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CASE REPORT

IgA Vasculitis in Middle Aged Man Followed by Influenza Virus Infection: A Case Report

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ABSTRACT

Introduction: Immunoglobulin A vasculitis (formerly known as Henoch Schonlein Purpura) is the most common pediatric vasculitis, which occurs typically at the age of 3-15 years. Mononucleosis, group A Streptococcus, Campylobacter and Mycoplasma are some of the common infectious causes of immunoglobulin A vasculitis. Immunoglobulin A vasculitis is a clinically diagnosed disease. Most common clinical features include nonthrombocytopenic nonpruritic palpable purpura, gastrointestinal involvement, arthritis or arthralgia and renal involvement. Biopsy of skin and/or gastrointestinal lesions confirm the diagnosis, although this is not necessary. Immunoglobulin A vasculitis is a self-limiting disease, which resolves spontaneously. Steroids can be used for the treatment of moderate to severe disease and for the prevention of renal complications. Prognosis relies upon various factors among which involvement of kidneys dictates poor prognosis and requires close follow up. **Case Presentation:** We are presenting a case of immunoglobulin A vasculitis in a 39-year-old German male following influenza virus infection. The infectious agent that cause immunoglobulin A vasculitis in our patient was Influenza A virus. Biopsy of the skin lesion confirmed the diagnosis of immunoglobulin A vasculitis in our patient. The patient responded to steroids and his skin and GI findings resolved. **Conclusion:** Our case report adds to the literature of medicine by describing influenza virus as a cause of immunoglobulin A vasculitis in young adult patients. Considering immunoglobulin A vasculitis after influenza and outside of the typical age of 3-15 years, can help to make earlier diagnosis and prevent complications.

INTRODUCTION

Immunoglobulin A (IgA) vasculitis is a self-limiting, autoimmune, systemic, most commonly pediatric, small vessel vasculitis (1,2,5). The classic age range of IgA vasculitis occurrence is 3 to 15 years (8). This disease is uncommon in adults. Precipitating factors of IgA vasculitis include infections, medications, vaccinations, insect bites, and food allergies. The common infectious causes that are responsible for causing IgA vasculitis include Group A streptococcal infection, Campylobacter enteritis and Mycoplasma infection (8,11). This report presents a case of a 39-year-old male who developed classic features of IgA vasculitis10 days after being diagnosed with influenza A virus infection.

CASE PRESENTATION

A 39 year old German male came to the clinic with history of high fever, chills, headache, cough, back pain, and runny nose for 3 days. He was diagnosed with influenza A virus infection on rapid influenza test and was prescribed Tamiflu for 5 days. He came back to the clinic 10 days later with a rash, which he noticed on his legs while travelling for work. The rash was nonpruritic, non-painful, and purpuric. It progressed from his bilateral legs and inner thighs to his abdomen, back, chest, and face. He was referred to a dermatologist who performed a skin biopsy. Pathology confirmed the diagnosis of IgA vasculitis. Four days later, he returned with severe worsening abdominal pain, progressive rash on his thighs, legs, back, and arms. He also complained of arthralgia of bilateral knees and elbows. Abdominal pain was episodic, located in the epigastrium, 8/10 in intensity, colicky in nature, non-radiating, and had no identified aggravating or relieving factors. Each episode lasted for a few minutes. He was referred to emergency room (ER) where he was started on 20 mg prednisone and discharged. Six days later, he developed nausea, diarrhea, hematemesis and severe abdominal pain. Review of the systems was normal except above mentioned problems. Hypertension, fatty liver disease and, obesity are his significant comorbidities. His past medical history includes a transient ischemic attack (TIA) 2 years ago. He is allergic to bee stings. His family history includes coronary artery disease and type 2 diabetes mellitus (DM) in

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his father. Physical exam showed diffuse palpable purpura of the legs, thighs, back, arms and face (Figure 1 a-d). His abdomen was moderately tender to palpation. There was no redness, warmth, or swelling of his knee and ankle joints.

INVESTIGATION

His laboratory tests include:

- Complete blood count (CBC), White blood cells (WBCs): 21.9 X 1000/ul; Hemoglobin 18g/dl; absolute neutrophils 15.8 X 1000/ul; absolute lymphocytes 4.4 X 1000/ul; absolute monocytes 1.5 X 1000/ul; platelets 416 X 1000/ul
- Urinalysis was normal with no hematuria or proteinuria
- Urea nitrogen: 18 mg/dl
- Creatinine: 0.72 mg/dl
- Erythrocyte sedimentation rate (ESR): 6mm/hr
- C-reactive protein (CRP): 5.94 mg/dl
- Hepatitis A Antibody: non-reactive
- Hepatitis B surface antigen (HbsAg): non-reactive
- IgA Total Serum: 304 mg/dl
- IgE Total Serum: 79.5 mg/dl
- IgG Total Serum: 891 mg/dl
- IgM Total Serum: 51mg/dl
- Skin biopsy was taken from right inner thigh and showed deposition of IgA, C3 and fibrinogen in vessels.
- Endoscopy showed 5 cm long, dusky appearing, localized hemorrhagic and edematous friable circumferential mucosa without active bleeding but with bloody exudate in the second part of duodenum. (Figure 2). Follow up endoscopy 2 months after starting steroid therapy showed normal duodenal mucosa having no evidence of inflammation or ulceration.
- Duodenal biopsy showed ulceration and fibrinous exudate
- Computed tomography (CT) of the abdomen showed abnormal thickening of cecum and terminal ileum with surrounding inflammatory changes as well as thickening of adjacent distal duodenum and proximal jejunum.

