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Helicobacter Pylori Infection Induced Henoch Schonlein Purpura: A Case Report

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Abstract

Henoch Schonlein Purpura (HSP) also known as IgA vasculitis, is an immune complex vasculitis affecting small vessels. We report a case of 53 years old female who presented with multiple reddish-brown purpuric papules over bilateral lower limb for 10 days. Her cutaneous findings were suggestive of HSP. She also had a history of recurrent dyspepsia for the past 6 months for which she had been taking antacid from a local pharmacy infrequently. To confirm her diagnosis, skin biopsy was sent for both histopathological examination and direct immunofluorescence which were suggestive of HSP. We referred her to the medicine department for her gastrointestinal complaints where upper gastrointestinal endoscopy was performed and the finding was consistent with *Helicobacter pylori (H. pylori)* infection. The patient was started on a treatment regimen for *H. pylori* eradication which resulted in a dramatic improvement in both gastrointestinal complaints as well as cutaneous lesions. There are very few cases in the literature showing an association between HSP and *H. pylori* infection and none from our part of the world.

Key words: Cutaneous vasculitis; *Helicobacter pylori*; Henoch Schonlein Purpura.

Introduction

Henoch-Schonlein Purpura (HSP) is a leukocytoclastic vasculitis of small vessels commonly associated with skin, joints, gastrointestinal (GI), and kidney involvement.[1] HSP is an uncommon vasculitis in adults, with an estimated incidence of 8–18 cases per million per year.[2] The exact pathogenesis of HSP remains unknown, however, a wide variety of conditions like bacterial or viral infections, vaccinations, drugs, and other environmental exposures may be implicated in the onset.[3] Recently, few reports have been focusing on the possible relationship between *H. pylori* infection of gastric mucosa and vasculitis.[4] This association may be underestimated as it is not deliberately sought.[2]

Case report

A 53 years Nepalese female presented to the dermatology outpatient department (OPD) with the chief complaints of multiple red elevated non-pruritic lesions on bilateral lower limbs including buttocks for 10 days. Lesions were progressively increasing in number. She also complained of mild joint pain over the bilateral knee and ankle joint. She had history of mild fever with sore throat 2 weeks before this episode which resolved on its own without any medication. Besides this, she gave the history of recurrent dyspepsia in the last 6 months for which she had been taking antacids infrequently from a local pharmacy. There was no significant medical illness or surgical intervention in the past. There was no history of hematemesis, hematuria, melena, diarrhoea, constipation, weight loss, or history of any other drug intake.

On examination, there were multiple reddish-brown purpuric macules and papules of size approximately 3X2 mm² present discretely in a symmetrical manner over the gluteal region, extensor aspect of bilateral thighs and legs a shown in figure 1A and 1B.

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Examination of mucosa, scalp, and nail didn't show any abnormality. Other systemic examination findings were unremarkable.

Our differential diagnoses were HSP with *H. pylori* infection and HSP associated with Coronavirus Disease 2019 (COVID-19) infection. Her baseline investigations including complete blood count, renal function test, liver function test, urine routine microscopy, bleeding profile, erythrocyte sedimentation rate, and C-reactive protein were within normal limits. Ultrasonography of the abdomen and pelvis revealed no abnormality. Reverse transcription-polymerase chain reaction (RT-PCR) for COVID-19 was also negative. Skin biopsy was taken from fresh lesion over the right leg and was sent for histopathological examination which revealed superficial perivascular and interstitial infiltrate with neutrophils, karyorrhexis, focal fibrinoid necrosis along with erythrocyte extravasation as shown in figure 2(A). The vessels involved were primarily postcapillary venules and endothelium were intact. Direct immunofluorescence (DIF) showed IgA positivity of 3+ along dermal vessels as shown in figure 2(B).

As our patient had recurrent and multiple episodes of gastritis for the last 6 months, we referred her to medicine OPD for further evaluation. Upper GI endoscopy was done which revealed multiple erosions with few small superficial ulcers in the antrum and first part of duodenum with an impression of gastro-duodenal ulcer. Biopsy taken from antrum for rapid urease test came out to be positive for *H. pylori*.

After confirmation of infection, she was started on eradication therapy for *H. pylori* from the medicine department comprising of Tab. Pantoprazole 40 mg, Tab. Clarithromycin 500 mg and Cap. Amoxicillin 1 gram twice daily for 2 weeks. For cutaneous lesions, antihistamines along with emollients were advised from our side. The patient was followed after 2 weeks which showed dramatic improvement in her skin lesion as shown in figure 3 along with improvement in gastric complaints. Patient was followed up for 2 months and there were no new lesions during follow-up.

