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Henoch schönlein purpura: A review

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Abstract
Henoch–Schönlein Purpura (HSP) is a disease that involves palpable purpura on the skin, joint pain, and gastrointestinal problems. An acute bacterial infection is one of the causes of HSP. Oral and dental condition may be the trigger cause of HSP attack. Odontogenic infectious diseases have been implicated in causing HSP. Only few reports describe the correlation between HSP and odontogenic infectious diseases. Diagnosis depends on clinical manifestations and no single diagnostic test can confirm the disease. Therefore, it is important for dental practitioner to be aware of the course of the disease in order to limit the expanding complications.

Keywords: Henoch-Schönlein purpura, skin rash, Vasculitis syndrome, Focal infection children

1. Introduction
It is a diffuse vasculitis that is secondary to hypersensitivity characterized by Ig a dominant immune complexes in smaller venules, capillaries and arterioles. The renal lesions are histopathologically indistinguishable from IgA nephropathy (Berger's disease). Both diseases can progress to renal insufficiency. [1]
The syndrome takes its name from two German physicians. In 1837, Johan Schönlein first described several cases of peliosis rheumatica or purpura associated with arthritis. [2] Thirty years later, Edwuard Henoch described the GI manifestations, including vomiting, abdominal pain, and melena. [3]

Etiology
The precise etiology of the disorder is unknown. It can occur in response to infectious agents such as group A streptococci, Mycoplasma, Epstein Barr virus, Varicella virus, Parvovirus B19 and *Campylobacter enteritis*. [4, 5] Cases have been reported following vaccinations for typhoid, measles, cholera and yellow fever. In addition, exposure to allergens in drugs or food, exposure to cold, and insect bites have been linked to the development of Henoch Schönlein purpura (HSP). [6]
Odontogenic focus infection (OFI) can trigger HSP attack and can be considered as one of the risk factors for HSP. OFI is a bacterial infection that tends to be overlooked by dermatologists. Dental screening of HSP patients could help to decrease the risk of renal and/or abdominal complications and facilitate treatment. [7] The concept that focal infection may produce chronic systemic diseases has now been generally accepted. Local, septic, or mucosal infections foci anywhere in the body can be sources of systemic diseases. To date, foci of specific infections of the gums and the presence of abscesses around the roots of the teeth, often unsuspected, have not received attention in the treatment of pediatric diseases. [8]
The etiological role of chronic oral infection in HSP is supported by several other studies. The antigens of the outer membranes of Haemophilus parainfluenzae, a common bacterium within apical periodontitis, and antibodies against these have been identified in the glomerular mesangium and sera of HSP and IgA nephropathy patients. [9] Given that IgA nephropathy and HSPN are pathologically identical diseases, all of these data suggest that chronic infections in the oral cavity may play pathogenic roles in HSP. The high caries levels of HSP children may support this view. Dental caries in premature teeth easily invade through infected root canals into surrounding bony tissues, forming apical periodontitis. [10] the most commonly identified OFI in HSP patients was apical periodontitis in association with dental caries.

Although both are infectious diseases by nature, apical periodontitis, which is mostly initiated from dental caries by oral bacteria invading through infected root canals, is a much more complex disorder in regard to infection as well as inflammation. A thousand billion bacteria colonize a single lesion, and more than 300 species of aerobic and anaerobic bacteria can be isolated. Within the associated lesions, various inflammatory cytokines are produced by cellular components of the periapical lesion, resulting in the persistence of active immune reactions. [10]

Many degraded bacterial products and the decomposition products of pulp tissue stagnate there. Meanwhile, bacteria and their toxic derivatives and destroyed peripheral tissues may egress through the apical foramen and be captured continuously within tonsils through their surface epithelium. [11] However, the innate secretory IgA-mediated oral mucosal defense system may fail to eliminate bacterial antigens owing to the presence of a tremendous amount of bacteria. On the other hand, bacterial pathogens may enter the blood stream during transient bacteremia, damaging the inside smooth lining of the blood vessel walls. Collectively, chronic and long-standing apical periodontitis have the potential to trigger HSP. [12]

Incidence

Based to ethnic groups, HSP has a higher prevalence in Caucasians and Asians than in those of African descent. and is the most common vasculiic syndrome in children. The disease occurs more often in the colder months. The peak incidence is in the 4–6 year-old age group with figures around 70/100 000 population. [12]

Clinical Feature

Purpura, arthritis and abdominal pain are known as the "classic triad" of Henoch–Schönlein purpura. Henoch-Schönlein purpura nephritis (HSPN) and gastrointestinal bleeding are also major clinical manifestations. [10] These can vary in the order that they present and develop over days to weeks. Palpable purpura and joint pain are the most common and consistent presenting symptoms. [13]

The classic rash of HSP begins as erythematous, urticarial and macular wheals. It then coalesces and develops into the typical ecchymoses, petechiae, and palpable purpura. The rash occurs in 96% cases, often manifests in a symmetrical pattern at pressure dependent areas, such as the lower extremities and the buttocks and cause edema. Face, trunk, and upper extremities may be more affected in non-ambulatory children. [13]

Joints are involved in the majority of cases, involving lower limbs (ankles and knees) more commonly. [14] Arthralgia occurs in 84% of HSP patients and often coexists with other symptoms. Pain, tenderness and restricted movement occurs. [13]