TREATMENT

Patient was started on prednisone 20 mg which was increased to 60 mg on development of severe abdominal pain, hematemesis, nausea, and diarrhea. He was also prescribed clobetasol for the skin rash.

OUTCOME AND FOLLOW UP

The patient's symptoms improved with prednisone. Early diagnosis and treatment prevented renal complications. He is now completing a slow prednisone taper and is doing very

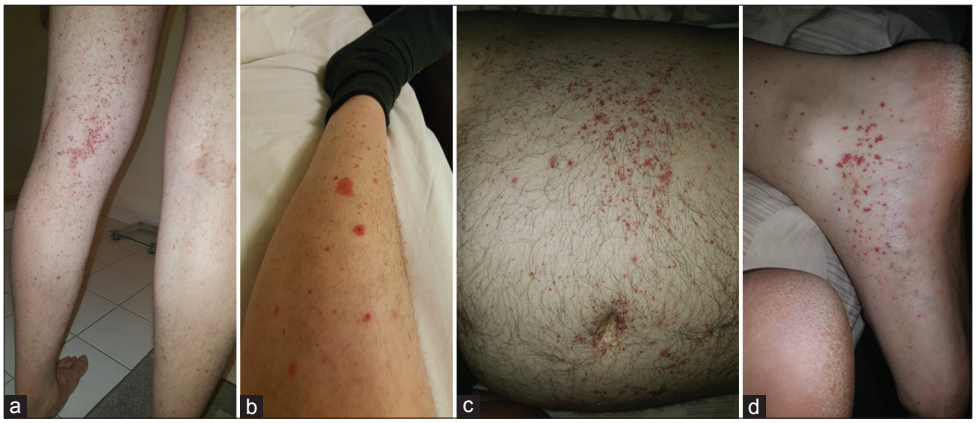

Figure 1. (a) Cutaneous lesions on lower extremities: Palpable purpura on left lower thigh and popliteal region, (b) Skin lesions on lower extremities: Palpable purpura on right leg, (c) Cutaneous lesions on abdomen: Scattered palpable purpura around umbilicus, left abdominal quadrant and hypogastrium, (d) Skin lesions on right ankle and foot: Multiple scattered palpable purpura on right ankle and foot

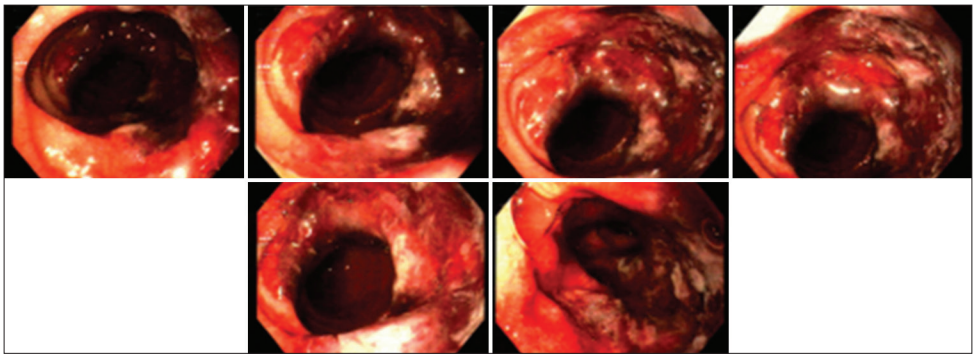

Figure 2. Endoscopic findings: Hemorrhagic and edematous mucosa of second part of duodenum

well. He has had no subsequent abdominal pain and no new rash since his last visit to the ER.

DISCUSSION

IgA vasculitis is a systemic, autoimmune, small vessel vasculitis, which commonly involves the vessels of the skin, joints, gastrointestinal tract, and kidneys. (1) It is most common in pediatric populations with 90% of IgA vasculitis cases occurring before the age of 10 years old (10). However, our patient developed IgA vasculitis at the age of 39, which is noteworthy. IgA vasculitis can be considered in the differential diagnosis of patients outside of typical age range and timely treatment should be provided.

Statistically, IgA vasculitis is twice as common in males as females and occur predominantly in winter and spring. The gender of our patient is male and he developed this disease in winter season. (1, 4, 10). Globally, the incidence of IgA vasculitis is highest among Asians. In the United States, Caucasians most commonly present with this disease. (19). Our patient was Caucasian which is consistent with the typical presentation of IgA vasculitis.

