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Ulcerative Pyoderma Gangrenosum and CIDP Associated with Chronic Renal Failure: a Case Report and Review of The Literature

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Pyoderma gangrenosum (PG) is a rare skin disease characterized by non-infective ulcers. Chronic inflammatory demyelinating polyneuropathy (CIDP) is a nervous system disease characterized by progressive or recurrent muscle weakness and minor sensory problems. Both diseases are associated with systemic disorders. A 30 year old male patient with overlapping PG and CIDP on the background of chronic renal failure was reported here. Treatment with etanercept, an anti-TNF-α agent, improved PG manifestations but did not improve the neurological complaints and signs of CIDP.

Key Words**:** *Chronic inflammatory demyelinating polyneuropathy, etanercept, pyoderma gangrenosum, renal failure.*

Kronik Böbrek Yetmezliği ile İlişkili Ülseratif Piyoderma Gangrenozum ve CIDP: Bir Olgu Sunumu ve Literatürün Derlemesi

Piyoderma gangrenozum (PG), non-infektif ülserlerle karakterize nadir görülen bir deri hastalığıdır. Kronik inflamatuvar demiyelinizan polinöropti (CIDP), progresif veya tekrarlayıcı kas güçsüzlüğü ve minor duysal problemlerle karakterize sinir sistemi hastalığıdır. Her iki hastalık immun hastalıklarla ilişkilidir. Bu yazıda, kronik böbrek yetmezliği zemininde, PG ve CIDP çakışması olan bir hastayı sunmaktayız. Bu hastada, bir anti-TNF-α ilaç olan etanersept PG lezyonunu yatıştırdı, ancak nörolojik yakınmalar üzerine etkisizdi.

Anahtar Kelimeler: Böbrek yetmezliği, etanersept, kronik inflamatuvar demiyelinizan polinöropati, piyoderma gangrenosum.

Introduction

Pyoderma gangrenosum (PG) is an idiopathic neutrophilic reactive inflammatory dermatosis (1) and it is associated with underlying systemic diseases in about 50% of the patients (2). The relations of PG with rheumatological, infectious and inflammatory intestinal diseases, immune disorders, medications and hematological diseases including myeloproliferative ones, myeloma and paraproteinemias are well established (1, 2).

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an autoimmune nervous system disease that either occurs with monophasic relapses or has a progressive course. CIDP characterized by progressive symmetrical muscle weakness, hyporeflexia or areflexia (3,4) is associated with connective tissue diseases, chronic infectious diseases and paraproteinemias (3).

The pathogenesis of PG and CIDP are not fully elucidated. However, it appears that they have immunological basis (3) and they are associated with similar immune and/or inflammatory disorders and treated with similarly such as with corticosteroids and immunosuppressives (2,3).

Chronic renal failure (CRF) causes various impairments in immune functions, and thus various immune diseases may develop in uremic cases (5). A patient with overlapping ulcerative PG and CIDP on the CRF background are reported herein.

Case report

A thirty-year-old male patient complaining from ulcerative lesions on knees and loss of strength in legs for 5 months is presented here. He was diagnosed 4 years ago as CRF secondary to chronic glomerulonephritis and did not receive any treatment for CRF until his admission to our clinic. A year ago he received tetracycline and rifampicin for the treatment of brucellosis. Ulcerative lesions developed on his knees during that period and recovered spontaneously within 2 months.

His physical examination showed ulcerative lesions on knees at his recent admission to our clinic (fig. 1). The bases of the ulcers were covered by red granulation tissue and purulent exudate. The margins were elevated all around and smoothly bordered by granulation tissue. He had not any cognitive disorder or meningial irritation signs. Pupils were bilaterally isochoric, isocyclic, and responded to light well. Evident loss of strength in the distal and proximal parts of the lower extremities, muscular atrophies and bilateral dropped foot were verified. The patient was unable to walk without support. Superficial senses of the lower extremities were impaired and there was no deep tendon reflexes. The senses of position and vibration were moderately impaired. Autonomous functions like urination and defecation were normal. He had not organomegaly or lymphadenopathy.