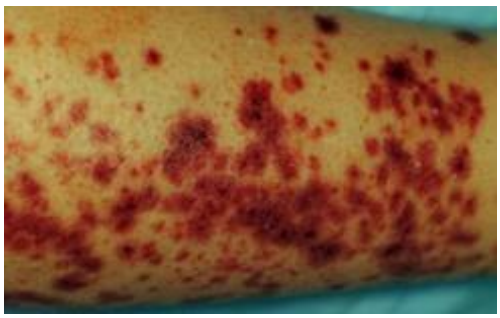

Fig 1: Showing typical purpuric rash of HSP

GI pain is often the most debilitating of the HSP symptoms, and can be further complicated by GIT hemorrhage (14–38%), intussusception, obstruction or perforation. [14] GI problems start as cramping abdominal pain, often with vomiting and appears about a week or more after the rash begins. Although there are cases where GI problems occur without a rash. 25% have GI bleed and 50% have occult blood loss. On endoscopy pupuric lesions +/– edema, ulceration, or bowel spasm can be seen. [12]

Renal symptoms have a wide range of severity, from asymptomatic microscopic hematuria, to full-blown nephritic syndrome or nephritis. Most renal involvement occurs early, 85% within the first month, although it can develop later; follow-up to 6 months is recommended. [14] Urine analysis can show proteinuria (mild), red blood cells, and cellular casts. Many patients will be asymptomatic, but others can develop nephritic syndrome. [8]

Other symptoms are rare and usually involve the central nervous system or lungs, from pulmonary hemorrhage to convulsions. Children younger than two years showing predominantly cutaneous symptoms and signs, as well as a much lower incidence of renal and gastrointestinal involvement. [12]

Complications of Henoch Schönlein purpura:-

- Hepatosplenomegaly
- Myocardial infarction
- Pulmonary hemorrhage
- Pleural effusion
- Unnecessary abdominal surgery
- Intussusception
- Hemorrhage
- Shock
- Gastrointestinal bleeding
- Bowel infarction
- Renal failure
- Hematuria
- Proteinuria
- Seizures

The diagnosis of Henoch Schönlein purpura depends on clinical findings and history. There is not a specific laboratory test for the disorder, although an elevated serum IgA level is suggestive. The complete blood count may reveal a normal or elevated white blood cell count and possible eosinophilia. Sedimentation rate and platelet count may be elevated. Electrolytes may be affected secondary to gastrointestinal involvement. Urinalysis may show hematuria. Renal manifestations may follow the development of the rash by up to three months; Therefore, urinalysis should be performed monthly, as well as measurements of blood urea nitrogen and creatinine levels in the presence of continued hematuria. A stool guaiac test may be positive. An underlying infectious etiologic agent should be excluded when clinically indicated. A normal platelet count differentiates Henoch Schönlein purpura from thrombocytopenic purpura. Skin biopsy may show a leukocytoclastic vasculitis. [15]

Selected Differential Diagnosis of HenochSchönlein [15]

- Purpura
- Acute abdomen
- Meningococcal meningitis or septicemia
- Rheumatoid arthritis
- Rheumatic fever
- Idiopathic thrombocytopenic purpura

- Systemic lupus erythematosus
- Child abuse
- Drug reactions
- Bacterial endocarditis
- Rocky Mountain spotted fever

Management

There is no specific treatment for Henoch Schönlein purpura. Bed rest and supportive care, such as assuring adequate hydration, are helpful. Non-steroidal anti-inflammatory drugs can relieve joint and soft tissue discomfort. Corticosteroids have some use in patients with severe abdominal pain. However, corticosteroids are not recommended for treatment of rash, joint pain or renal disease alone. Treatment with cyclophosphamide (Cytoxan, Neosar), plasmapheresis, cyclosporine (Neoral) and azathioprine (Imuran) is controversial. In the absence of renal disease and central nervous system involvement, the prognosis for patients with Henoch Schönlein purpura is excellent. The illness lasts four to six weeks in most patients. One half of patients have a recurrence. Long term follow up is necessary for patients with renal disease. The renal disease may not arise for several years. Renal biopsy may be performed to establish the diagnosis and determine the prognosis. Prognosis overall is excellent. The primary long term complication is renal disease, which develops in 5 percent of patients. One study suggests that corticosteroids and azathioprine may be helpful in treating renal disease once it develops. [16]

Discussion

HSP is considered to be associated with odontogenic infectious diseases as well. However, there is little evidence as to the causal relationships and efficacy of dental treatment in easing HSP. There are a few reports that mention the correlation between HSP and odontogenic infectious diseases. Jinous *et al*. have reported on a case of HSP that had developed after endodontic treatment. This report suggested that root canal treatment could be a trigger for HSP. [17] The authors assumed that trepanation of the apex may cause a streptococcal bacteremia and that the change of the environment and microbiological flora of the root canal may cause a bacteremia. Inoue *et al*. have reported on the efficacy of dental treatment in preventing nephropathy in pediatric HSP. In their study, children with HSP underwent antimicrobial and dental, ear, nose, and throat treatment. Almost all the patients were cured by the treatments. [10] Igawa *et al*. reported that an oral focal infection could be a precipitating factor for adult HSP. Half of the patients having adult HSP presented with an oral focal infection and underwent dental treatment, including tooth extraction. A few weeks after tooth extraction, improvements in the skin lesions were observed. [7]

In conclusion, dental screening of patients should be considered in HSP keeping in view the improvement shown by the patients after dental treatment.

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