
4	2004	64/F	*C. spp*	NA	Blood	MEM/PAN/ ERY+CIP	Yes	0	Resolved	Japan	[22]
5	2010	57/M	*C. spp*	Sepsis	Blood	NA	NA	NA	Resolved	France	[29]
6	2012	62/M	*C. jejuni*	Sepsis/ Cellulitis/ Acalculous cholecystitis	Blood	CRO/MEM	Yes	18	Relapse	Taiwan	PR

cephalosporins were also one of the most commonly used empirical antibiotics, and our patient's cellulitis had indeed been much improved after ceftriaxone use initially. However, it is reported that mortality of *Campylobacter* bacteremia was higher for patients treated with them, and the resistance rate of *C. jejuni* is also very high[11,21]. In 2009, 20% (12/110) of resistance rate was observed in our hospital. In the eighth episode, the *C. jejuni* isolated from our patient also showed resistance to ceftriaxone.

Combination therapy with meropenem and IVIG, which has been a successful treatment in a GS patient with *Campylobacter* bacteremia and sepsis[22], was selected in our case. Several articles reported that *Campylobacter* species isolated from blood cultures remain highly susceptible to imipenem[11,12,21,23] and meropenem[12], including macrolide- and multidrug-resistant strains, and suggest that carbapenems may be the drugs of choice for empirical therapy of severe invasive campylobacteriosis before until a definitive antibiogram is available[11,12,21,23]. The clinical experience with meropenem[9], imipenem[24], and panipenem[25] use for *Campylobacter* bacteremia have been evaluated in immunocompromised patients[9,24,25], including hypogammaglobulinemia[24] and agammaglobulinemia[25], and duration of therapy may be extended to at least 2-6 weeks. Regularly scheduled treatment with IVIG leads to fewer hospitalizations and reduces severe infections and excess use of antibiotics[3,26]. Approximately 38% of patients with GS had reduced incidence of infections after treatment with IVIG[4]. Our patient had once recovered from several episodes of *Campylobacter* bacteremia without other complications only with the treatment of immunoglobulin replacement.

In summary, *Campylobacter* sepsis and cellulitis remain an uncommon but severe disease linked to GS, and should be considered in such populations with periodic fever and cellulitis. A protracted course and recurrences are the characteristics and prolonged combination therapy with carbapenem, and IVIG may be required.

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古德綜合症併發反覆的空腸曲狀桿菌敗血症和蜂窩性組織炎

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摘 要

空腸曲狀桿菌很少引起菌血症或腸道外的感染。我們報導一位患有古德綜合症的病患，其遭受了空腸曲狀桿菌蜂窩性組織炎及敗血症的反覆感染，其中間隔有將近一年之久未發病。血清學檢查顯示低丙球蛋白血症，淋巴細胞亞群的分析並且發現，帶有 CD20+ 及 CD4 + 細胞標誌物的比例皆顯著的降低。脈衝式膠體電泳顯示存在於此患者中的微生物屬於同源菌株，其表示了空腸彎曲桿菌的潛伏感染。蜂窩組織炎和敗血症的反覆發作反映了這種難以治療的感染之臨床特性。