IgA vasculitis is considered a multifactorial disease with genetic, environmental and immunologic components. Infections (viral and bacterial), vaccination (typhoid, cholera, measles and yellow fever), drugs (antibiotics, non-steroidal anti–inflammatory drugs (NSAIDS) and angiotensin receptor blockers (ARBS), insect bites and certain foods are the precipitating factors. The common infectious causes of IgA vasculitis are group A streptococcus infection, Mycoplasma infection, Campylobacter enteritis, Parvovirus, and Adenovirus (8, 11). IgA vasculitis in our patient followed influenza virus infection which is a very rare precipitating factor of IgA vasculitis. Two published case reports from China and Rome also presented influenza as a precipitating factor of IgA vasculitis. Chan Won Park and colleagues presented a case of IgA vasculitis in China in 2014. They reported IgA vasculitis in 4 years old girl with GI and renal manifestation of IgA vasculitis without skin involvement (3). However, our patient was much older and presented with typical clinical findings of IgA vasculitis. We confirmed the diagnosis with a skin biopsy.

Another report was published by Rocco Urso in Italy in 2009. In that report, a 23-year-old Caucasian woman developed IgA vasculitis following H1N1 infection (7). The case presented with typical findings of IgA vasculitis. Our patient was different from their patient as our patient was a 39 years old male who developed typical signs and symptoms of IgA vasculitis following influenza A virus infection confirmed on rapid influenza testing. Moreover, we confirmed IgA vasculitis by skin biopsy.

Rashes occur in 95-100% of cases (11). The common skin manifestation includes nonthrombocytopenic nonpruritic palpable purpura which occurs mostly in dependent parts of the body such as buttocks and lower extremities. Skin lesions can progress to upper parts of the body. Our patient presented with typical skin rashes of IgA vasculitis. Rashes appeared in both legs and thighs first and after few days the skin lesions spread to the back, arms, and face.

Gastrointestinal symptoms occur in 70 % of cases. Colicky abdominal pain is the most common gastrointestinal manifestation (8). Patients can also develop nausea, vomiting, melena, and hematochezia. Mesenteric vasculitis is the cause of the GI symptoms. Abdominal pain in our patient developed one day after developing rashes and it occurred with hematemesis, nausea, and diarrhea.

60- 84% of IgA vasculitis patients develop joint pain. Knee and ankle joints are the most commonly involved (13). Our patient developed arthralgia in bilateral knees and ankles.

In IgA vasculitis, kidneys are affected days to months after skin manifestations. 20-50% of patients have renal involvement and this plays a very important role in the prognosis of IgA vasculitis (1). It is the most common cause of morbidity and mortality (11). Renal compromise with IgA vasculitis is more common in adults. Microscopic or gross hematuria are the most common renal findings. Patients can also develop proteinuria (1). Luckily, our patient did not develop any of the abnormal renal findings but he is still under surveillance for renal disease.

IgA vasculitis is generally a clinical diagnosis. Blood tests may reveal normal or elevated platelets, mild leukocytosis, elevated IgA levels. In about 50% of cases ESR and CRP are elevated. (8) Skin or kidney biopsies can be done in unusual cases to confirm the diagnosis. Skin biopsy usually shows leukocytoclastic vasculitis with IgA deposition.

IgA vasculitis resolves spontaneously in 89% of adults. Analgesics are usually provided for abdominal and joint pain (15). Oral or intravenous (IV) steroids should be provided to the patient with moderate to severe diseases. (14). Steroid therapy early in the disease process decreases the duration of symptoms, prevents recurrence and benefits kidneys. Our patient was started on steroids early in his disease process which, may have prevented renal complication and decreased the disease duration. His blood urea nitrogen (BUN), creatinine, and urinalysis are all within normal limits.

Previous case reports have described the development of IgA vasculitis after influenza virus infection and influenza vaccination, which divert our attention towards conducting research on the pathogenesis of IgA vasculitis after influenza. (6, 20, 21, 22) Additionally, more research is needed to determine the type or strand of influenza virus that may cause IgA vasculitis. In our case, IgA vasculitis followed influenza A virus infection

PROGNOSIS

Prognosis of IgA vasculitis is good with a 95% survival rate after 5 years. (1) Short term prognosis depends upon gastrointestinal (GI) involvement and long term prognosis depends upon renal involvement. (12)

Older age at diagnosis (age more than 8 years), renal abnormalities (high creatinine, proteinuria, hematuria), and rashes above waist line are considered poor prognostic factors. (1, 16, 17, 18) The older age of our patient and rashes above waist line were two negative prognostic factors in our patient. However, early diagnosis and treatment may have prevented renal complications in our patient.

CONCLUSION

Non palpable purpura in middle aged patients after influenza should prompt consideration of IgA vasculitis as a potential diagnosis. In this way complications and multi organ failure can be avoided. Early and proper diagnosis would also help avoid unnecessary treatment.

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AUTHER CONTRIBUTIONS

All authors contributed equally.

CONFLICT OF INTEREST

None.

ETHICAL STANDARDS

We obtained written consent from the patient for the publication of this case report.

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