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CASE STUDY

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A rare case of papular-purpuric "gloves and socks" syndrome associated with influenza

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Abstract

Papular-purpuric "gloves and socks" syndrome (PPGSS) is a unique, self-limited dermatosis characterized by edema, erythema, and pruritic petechiae and papules in a distinct "gloves and socks" distribution. This is often accompanied by systemic symptoms, including fever, lymphadenopathy, asthenia, myalgia, and arthralgias. PPGSS has also been described as a manifestation of an underlying immunological mechanism that can be triggered by viral or drug-related antigens. A 32-year-old male developed a painful eruption on the bilateral hands and feet after being diagnosed with influenza B. On examination, scattered papular purpura with occasional overlying scale was noted on the bilateral hands, fingers, feet, toes, volar wrists, and ankles. Histopathologic sections showed a mixed pattern of inflammation with interface and spongiotic changes. A parakeratotic scale with overlying basket-weave orthokeratosis was also seen. Within the epidermis, there was intraepidermal vesicles and Langerhans cell microabscess formation with scattered apoptotic keratinocytes. The underlying dermis showed a superficial perivascular lymphocytic infiltrate with mild edematous changes, and extravasation of red blood cells. Clinicopathologic correlation strongly supported a diagnosis of popular-purpuric gloves and socks syndrome. The influenza virus has never been reported in association with PPGSS; thus, this case outlines an important new variant that clinicians should be familiar with.

KEYWORDS
gloves and socks syndrome, papular-purpuric "gloves and socks" syndrome, PPGSS

1 | INTRODUCTION

Papular-purpuric "gloves and socks" syndrome (PPGSS) is a distinctive, self-limited dermatosis characterized by pruritic edema and erythema with petechiae and papules in a distinct "gloves and socks" distribution.[1] Classically, it manifests because of an underlying immunologic mechanism induced by viruses, most commonly parvovirus-B19.[2] However, the influenza virus has never been reported in association with PPGSS. In this report, we present a case of a 32-year-old male who developed PPGSS following the diagnosis of influenza.

2 | CASE PRESENTATION

A 32-year-old male without a relevant past medical history presented to the Dermatology clinic with a 2-week history of tender papules that began on his bilateral wrists. Shortly after, they progressed to

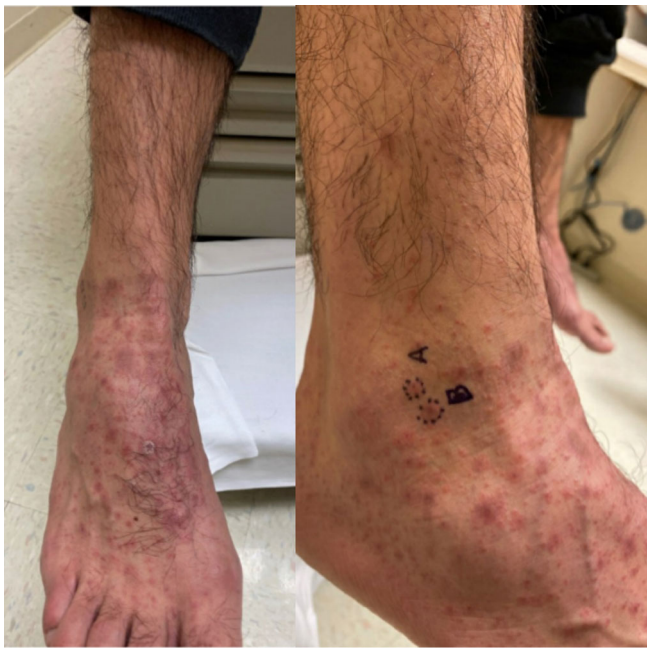

FIGURE 1 Photographs show the scattered papular purpura present on the patient's feet. His hands, wrists, and ankles were also involved with similar lesions. Letters A and B in the figure were the exact sites the biopsies were performed

involve both of his feet, hands, wrists, and ankles. Approximately 1 week prior to the appearance of the rash, the patient presented to the emergency department with fever, chills, rhinorrhea, headache, and myalgias for 5 days. The patient was found to be febrile (100.2 °F), and was diagnosed with influenza B by a point-of-care test (sensitivity: 93.2%, specificity: >97%).[3] No other lab abnormalities were identified and no other viral etiologies were considered. However, a full viral workup was performed, and polymerase chain reaction (PCR) testing for parvovirus B19 was negative.[4] Notably, this patient presentation was pre-Covid-19. He was not given oseltamivir, as he was deemed outside the window of benefit and was only treated symptomatically with anti-pyretics and NSAIDs, and discharged from the emergency department.

