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Case Report

Pseudomonas bacteremia as an initial presentation of SLE

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ABSTRACT

Infections have been commonly implicated in lupus relapses and in some cases as initiating the diagnostic work up of systemic lupus erythematosus (SLE). We describe here the case of a young patient who presented with *Pseudomonas aeruginosa* bacteremia and was found to have a new diagnosis of SLE. 53% of patients with active SLE and abdominal pain have intestinal vasculitis. These vasculitic changes can cause intestinal ischemia with consequent translocation of pathogens from the gastrointestinal tract to the bloodstream causing sepsis.

Background

Infections have been commonly implicated in lupus relapses and in some cases as initiating the diagnostic work up of systemic lupus erythematosus (SLE). A previously described association in literature is that of *Salmonella typhi* bacteremia and *Cryptococcal neoformans* meningitis occurring concurrently with the first presentation of SLE [1–3]. We describe here the case of a young patient who presented with *Pseudomonas aeruginosa* bacteremia and was diagnosed to have SLE.

Case report

A 24 year old Chinese male was admitted to the hospital with fever with rash. Two weeks prior to presentation, patient developed subjective fevers with malaise and fatigue. He then developed a non-pruritic, non-vesicular painless rash in a centrifugal distribution. Rash started on trunk and progressed to arms, palms and face, sparing the lower extremities. He also developed painful ulcerations of oral mucosa. Associated symptoms were a cough productive of clear sputum, myalgias, constipation and 10 lb weight loss in 2 weeks. Patient denied any past medical history, sick contacts, recent hospitalization, smoking, drug use, animal contact, unprotected sexual encounter or foreign travel. Family history was non-contributory. Vital signs on admission were as follows-temperature 103.5, pulse rate 91 bpm, blood pressure 121/60 mmhg, respiratory rate 17/min, oxygen saturation 97% on room air. Physical exam showed oral mucosal ulcerations with thrush, cervical lymphadenopathy, splenomegaly, multiple discrete 'salmon colored' spots on chest, back, palms and arms. Initial blood tests revealed pancytopenia (Table 1). Chest X-ray did not show any infiltrates.

Initial presentation suggested a mononucleosis-like syndrome hence serologies for EBV, HIV, CMV, Coxsackie, Rubella, Measles were drawn that later returned no diagnostic. Blood culture on admission was positive for *P. aeruginosa* sensitive to cefepime, gentamicin, imipenem and ciprofloxacin. He was started on intravenous cefepime. Despite appropriate antibiotics, patient remained febrile and gentamicin was added to the regimen. An abdominal ultrasound done for elevated liver enzymes revealed splenomegaly. Due to multi-system involvement, an autoimmune etiology was suspected. Corresponding workup found (Table 1) ANA positive at 1:160, positive anti-DS DNA and anti-SM and hypocomplementemia with C3 21 and C4 5. Given these laboratory tests consistent with the diagnosis of SLE, patient was started on steroids and hydroxychloroquine. Over the next few days, patient improved clinically with resolution of fevers, fatigue, and malaise. His oral ulcers were healing and he was able to tolerate oral

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Table 1
Laboratory values.

Parameter	Reference range	At initial evaluation	Follow up
White blood cell count, cell/mm^3	4500–10,800	2600	4500
Absolute neutrophil count	1800–8000	2100	3800
Hemoglobin, g/dL	13.5–17	12.8	12.3
Platelets, cell/mm^3	150,000–450,000	55,000	172,000
Erythrocyte sedimentation rate, mm/h	0–30	14	15
C reactive protein, mg/dl	0–1	<0.5	0.7
Ferritin, ng/ml	22–322	10,262	3355
Creatinine kinase, units/L	55–170	10,887	1299
Aspartate transaminase, units/L	15–46	812	19
Alanine transaminase, units/L	13–69	511	24
Alkaline phosphatase, units/L	38–126	107	57
Total bilirubin, mg/dL	0.2–1.3	0.8	0.3
ANA		1:160	–
Rheumatoid factor, IU/ml	<11	<11	–
Anti Smith antibody, EU/ml	<16	25	–
ds DNA, IU/ml	0–29	>300	>300
Sjogren Ab, EU/ml	<16	156	–
SM – RNP Ab, EU/ml	<16	80	–
Complement – C3, mg/dL	88–201	21	43
Complement – C4, mg/dL	16–47	5	7

feedings. Liver function abnormalities improved from those at admission. Repeat blood cultures were negative. Patient was discharged on intravenous antimicrobials to complete treatment in addition to prednisone and hydroxychloroquine.

Discussion

SLE is a chronic inflammatory multisystem disease with immunological abnormalities and seen more often in women than men. There is a known association between immunosuppression caused by medications used to treat SLE and the increased propensity of infections [5]. There have been few cases described of *Salmonella enterica serotype typhi* bacteremia as initial presentation of SLE in patients not on immunomodulators [3]. *P. aeruginosa* is frequently encountered in nosocomial infections especially in immunocompromised patients. *P. aeruginosa* bacteremia can be traced back to several sources including but not limited to contaminated water, gastrointestinal tract, lungs and indwelling catheters [6]. 53% of patients with active SLE and abdominal pain have intestinal vasculitis. A negative abdominal examination does not rule out disease as SLE involves small vessels [4]. These vasculitic changes can cause intestinal ischemia with consequent translocation of pathogens from the gastrointestinal tract to the bloodstream causing sepsis.

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