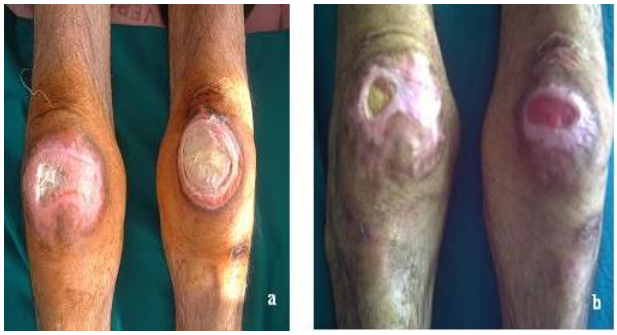

Figure 1. Ulcerative PG on the legs (a) and significant improvement of the lessions after etanercept therapy (b).

Laboratory analyses showed that hematocrit level decreased and erythrocyte sedimentation rate, C-reactive protein, urea and creatinine levels increased. Leukocyte count, ALP, LDH, bilirubin, albumin, globulin, lipids profile and fasting blood glucose were within the normal range. Serological analysis conducted for HIV, HAV, HBV, HCV, and Bence-Jones protein, β_2 microglobulin, protein electrophoresis, complement components (C_3 and C_4), ANA levels were either in the normal range or negative. PPD test was anergic. Colonoscopy and bone marrow examinations were normal. No microorganisms was isolated in the cultures taken from the ulcerative regions. The histopathological examination of the biopsy materials taken from the ulcers revealed edema, fibrosis and dermal inflammation marked with neutrophils and lymphocytes.

X-ray graphies, cerebral and lumbar computerized tomographies were normal. Cerebrospinal fluid did not reveal cells and had high protein levels. The electromyographic (EMG) examination showed significant decrease in motor nerve conduction velocity and muscular action potentials were significantly slower without a conduction block. Sensory conduction studies revealed moderately slower potentials in the upper and lower extremities. The patient was interpreted as CIDP according to his symptoms, physical examination, EMG and cerebrospinal fluid findings.

Treatment with 1 mg/kg/day methylprednisolone was administered. The response to corticosteroid was not sufficient at the end of the four-week follow-up time. An anti-TNF agent etanercept which was reported to be efficient in PG (6-9) and CIDP (10) was added to the treatment regimens with a dosage of 25 mg/week/sc. The corticosteroid dosage was gradually tapered to 24 mg/day. In the eighth week of the etanercept treatment, about 80% regression was noted in PG lesions (fig. 1). However, the neurological complaints and signs pertaining to CIDP did not improve.

Discussion

PG is a rare ulcerative skin disease and has been firstly defined by Brocel[2] in 1908 as *phagedisme geometrique*. Brunstig *et al*. (11) have proposed the term PG in 1930. PG presents as erythematosus papulopustules or vesicles and gradually shows smooth-bordered ulcers with irregular bases. The ulcers spontaneously either grow or recover after leaving atrophic scars (1-3). It has been reported that 50-70% of the PG cases have an underlying systemic disease (2). The patient presented in this report had not any diseases other than CRF. So far, PG has been reported in 5 cases with overlapping PG and renal diseases [one case as acute renal failure (12), two cases as rapidly progressive glomerulonephritis (13, 14) and two cases as CRF (15, 16)] in the literature (Table 1).

Pathogenesis of PG is not known in the whole (2), but the level of IL-8, a potent chemotactic agent, elevates in PG cases (17). TNF-α is a proinflammatory cytokine, which induces the production of IL-8, nevertheless there is no study related to the role of TNF-α in the pathogenesis of PG.

Management of PG initiates by dealing with the underlying disease and primary targets are diminishing pain, helping re-epithelization and finally minimizing the lesion and scar diameters (18). Although systemic corticosteroids are popular and the first-line agents in the treatment of PG some of the cases are resistant to this therapy. Additionally, corticosteroids are used in high

Table 1. Summary of pyoderma gangrenosum associated with renal disease reported in the literature

Age (years)	Sex	Associated conditions	Type of PG	Ref.
23	M	Acute renal failure	Vegetative	12
50	M	RPGN, IgA monoclonal gammopathy	Ulcerative	13
66	M	RPGN, Polycythemia vera	Ulcerative	14
58	F	CRF	Vegetative	15
N/A	N/A	CRF, rectal carcinoma	Vegetative	16

