Case Report

Diffuse Large B Cell Lymphoma Mimicking Granulomatosis with Polyangiitis

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In a patient with systemic multiorgan disease with overlapping features, the differential diagnosis included infectious diseases, malignancies, and systemic autoimmune or inflammatory diseases. We present an unusual case of a young male with B cell lymphoma who presented with symptoms mimicking systemic vasculitis and review the existing literature.

1. Introduction

Granulomatosis with polyangiitis (GPA) is a small and medium-sized vessels vasculitis which is highly associated with positive anti-neutrophil cytoplasmic antibodies (ANCA), mainly with antibodies against proteinase 3 (PR3) [1]. Among reported clinical manifestations of GPA, necrotizing granulomatous inflammation in the upper respiratory tract, pulmonary nodules (often with cavitations), and necrotizing glomerulonephritis are prominent; their combination is a classic GPA triad [1]. Involvement of other organs and systems, such as the skin, eyes, ears, and peripheral nervous system, is less specific but definitely is not rare. The involvement of gastrointestinal tract (GIT) in GPA is infrequent [2].

2. Case Presentation

A 22-year-old male was admitted because of fever (39.5°C), weakness, excessive sweating, vomiting, and weight loss during the previous month. His medical history was unremarkable, and physical examination did not show any pathological signs. Laboratory data revealed white blood count 2100 (WBC, 4000–10800/μL), hemoglobin 12.5 (Hb, 13–17 g/dL), and platelet count 113000 (PLT, 130000–350000/μL); aspartate aminotransferase 65 (AST, 5–40 U/L), alanine aminotransferase 63 (ALT, 30–65 U/L), alkaline phosphatase 123 (AlkPh, 30–115 U/L), lactate dehydrogenase 997 (LDH, 60–225 U/L), creatinine 0.73 (0.4–1.3 mg/dL), blood urea nitrogen 8 (BUN, 5–20 mg/dL), creatinine kinase 20 (CK, 35–300 U/L), albumin 3.9 (3.4–5 g/dL), and ferritin 469 (15–300 ng/mL); erythrocyte sedimentation rate 24 mm/1 hour (ESR, <20 mm/1 hour) and C-reactive protein 18 mg/L (CRP, 0–5 mg/L). Coagulation profile and thyroid function tests were normal. Urine dip-stick was negative for protein or blood. Blood, urine, throat, and stool cultures were all sterile. Serology tests, including human immunodeficiency virus, hepatitis B and C virus, Cytomegalovirus, Epstein Barr virus (EBV), Rickettsia, Chlamydia, Toxoplasma, Brucella, and Q-fever, were negative. Anti-nuclear (ANA) and anti-dsDNA antibodies were positive; anti-cardiolipin, and B2-glycoprotein antibodies and cryoglobulins were negative; complements C3 and C4 levels were normal; protein electrophoresis and immunoelectrophoresis were within normal limits. Test for anti-neutrophil cytoplasmic antibodies (ANCA) was positive for myeloperoxidase (MPO) and negative for proteinase 3 (PR3). Computed tomography (CT)
Figure 1: Coronal reconstruction of thoracic CT scan in lung window irregular pulmonary nodule with surrounding ground glass opacity in the left lower lobe of the lung.

demonstrated several bilateral peripheral nodular infiltrates in the lungs (Figure 1) and several nodular lesions in the liver and kidney; the spleen was mildly enlarged. Bone marrow biopsy was negative for granulomas and mycobacterium tuberculosis as well as for malignancy. The patient was discharged with a recommendation for ambulatory treatment with doxycycline for suspected atypical infection.

One month later, due to the ongoing fever, abdominal pain, repeated vomiting, constipation, weight loss, and loss of smelling ability, he was admitted again. Repeated CT scan showed resolution of previous pulmonary nodules with the concomitant appearance of several new ones. The number of hepatic nodules had increased with further enlargement of the spleen and the appearance of mild mesenteric lymphadenopathy. CT-guided fine needle aspiration of the pulmonary lesion failed and an open lung biopsy was performed. At that time, the patient was transferred to our hospital.