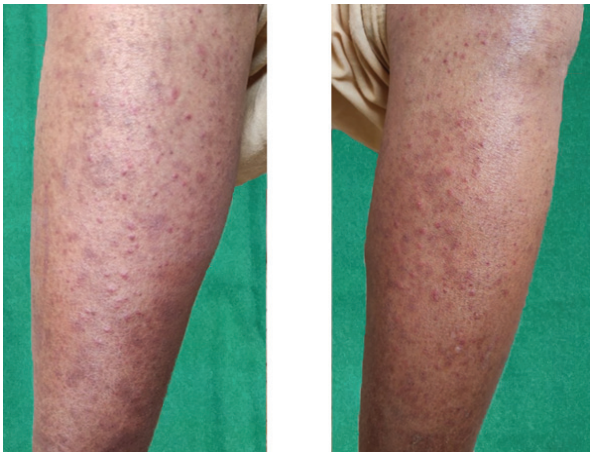

Figure 1: Multiple reddish-brown purpuric macules and papules present in a symmetrical manner over bilateral legs (A) Right leg (B) Left leg

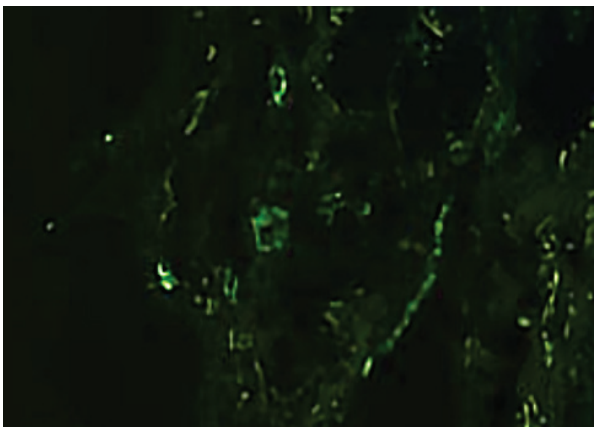

Figure 2(B): DIF showing perivascular IgA deposition along dermal vessels

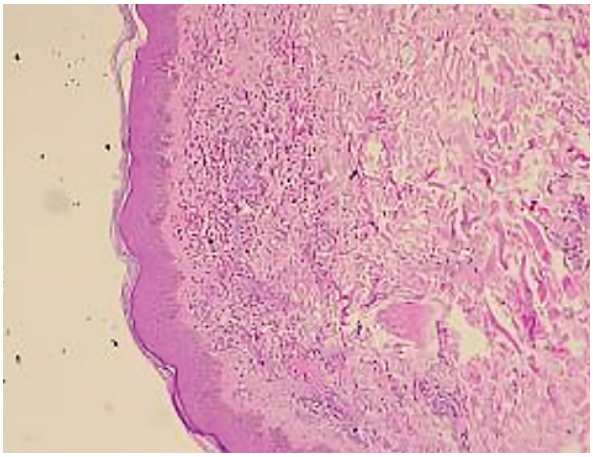

Figure 2(A): Low magnification view showing superficial perivascular and interstitial infiltrate of neutrophils along with extravasation of erythrocytes (10X magnification)

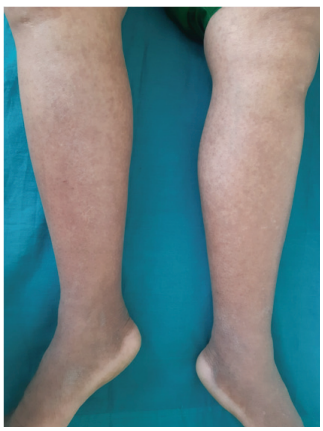

Figure 3: Resolution of skin lesions over bilateral legs at 2 weeks follow up period

Discussion

HSP is a leukocytoclastic vasculitis of small vessels characterized by IgA deposition in the affected tissues.[5] The diagnostic criteria for HSP include the presence of at least 2 of the following: palpable purpura, age younger than 20 years at the onset of symptoms, bowel angina, and a biopsy with granulocytes in the vascular wall (leukocytoclastic vasculitis).[6] In most cases, HSP is a self-limited condition. However, recurrent symptoms have been reported in one-third of patients.[3] HSP in adults represents a more severe clinical syndrome with a higher frequency of renal involvement.[7] Regarding GI involvement, 10% to 20% of patients in general present with abdominal complaints, while 85% have an abdominal complaint associated with other symptoms.[8] The most common GI symptoms in patients with HSP include colicky abdominal pain, nausea, vomiting, and anorexia; hematochezia can also occur, although less commonly.[6]