M; male, F; female, RPGN; rapidly progressive glomerulonephritis, CRF; chronic renal failure, N/A; unknown data

doses and with long periods of time for the treatment of PG and they can also lead to severe side effects. Immunosuppressive agents, such as cyclosporine, cyclophosphamide, methotrexate and tacrolimus are alternative agents for the treatment in the cases resistant to corticosteroids (1-3). Cyclosporine which is effective fairly is administered orally or intralesionally (18). However, the use of cyclosporine is restricted due to the facts of nephrotoxicity in oral usage and severe pain in intralesional usage. Hepatotoxicity, myelosuppression, infection and malignancy might be the potential harmful effects of the other immunosuppressives. Although corticosteroids and conventional immunosuppressives take important places in PG management, their effects may be variable (18, 19). In addition, there is no acceptable treatment guideline for PG.

Infliximab, etanercept and adalimumab the anti TNF-α agents are successfully used in the treatments of rheumatoid arthritis and spondilarthropathies. Anti-TNF therapy has been firstly used by Tan *et al*. (20) in PG and is reported as to be effective. In the following years, anti-TNF agents have been used in approximately 90 cases with PG [some of them are shown in the Table 2] (6-9, 19-37). Infliximab which has been administrated to 13 PG cases associated with inflammatory bowel disease completely recovered skin lesions in all of them (35). In three cases, single dose treatment has provided healing (35). In a controlled prospective study (19), successful results have been obtained with a proportion of 69% by a single dose of infliximab in the treatment of PG. In similar studies, infliximab has been found successfully effective as 80%[36] and 100%[35] in the scheme of recommended use in clinical practice. Ljung *et al*. (37) have reported complete recovery in 3 cases, partial recovery in 3 and permanent recovery in 2 cases for the treatment of PG associated with Crohn's Disease (CD) by using infliximab. In this study (37), fever, abdominal pain and pneumonia developed as side effects of infliximab. Hubbard *et al*. (28) have treated a case of PG with infliximab but they could not continue the treatment due to anaphylaxis. In that case, during follow-up, etanercept has been determined to be ineffective in recurrence that developed later, and healing has been provided in skin lesion by using adalimumab. In a PG case associated with PAPA (pyogenic sterile arthritis, PG and acne) syndrome (32), tacrolimus, mycophenolate mofetil, cyclosporine, etanercept and anakinra administrations have been found to be ineffective, and then infliximab supplied an effective treatment.

Table 2. Some of the pyoderma gangrenosum reported cases treated with anti-TNF-α agents in the literature

Age (yrs)	Sex	Related illness	Anti-TNF-α	Time to Response	Relapse	Concomitant medications	Ref.
46	F	CD	Infliximab	CR 4 wk	No relapse 13 mo	CS	21
41	F	CD	Infliximab	CR 11 wk	-	-	22
52	F	CD (only PPG)	Infliximab¶	CR 4 wk	No relapse 8 mo	Mycophenolate mofetil	23
81	F	CD	Infliximab	PR 6 wk, CR 6 mo	-	Azathioprine	23
60	F	CD	Infliximab	PR 6 wk, CR 6 mo	-	CS	23
58	M	RA, acne rosacea, CD	Infliximab	CR 8 wk	No relapse 9 mo	Azathioprine, CS	24
39	M	Idiopathic	Infliximab	CR 16 wk	No relapse 12 mo	-	25
60	F	CD	Infliximab	CR 12 wk	-	-	26
59	F	CD	Infliximab	CR	-	-	26
55	F	CD	Infliximab	CR	-	-	26
58	F	CD (only PPG)	Infliximab	CR	-	-	26
45	M	Ulcerative colitis	Infliximab	CR 3 mo	No relapse 12 mo	Azathioprine	27
43	M	CD	Infliximab‡	CR 3 wk	-	-	29
57	F	CD	Infliximab	CR 8 wk	No relapse 12 mo	CS	30
23	M	Idiopathic	Infliximab	CR 4 mo	No relapse 6 mo	Cyclosporine, CS	31
13	M	PAPA syndrome	Infliximab	CR 8 wk	No relapse 8 mo	-	32
63	M	CLL	Infliximab	CR 3 mo	No relapse 6 mo	Cyclosporine, CS	33
52	M	CD	Infliximab	CR	-	-	34
26	F	CD	Infliximab	CR	-	-	34
21	F	CD	Infliximab	CR	-	-	34
24	F	CD	Infliximab	CR	-	-	34
30	F	Autoimmune hepatitis	Etanercept	CR 5 mo	-	Tacrolimus ointment, CS	7
34	F	Idiopathic	Etanercept	CR 4 wk	10 wk relapse§	CS§	6
44	F	RA, SLE	Etanercept	CR 2 mo	-	Hydroxy-chloroquine, CS	8
48	F	RA	Etanercept	PR 210 day	-	-	8
38	M	Idiopathic	Etanercept	CR 2 mo	-	-	8
83	F	Recurrent aseptic abscess	Etanercept	CR 2 mo	No relapse 12 mo	-	9
27	M	Idiopathic	Adalimumab	CR 3 wk	-	-	28