On admission the patient looked ill, pale, and weak. Heart and lung evaluation was unremarkable. There was mild tenderness of the upper abdomen without signs of peritoneal irritation or liver enlargement; the spleen was mildly enlarged and palpable. Repeated blood tests showed WBC 4200/µL, HB 12.4 g/dL, PLT 248000/µL, creatinine 0.65 mg/dL, albumin 3.9 g/dL, AST 25 U/L, ALT 43 U/L, GGT 52 U/L, ALKP 123 U/L, LDH 234 U/L, CRP 16.43 mg/L, and ESR 24 mm/1 hour. Repeated ANA and anti-dsDNA were negative; the test for ANCA was pending. Gastroscopy showed 24 mm/1 hour. Repeated ANA and anti-dsDNA were negative. The patient was treated with the CHOP-R protocol (cyclophosphamide, doxorubicin, vinblastine, prednisone, and rituximab) with achievement of rapid clinical and hematological remission.

3. Discussion

We describe a unique case of a young male who developed high-grade EBV-associated diffuse large B cell lymphoma mimicking granulomatosis with polyangiitis (GPA). Our patient presented with constitutional symptoms (prolonged fever, severe fatigue, and weight loss), nasal involvement (anosmia), multiple pulmonary, hepatic, and spleen nodules, elevated inflammatory markers, anemia, transiently positive MPO, and findings on open lung biopsy suggestive of necrotizing vasculitis. All together, these findings were “strong” clues for the diagnosis of ANCA-associated vasculitis.

GPA is a small and medium-sized vessels vasculitis which is highly associated with positive ANCA, mainly with antibodies against PR3 [1]. Among reported clinical manifestations of GPA, necrotizing granulomatous inflammation in the upper respiratory tract, pulmonary nodules (often with cavitations), and necrotizing glomerulonephritis are prominent; their combination is a classic GPA triad [1]. Involvement of other organs and systems, such as the skin, eyes, ears, and peripheral nervous system, is less specific but definitely is not rare. The involvement of gastrointestinal tract (GIT) in GPA is infrequent [2]. Akbulut reported a patient with severe GPA who developed bowel perforation and sepsis and eventually died. The author reviewed thirteen similar cases and summarized the main clinical features of GIT involvement in GPA as follows: gingivitis, peptic ulcers, bloody diarrhea, bowel ulcerations, persistent perianal ulcers, pancreatitis, cholecystitis, small bowel perforations, and colitis [2]. Involvement of the GIT in GPA has been usually reported during relapses, in uncontrolled diseases, and late in the disease course (2-3 years later or more). The most common GIT complications in vasculitis in general and in GPA particularly are oral ulcers, intestinal ulcerations, bleeding,
and perforations. In this regard, treatments such as corticosteroids and non-steroidal inflammatory drugs have been suggested as triggering agents. The presence of active vasculitis-related changes at the sites of bowel perforations in about 61% of reported cases supports the idea that uncontrolled vasculitis in the intestinal wall was the main reason for perforation and not corticosteroids as was presumed previously [2]. Involvement of the GIT as a presenting symptom of GPA is extremely rare. We could find in the English language literature only 20 cases with GIT involvement as an early (less than one-year disease duration) or even initial GPA presentation (Table 1). Patients were mainly males (15/20) with the mean age of 41.7 years (our patient was much younger) [3–22]. According to case descriptions, 4 (20%) patients had appetite and weight loss, 9 (45%) patients had oral and/or tongue ulcers, 6 (30%) had diarrhea, 9 (45%) had blood in stool, melena, or bloody diarrhea, 2 (10%) had anal ulcers, and 1 had odynophagia (2%); 9 (45%) patients developed “acute abdomen” due to perforation, intra-abdominal bleeding, or obstruction. Our patient had abdominal symptoms for more than two months but never had oral ulcers, diarrhea, or GIT hemorrhage. Only in one reported patient was the GIT involvement an isolated GPA feature but was accompanied by fever [3] while, in the majority of cases, there were signs of multiorgan disease with almost an obligatory involvement of the upper airways (our patients had anosmia but not involvement of sinuses, eyes, or ears) and lungs (our patient had nodules without cavitations) and less often the kidneys (our patient did not have signs of nephritis).