In his history and review of symptoms, he denied any oral or ocular involvement, throat closure, lip swelling, difficulty breathing, arthritis, or any history of allergies, eczema, psoriasis, or drug rashes in the past. His only current medications were acetaminophen and a multivitamin. On physical exam, scattered papular purpura with a few lesions having an overlying scale were noted on the bilateral hands, fingers, feet, toes, volar wrists, and ankles (Figure 1). The remainder of the physical exam was unremarkable. Subsequent punch biopsies were performed from the patient's right lateral malleolus, one for hematoxylin and eosin staining, and the other for direct immunofluorescence studies.

Histopathologic examination (Figure 2) revealed a mixed pattern of inflammation, spongiosis, and overlying basket-weave orthokeratosis and parakeratosis. Within the epidermis, there was

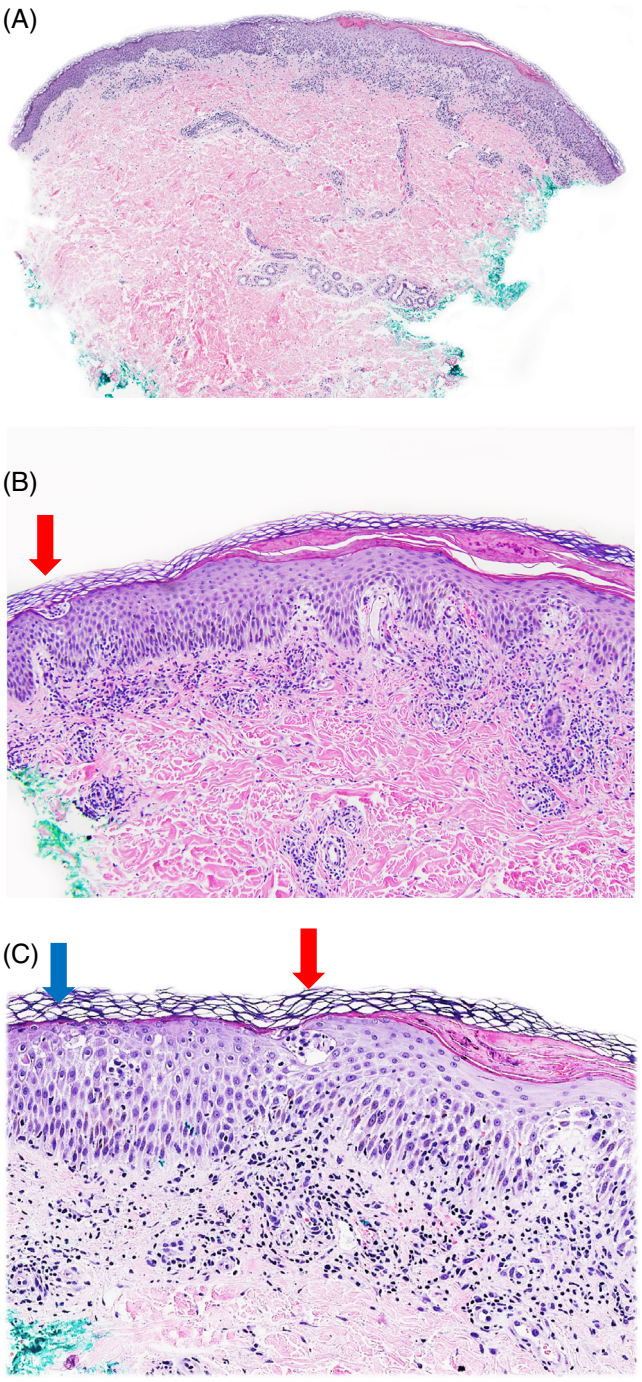

FIGURE 2 At low power, a punch biopsy of the right lateral malleolus demonstrates a mixed interface and spongiotic dermatitis with accompanying perivascular lymphocytic inflammation (A, H&E, ×20). Higher-power views better highlight the parakeratosis and serum deposition with mild underlying epidermal spongiotic change and vesicle formation. The higher-power view also demonstrates Langerhans cell microabscess formation (highlighted by red arrow). Within the epidermis, there is a prominent basovacuolar change with rare apoptotic keratinocytes (highlighted by blue arrow). The underlying dermis shows a superficial lichenoid and perivascular lymphocytic infiltrate with mild edematous changes and extravasation of red blood cells (B,C, H&E, ×100, ×200)

intraepidermal vesicle and Langerhans cell microabscess formation with scattered apoptotic keratinocytes. The underlying dermis showed a superficial perivascular lymphocytic infiltrate with mild edematous changes, and extravasation of red blood cells. Special stain periodic acid-Schiff was performed on the histopathologic sections and was negative for fungal organisms. Direct immunofluorescence studies were performed, which showed a negative or non-diagnostic staining pattern. At a follow-up appointment 3 weeks after the initial appointment, and the patient's lesions were resolving as expected. In a subsequent appointment, the lesions were fully resolved without any pigmentation or scarring.