M; male, F; female, CD; Crohn's disease, PPG; peristomal pyoderma gangrenosum, RA; rheumatoid arthritis, SLE; systemic lupus erythematosus, CR; complete remission, PR; partial remission, CLL; chronic lymphocytic leukaemia, CS; corticosteroid, mo; month, wk; week.

‡Treatment with infliximab had been complicated by the development of miliary tuberculosis.

¶At the end of the 10th week, having observed enlargement in ulcerative lesion and mild pain, preserved treatment has been used with 6 weeks intervals.

§When CS was lowered as 2.5 mg/day, having developed a relapse and the second relapse after a motorbike accident, etanercept was continued to be given as 100 mg/wk.

Etanercept therapy has been reported in 8 cases with PG (6-9, 28, 32) and complete remission has been observed in 5 cases and partial remission in one, during 1-5 months. The remaining two which are associated with PAPA syndrome (32) and idiopathic PG (28) have not responded to the etanercept therapy. In our case, etanercept was also effective in PG treatment. Although anti-TNF-α agents are not the first-line options, they might be preferred in induction of remission when good responses are not found with conventional treatments and/or severe side effects are observed. Another advantageous of anti-TNF-α agents might be their rapid effectivity in a short time period.

Reversible demyelinating motor neuropathy might also develop on the base of renal failure (38). In the literature 6 CIDP cases related to glomerulonephritis are reported as case reports (39-44). In the case presented here, we suggest that CIDP is related to CRF, since there is no other provocative disease. In 25% of the cases diagnosed as CIDP, it is reported that the level of TNF-α elevates (4). In addition, it has been found that neutralization of TNF-α has a diminishing effect on Schwann cell apoptosis in experimental autoimmune neuropathy (45). Anti-TNF-α agents have also been reported to be effective in the management of CIDP (10).

Corticosteroids, intraveneous immunoglobulin (IVIg) and plasma exchange are effective treatments in CIPD (46). In cases resistant to the above treatments, azathioprine, cyclosporine, methotrexate, cyclophosphamide and interferons might be used (46). Nevertheless, it has been reported that complete remission has been obtained in only one of the three cases with CIDP by using above agents in a retrospective study (47) and response to the treatment**s** even with the second and the third generation drugs has been observed to be in 66% of the cases (48).

The case presented here was determined as resistant to the steroid administration. As the frequency of acute renal failure has been reported to be as 13% in the cases treated with IVIg (49), we did not prefer usage of IVIg due to the presence of renal failure in our case, and etanercept was administreted. Chin *et al*. (10) have reported remarkable recovery in three and partial recovery in three cases with CIDP treated with etanercept (25 mg, twice weekly) for 4 to 6 months. They have also reported recovery in functional scoring in contrary to getting worse in sensorial scores and impairment of all the scores in another three patients and no response in their last case (10). As a result, they have suggested that etanercept might be used in CIDP cases refractory and/or intolerant to conventional treatments (10).

In our case, while PG manifestations have recovered by etanercept treatment, the adequate response for CIDP has not been observed. This inadequate effect might be resulted from the administration of etanercept for a short time period and its low dose because of CRF in this patient.

To our knowledge, the case presented here is the first one in whom PG and CIDP overlapped on the base of CRF. Etanercept, an anti-TNF-α agent, might be useful in the treatment of PG when conventional modalities are inefficient.

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