Walton analyzed clinical and pathology features in 56 cases of GPA (Wegener’s granulomatosis at that time). He found granulomatous lesions in the spleen (55.6%), kidney (66.7%), and liver (16.7%) and fibrinoid necrosis in blood vessels in the spleen (77.8%), kidney (77.8%), liver (18.5%), intestine (24.1%), pancreas, and gallbladder (7.4% each) [23]. One patient had severe refractory anosmia which later was complicated by nasal septum destruction and deafness (our patient also had anosmia but not structural or mucosal damage in the oral or nasal cavity). Described liver and spleen nodules were mainly microscopic findings but not macroscopic lesions on imaging films. Although the classic splenic manifestations of GPA are splenic infarcts, hemorrhage and spleen necrosis may also occur [24–27]. In one GPA case, multiple small bowel ulcers and perforations were accompanied by spleen infarctions [9]. Walton and Leggat described a patient with anosmia, lung involvement, multiorgan failure, and spleen granulomas [15].

Involvement of the liver is extremely rare in the course of GPA. Among liver manifestations, nonspecific nontraumatic hepatic hematoma and incomplete septal cirrhosis were
Table 1: Analysis of cases with gastrointestinal symptoms in the early course of granulomatosis and polyangiitis (one year or less).

<table>
<thead>
<tr>
<th>Author, year of publication</th>
<th>Gender, year of publication</th>
<th>Presenting symptoms</th>
<th>Disease duration</th>
<th>APR elevation</th>
<th>Concomitant respiratory system/kidney/other involvement</th>
<th>CT/endoscopic findings</th>
<th>Biopsy findings</th>
<th>ANCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yoshikawa et al., 2014 [3]</td>
<td>Male, 30</td>
<td>Oral ulcer, bloody stool</td>
<td>1 month</td>
<td>ESR, CRP</td>
<td>Fever</td>
<td>CT: thickening of the transverse colon wall</td>
<td>Superficial inflammatory cell infiltration and edema noted in lamina propria mucosa</td>
<td>PR3</td>
</tr>
<tr>
<td>Sinnott et al., 2013 [4]</td>
<td>Male, 29</td>
<td>Bloody diarrhea</td>
<td>2 months</td>
<td>ESR, CRP</td>
<td>Lung/kidney</td>
<td>CT: inflammatory changes in caecum and ascending colon</td>
<td>No bowel biopsy</td>
<td>PR3</td>
</tr>
<tr>
<td>Dag et al., 2013 [5]</td>
<td>Male, 29</td>
<td>Bloody stool, rectal bleeding</td>
<td>6 months</td>
<td>CRP, ESR</td>
<td>Lung, kidney, joints, skin</td>
<td>CT: distal ileum perforations</td>
<td>Active chronic inflammation and ulcer bases</td>
<td>PR3</td>
</tr>
<tr>
<td>Qian et al., 2010 [6]</td>
<td>Female, 79</td>
<td>Bloody diarrhea</td>
<td>Presenting symptom</td>
<td>Not reported</td>
<td>Pulmonary hemorrhage, acute renal failure</td>
<td>Colonoscopy: pancolitis</td>
<td>Mucosal ulcerations with foci of acute inflammation and hemorrhage</td>
<td>PR3</td>
</tr>
<tr>
<td>Samim et al., 2010 [7]</td>
<td>Male, 35</td>
<td>GI bleeding</td>
<td>1-2 weeks</td>
<td>CRP</td>
<td>Sinusitis/kidney/arthritis/skin rash</td>
<td>CTA: bleeding from one mesenteric artery</td>
<td>Central ulcerative inflammation and occluded small arteries in intestinal wall</td>
<td>PR3</td>
</tr>
<tr>
<td>Marie et al., 2010 [8]</td>
<td>Male, 31</td>
<td>Epigastric pain, rectal bleeding</td>
<td>Before diagnosis of GPA</td>
<td>ESR, CRP</td>
<td>Arthritis, purpura</td>
<td>Colonoscopy: multiple rectal and sigmoidal ulcerations with spontaneous bleeding</td>
<td>Inflammatory cell infiltrates surrounding small vessels</td>
<td>PR3</td>
</tr>
<tr>
<td>Deniz et al., 2007 [9]</td>
<td>Male, 44</td>
<td>Acute abdomen</td>
<td>4 weeks</td>
<td>Not reported</td>
<td>Sinusitis, lung cavitation nodules</td>
<td>CT: intra-abdominal free air and fluid, small bowel wall thickening, chronic splenic infarction</td>
<td>Ulcerations, necrotizing transmural granulomatous inflammation, fibrinoid necrosis of small to medium-sized submucosa vessels</td>
<td>ANCA</td>
</tr>
<tr>
<td>Kuwahara et al., 2006 [10]</td>
<td>Male, 30</td>
<td>Bloody diarrhea, oral and anal ulcers</td>
<td>Before diagnosis of GPA</td>
<td>CRP</td>
<td>Nasal bleeding, skin and mucosal ulcers, conjunctivitis</td>
<td>Colonoscopy: multiple ulcerations with irregular shapes, macroscopic bleeding, pseudopolyposis in terminal ileum, colon, rectum</td>
<td>Inflammation and epithelioid granuloma with multinuclear giant cells</td>
<td>PR3</td>
</tr>
</tbody>
</table>
### Table 1: Continued.