Although there are various causes for HSP, infectious agents are considered the most important etiological factors.[9] *H. pylori* is a spiral gram-negative, microaerophilic, and urease-positive bacterium, that colonizes the gastric mucosa.[3] *H. pylori*-induced gastric mucosal inflammation is responsible for several upper gastrointestinal disorders, including chronic gastritis, peptic ulcer disease, gastric mucosa-associated lymphoid tissue (MALT) lymphoma, and gastric cancer.[10] Besides this *H. pylori* has been implicated in certain extra-digestive dermatological conditions like chronic urticaria, rosacea, sweet syndrome, systemic sclerosis, atopic dermatitis including HSP.[11] In 66% of HSP-affected adults the initial presentation can be malignant lung, breast, prostate, and intestine carcinomas while the rest 33% may present with hematological malignancy such as lymphoma and multiple myeloma.[1]

The association between HSP and *H. pylori* infection was reported for the first time in 1955 by Reinauer et al. in a 21-year female by 13C-urea breath test in which purpura, intestinal symptoms, and albuminuria resolved after eradication treatment of *H. pylori*. However, the skin changes and gastric complaints recurred after a 10-month period which again resolved with retreatment.[12] Mytinger et al. reported *H. pylori* induced HSP in pediatric patients.[13] Likewise Xiong et al. in his meta-analysis reported the necessity of screening *H. pylori* infection in HSP children, particularly in those with gastrointestinal manifestations in China.[14]

The possible mechanism in the causation of cutaneous vasculitis by *H. pylori* is less understood. A strong inflammatory response is induced directly or indirectly by the presence of a more toxic strain of *H. pylori* which releases bacterial and host-dependent cytotoxic substances.[15] Besides this genetic predisposition of the individual, cross-reactivity of antibodies against *H. pylori* with some extragastric tissues, polymeric immunoglobulin A (pIgA), activated complements (C3 or C5), and certain fibrinogen/fibrin deposition in vessel walls, without IgG or IgM deposition are further responsible for disease pathogenesis. The virulence factors of *H. pylori* like vacuolating toxin gene A (vacA) and cagA further participate in the disease progression through an unknown mechanism.[1]

GI involvement in HSP is seen predominantly in the small bowel including the duodenum though the stomach can also be affected in a high percentage of cases.[3] The spectrum of endoscopic findings of HSP depends upon the severity of the vasculitis; usually, irregular, ulcerating, nodular lesions or hematoma-like protrusions are seen in the duodenum.[6] Gracia et al. have found ringed esophagus, esophageal mucosal erythema, gastric subepithelial hemorrhage, duodenal erythema with an ulcer on upper GI endoscopy.[2] Another study by Cecchi et al. have reported a scarring duodenal bulb with an active ulcer on the anterior aspect in esophagogastroduodenoscopy along with a positive rapid urease test for *H. pylori*.[16]

Due to the self-limited nature of HSP, patients are treated supportively or symptomatically and early treatment with steroids should be targeted at those patients who have a high risk of renal involvement or severe extrarenal symptoms. These risk factors are severe abdominal pain, age over six years, and renal symptoms at onset.[11] A study by Hoshino C has reported fading of purpuric skin lesion of HSP within 5 weeks of initiation of eradication therapy for *H. pylori* with no further treatment.[3] Aydi et al. have also reported similar findings of resolution of cutaneous lesion along with gastrointestinal complaints after *H. pylori* eradication therapy.[4] Similar result of dramatic improvement was also seen in our case when she was treated for *H. pylori* infection.

Conclusion

The association of *H. pylori* infection with HSP may be underlooked. We report a rare case that needed a multidisciplinary approach with speciality including dermatology, medicine, and pathologist for better management of both cutaneous and gastrointestinal manifestations. This study helps in raising awareness among dermatologists as well as treating physicians about the correlation between HSP and *H. pylori*

infection and also the importance of early referral of such cases. The chances of complications arising due to delay in diagnosis and treatment will be highly reduced resulting in ultimate benefits to both patients and treating physicians. Failure to correlate GI complaints with cutaneous manifestations like HSP may lead to persistence, recurrence, or deterioration of the preexisting condition. Future studies should be carried out to establish a relationship between *H. pylori* and HSP.

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