3 | DISCUSSION

PPGSS is an acral dermatosis first described by Harms et al in 1990 as, "pruritic and painful edema and erythema with subsequent acral petechiae localized to the distal upper and lower extremities as a gloves and socks distribution." It is most prevalent in young adults,[1] but childhood onset cases have been described as well.[2,5] PPGSS presents initially with edema and erythema of the dorsal and volar surfaces of the distal extremities (hands and feet). The lesions are sharply marginated at the wrists and the ankles and evolve to the formation of erythematopapular and purpuric lesions at these same sites. PPGSS may also be associated with oral lesions and constitutional symptoms including fever, arthralgia, headaches, and generalized lymphadenopathy. These symptoms may precede or accompany the skin features. However, these constitutional symptoms are more probably related to the underlying viral etiologies, rather than a direct symptom of PPGSS.[6-8] Clinical differential diagnostic considerations include (but are not limited to) Kawasaki disease, hand-foot-mouth disease, and erythema multiforme.

Parvovirus-B19 has been implicated as the major causative agent of this syndrome. Gutermuth et al identified 70 cases in the literature, and 66% of them were associated with a florid parvovirus-B19 infection. However, other viruses have been found to have an association, including cocksackie, rubella, hepatitis B, varicella zoster, cytomegalovirus, and Epstein-Barr.[9] Since the first description of PPGSS in 1990, there have been no other reports in the literature of PPGSS having an association with the influenza virus, as was seen in this case. The influenza virus, typically responsible for seasonal epidemics, is an enveloped, single-stranded RNA virus that is a part of the orthomyxovirus family. It typically presents with systemic symptoms including fevers and chills, myalgias and malaise, and nausea and vomiting. When clinical suspicion is high, screening for the influenza virus is performed with a rapid antigen detection test. While a positive test is very specific, a negative test should be followed up by a reverse transcription PCR detection method for definitive diagnosis.[10] Besides viral-induced PPGSS, cases of adverse drug reactions/drug-induced PPGSS have also been reported.[11]

Our patient presented with a typical clinical picture associated with the syndrome as well as characteristic histopathological findings. However, there was no clinical concern for any of the other more common viral etiologies, including parvovirus-B19. The patient did not have any known contact with any individuals infected (or suspected of infection) with parvovirus-B19. But a complete clinical workup was performed to rule out any other possible causes.

The histopathologic presentation of PPGSS has been classically described to be on a spectrum, with differing early- and late-stage findings. Smith et al describe the most common histopathologic findings associated with PPGSS. They described two phases of histopathologic changes, described as early and late phases. Early changes were seen within 1 week of the onset of rash, and late changes seen greater than 1 week. In the early stages of PPGSS, the histopathologic findings are very broad; thus, it can be very difficult to distinguish it from other common inflammatory dermatologic conditions. Some of the more common early phase findings are: superficial perivascular lymphocytes, papillary dermal edema, spongiotic changes, and mild acanthosis. There are also histopathologic findings common to both the early and late stages of PPGSS, most notably a superficial perivascular lymphocytic infiltrate. However, as the lesion progresses, there is a more robust vacuolar interface change with apoptotic keratinocytes, papillary dermal edema, and extravasation of red blood cells. Once these findings are seen, this is characteristic of the fully evolved form/late findings of the dermatosis.[12] This description is compatible with our patient's histopathologic findings, who presented over 1 week after the onset of the rash. Typical histopathologic findings seen in PPGSS are shown in Table 1.