<table>
<thead>
<tr>
<th>Author, year of publication</th>
<th>Gender, age (years)</th>
<th>Presenting symptoms</th>
<th>Disease duration</th>
<th>APR elevation</th>
<th>Concomitant respiratory system/kidney/other involvement</th>
<th>CT/endoscopic findings</th>
<th>Biopsy findings</th>
<th>ANCA</th>
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</thead>
<tbody>
<tr>
<td>Chow et al., 2003 [12]</td>
<td>Male, 46</td>
<td>Melena</td>
<td>4 weeks</td>
<td>Not reported</td>
<td>Sinusitis, glomerulonephritis</td>
<td>Endoscopy: patchy ulceration of small bowel mucosa</td>
<td>Resected jejunum: small vessel vasculitis of bowel, mucosal ulceration, submucosal inflammation, fibrosis</td>
<td>c-ANCA</td>
</tr>
<tr>
<td>Fallows et al., 2000 [13]</td>
<td>Female, 34</td>
<td>Odynophagia</td>
<td>4 weeks</td>
<td>ESR</td>
<td>Otitis, conjunctivitis, skin rash and ischemia, arthritis, mild kidney involvement</td>
<td>Esophagospopy: multiple punch-out ulcers</td>
<td>Fibrinoid necrosis with ulcerations and inflammatory cells infiltration surrounding small blood vessels</td>
<td>c-ANCA</td>
</tr>
<tr>
<td>Shaikh et al., 2006 [14]</td>
<td>Male, 44</td>
<td>Abdominal pain, peritonitis</td>
<td>Several weeks</td>
<td>ESR</td>
<td>Polyarthralgia, skin rash, polyneuropathy</td>
<td>X-ray and abdominal CT: pneumoperitoneum</td>
<td>Resected ileum: vasculitis with fibrinoid necrosis</td>
<td>c-ANCA</td>
</tr>
<tr>
<td>Walton and Leggat, 1956 [15]</td>
<td>Female, 42</td>
<td>Oral and tongue ulcers</td>
<td>10 months</td>
<td>Not reported</td>
<td>Fever, anosmia and nasal ulcers, deafness, lung infiltrates and pleurisy, arthritis, rash, neuritis, kidney involvement</td>
<td>ND</td>
<td>Autopsy (data restricted to GIT): multiple spleen solitary granulomata similar to those in lung, signs of vasculitis in pancreas</td>
<td>ND</td>
</tr>
<tr>
<td>Tokuda et al., 1989 [16]</td>
<td>Male, 37</td>
<td>Gingival ulcers, weight loss, abdominal pain, distention, ascites, peritonitis due to ileum perforation</td>
<td>Nasal symptoms 2 years; systemic disease several weeks</td>
<td>CRP</td>
<td>Rhinorrhea, arthralgia, fever, nasal blockade, necrotizing glomerulonephritis</td>
<td>X-ray: abdominal ascites</td>
<td>Biopsy from perforated ileum, granulomatous vasculitis</td>
<td>ND</td>
</tr>
<tr>
<td>Srinivasan and Coughlan, 1999 [17]</td>
<td>Female, 56</td>
<td>Crampy abdominal pain, perforation, weight loss</td>
<td>8 weeks</td>
<td>ESR, CRP</td>
<td>Rhinorrhea, arthralgia, fever, nasal bridge swelling</td>
<td>Barium study: normal Laparotomy: areas of perforation</td>
<td>Granulomatous reaction surrounding perforation without inflammation</td>
<td>c-ANCA</td>
</tr>
<tr>
<td>Author, year of publication</td>
<td>Gender, age (years)</td>
<td>Presenting symptoms</td>
<td>Disease duration</td>
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<td>Geraghty et al., 1986 [18]</td>
<td>Male, 46</td>
<td>Weight loss, palate ulcerations, peritonitis</td>
<td>4–8 weeks</td>
<td>ESR</td>
<td>Fever, lung and kidney involvement, fingers and toes ischemia</td>
<td>Chest X-ray: free air in abdominal cavity Laparotomy: inflammation of entire small bowel with numerous areas of necrotic ulceration, multiple perforations in terminal ileum Autopsy: large perforations of ascending colon and multiple punched out ulcers of distal small bowel and proximal large bowel; granulomata in lungs, spleen, and prostate</td>
<td></td>
<td>RF</td>
</tr>
<tr>
<td>Haworth and Pusey, 1984 [19]</td>
<td>Female, 43</td>
<td>Mouth ulcer, bloody mucous diarrhea, weight loss, anorexia</td>
<td>11 months</td>
<td>Not reported</td>
<td>Sinusitis, otitis, deafness, arthritis, iritis, rash, kidney involvement</td>
<td>Sigmoidoscopy: ulcerated rectal mucosa with spontaneous bleeding Barium meal: narrowed terminal ileum and cecum Biopsy: neutrophilic infiltration in rectal and sigmoid mucosa</td>
<td></td>
<td>RF</td>
</tr>
<tr>
<td>Aymard et al., 1990 [21]</td>
<td>Male, 46</td>
<td>Anal ulcerations</td>
<td>Six months before systemic vasculitis</td>
<td>ESR</td>
<td>Weight loss, polyarthritis, otalgia, hearing loss, Lt. peripheral facial nerve palsy, hemoptysis Colonoscopy: normal X-rays of small intestine normal Chest X-ray: right pulmonary apical lesion with cavitation CT: mastoiditis and otitis media Necrotizing vasculitis, ulcerations and microgranuloma</td>
<td></td>
<td>ANCA</td>
<td></td>
</tr>
<tr>
<td>Akça et al., 2005 [22]</td>
<td>Male, 56</td>
<td>Tongue plaques, intestinal perforation</td>
<td>Before diagnosis</td>
<td>ESR, CRP</td>
<td>Pulmonary nodules, severe skin vasculitis</td>
<td>Chest and abdomen X-ray: free air under the diaphragm Mucosal ulceration, mixed inflammatory cells infiltration and transmural infarcts</td>
<td></td>
<td>c-ANCA</td>
</tr>
</tbody>
</table>

Notes. APR = acute phase reactant; CT = computed tomography; ANCA = anti-neutrophil cytoplasmic antibody; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein; PR3 = proteinase 3; GI = gastrointestinal; CTA = computed tomographic angiography; GPA = granulomatosis with polyangiitis; c-ANCA = cytoplasmic anti-neutrophil cytoplasmic antibody; ND = not done.
reported [28, 29]. Holl-Ulrich and Klass described a patient with necrotizing granulomatous changes in the liver, lungs, parotid glands, and skin in a GPA patient with fatal liver failure [30]. Our patient had multiple nodules in the liver, spleen, and kidneys. Liver biopsy in our patient demonstrated interface hepatitis without granulomas.

Before the 1990s, the main tools for assessment of GPA patients were X-rays with contrast and endoscopy. CT imaging became available later and was found to be very useful. It was obvious from analyzed cases that endoscopy had limited value in the diagnosis of vasculitis, as the main findings were mucosal ulcerations which are not specific for GPA (our patient did not have mucosal damage on endoscopy). In cases with active GIT bleeding on endoscopy, the diagnosis of vasculitis was more reasonable. Inability to perform the deep transmural biopsy during endoscopy also limited the diagnostic value of endoscopy; in the majority of reported cases GPA diagnosis was confirmed in resected bowel specimens or, unfortunately, in autopsies. In the 1980s, the main immunologic abnormality was positive rheumatoid factor (three cases); later, ANCA became a standard diagnostic tool and was reported positive in all but one case, c-ANCA or PR3 in particular (our patient had a transient MPO positive test).

The definitive diagnosis of GPA is based on tissue biopsy, mainly from upper respiratory tract or lung tissue. It is very clear from the Walton report that the presence of granulomas with neutrophils accumulation is a prominent and specific sign, while vascular wall fibrinoid necrosis is nonspecific and probably could only reflect the damage to the blood vessel wall by granuloma [23].

The diversity of GPA clinical manifestations that includes systemic signs (sort of “B-signs”) and multiple organ and tissue involvement placed GPA in the list of great “imitators”, as it shares various clinical features with other systemic diseases such as hematologic malignances and infections, making the definite diagnosis very challenging. Given the fact that our patient had a combination of listed features, positive ANCA, and findings on open lung biopsy suggestive of necrotizing vasculitis, the working diagnosis of GPA was justified, as was treatment with glucocorticoid pulse therapy. Our patient developed intestinal perforations. “Acute abdomen” with intestinal perforations has been described in GPA patients with GIT involvement [9, 16, 17, 22]. In cases with acute abdomen, laparotomy is mandatory. In our patient, biopsies from resected bowel segments showed signs of diffuse large B cell lymphoma cells. Retrospective revision of lung pathology confirmed that the damage to the blood vessels was mainly due to an accumulation of lymphocytes in the involved tissue but not neutrophil accumulation or granulomas.

Different types of lymphoma may mimic different types of vasculitis. Lymphoma may be associated with ANCA positivity for both PR3 and MPO without vasculitis [31]. In a series of 119 patients with non-Hodgkin’s lymphoma and 60 patients with Hodgkin’s lymphoma, ANCA positivity was found in 8 patients (6 with p-ANCA and 2 with c-ANCA) with Hodgkin's lymphoma. None had vasculitis or rheumatic manifestations [32]. We could not explain the inconsistent results of positive MPO; in our institution the test for ANCA was negative. Despite the rare association between lymphoma and vasculitis, vasculitis in lymphoma patients mainly presents with skin purpuric rash and leukocytoclastic vasculitis on biopsy; our patient did not have any rash.

In searching the literature, we were able to find three cases of diffuse large B cell lymphoma mimicking GPA [33–35]. All these patients, like our patient, were males, aged between 20 and 40 years of age, and had pulmonary involvement; all had negative ANCA. None of these patients, in contrary to our patient, had liver or spleen involvement; one patient had renal involvement. In the case reported by Cohen et al., the patient was diagnosed with GPA and initially “responded” well to steroids and cyclophosphamide; only because of severe relapse under the treatment, revision of the biopsy leads to the proper diagnosis of lymphoma.

Did we have some clues to doubt the diagnosis of vasculitis in a patient who presented with systemic signs, multiorgan clinical presentations, presence of pulmonary nodules, positive MPO, and signs of vascular damage on open lung biopsy? A retrospective analysis of the case could teach us to pay attention to tiny discrepancies in the patient's condition: the anemia in the absence of upper respiratory tract involvement was probably a “red herring”; the presence of multiple small and not consistent pulmonary nodules without cavitational is not a typical GPA feature; the presence of nodular lesions on CT imaging (“macroscopic”) in the spleen, kidneys, and liver without signs of hemorrhage is not typical for GPA; severe dyspepsia (abdominal pain, vomiting, and constipation) without any mucosal damage (ulceration or bleeding) on upper endoscopy could not be explained by vasculitis, especially in the absence of diarrhea or intestinal/rectal bleeding. GPA is a disease with neutrophils activation and accumulation in organs and tissues; our patient did not have leukocytosis. Also, he had only mildly elevated ESR and CRP in contrast to very high levels in patients with GPA. It is unusual not to find c-ANCA (especially PR3) in patients with active GPA and multiorgan involvement as in our patient. Finally, the absence of granulomas in the involved lung tissue with only the pathological finding of vessel wall damage should raise the question regarding a diagnosis of vasculitis or a search for an alternative diagnosis.

We believe that in cases of atypical clinical course of systemic illness suggestive of vasculitis (GPA), the differential diagnosis should include lymphoma. Prompt efforts should be invested to confirm the diagnosis of vasculitis or to ensure that another condition, such as lymphoma, is not the cause of the disease. Our report pointed out the difficulties and obstacles on the way to making the correct diagnosis in patients with B cell lymphoma mimicking systemic GPA-like vasculitis.

Competing Interests

All authors declare no competing interests.

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References