The differential diagnoses to be considered include an eczematous dermatitis, syphilis, lichenoid drug reaction, and pityriasis lichenoides et varioliformis acute (PLEVA/Mucha-Habermann disease). Although spongiosis was seen in our case, the presence of interface change with scattered apoptotic keratinocytes and extravasated red blood cells/dermal hemorrhage did not fit a diagnosis of eczematous dermatitis. Syphilis can also present with overlapping features seen in our case, including a dense lichenoid reaction and epidermal apoptosis; however, the lack of plasma cells, superficial neutrophils, and negative laboratory workup led to the exclusion of this diagnosis. A lichenoid drug reaction is another important differential diagnosis because of the dense lymphocytic infiltrate and vacuolar change seen at the dermoepidermal junction. Lichenoid drug reactions can also show apoptotic keratinocytes, which were seen in our case. But given the lack of eosinophils and the patient's lack of medication use, this diagnosis was deemed less likely. PLEVA, in particular, also has many overlapping histopathologic findings and has even been directly linked to parvovirus B19,[10] which makes it an important differential diagnosis to consider in any case of PPGSS. However, the overall clinical presentation of PLEVA (precursor of edema and erythema, localization of the lesions to only distal extremities, and lack of varying ages of the lesions) helped us to rule out that diagnosis as well.[14]

In healthy individuals, the symptoms of PPGSS are typically mild and self-resolving, usually within 1 to 2 weeks without relapses. Thus, the treatment of PPGSS typically includes bed rest, NSAIDs, and topical steroids. However, in patients who are immunosuppressed, PPGSS can have more serious complications including chronic anemia, pancytopenia, and persistent cutaneous lesions, which would involve a higher level of treatment.[15,16]

TABLE 1 Previously reported cases of papular-purpuric "gloves and socks" syndrome with histopathologic findings

Year	Author	Day of biopsy	Histopathologic findings
1990	Harms et al[1]	Day 5	Mild acanthosis and superficial perivascular lymphocytes
1992	Halasz[17]	Day 7	"Hemorrhagic dermatitis"
1994	Trattner[18]	Day 7	Vacuolar interface change, papillary dermal edema, lymphocytic infiltrate, and dermal hemorrhage
1994	Puig[19]	Day 3	Perivascular lymphocytes and dermal hemorrhage
1995	Morrell[20]	Day unknown	Perivascular lymphocytes, eosinophils, and papillary dermal edema
1995	Carrascosa[21]	Day 4	Superficial perivascular lymphocytes
1996	Aractingi[22]	Day unknown	Lichenoid and vacuolar interface change, perivascular lymphocytes, dermal hemorrhage, and dyskeratotic basilar keratinocytes
1996	Aractingi[22]	Day unknown	Perivascular lymphocytes
1996	Vargas-Diaz[23]	Day 2	Acute and chronic superficial inflammation and mild acanthosis
1996	Vargas-Diaz[23]	Day 5	Perivascular lymphocytes, vacuolar interface change, parakeratosis, and dermal hemorrhage
1996	Vargas-Diaz[23]	Day 5	Mild perivascular lymphocytes, mild vacuolar interface change, papillary dermal edema, and dermal hemorrhage
1998	Ruzicka et al[13]	Day 3	Perivascular and interstitial infiltrate with lymphocytes, neutrophils, and eosinophils, papillary and reticular dermal edema, and mild acanthosis
1998	Leahy[24]	Day 5	Mild perivascular lymphocytes and dermal hemorrhage
1998	Larralde[25]	Day 1	Perivascular lymphocytes, spongiotic changes, papillary dermal edema, and dyskeratotic keratinocytes
1999	Grilli[26]	Day 4	Perivascular lymphocytes, spongiotic changes, vacuolar interface changes, and dermal hemorrhage
2000	Segui[27]	Day 5	Perivascular lymphocytes, papillary dermal edema, and dermal hemorrhage
2002	Smith et al[12]	Day 2	Perivascular lymphocytes
2002	Smith et al[12]	Day 7	Superficial and deep lymphocytic infiltrate, vacuolar interface change, dyskeratotic keratinocytes, and papillary dermal hemorrhage
2002	Higashi[28]	Day 2	Lymphocytic dermal inflammation and extravasation of red blood cells (RBCs)/dermal hemorrhage
2003	Alfadley[29]	Day 4	Perivascular lymphocytic infiltrate, papillary dermal edema, vacuolar interface changes, and extravasated RBCs/dermal hemorrhage
2004	Sklavounou-Andrikopopoulou[30]	Day unknown	Leukocytoclastic vasculitis
2005	Loukeris[31]	Day 5	Fibrinogen in vessel walls and nuclear dust
2006	Aguilar-Bernier[32]	Day 2	Perivascular lymphocytic infiltrate, vacuolar interface changes, interstitial histiocytic infiltrate, RBC extravasation, necrotic keratinocytes, fibrin deposition in vessels, and lymphohystiocytic perineural infiltrate
2008	Troeger[33]	Day 21	Perivascular lymphocytic infiltrate, and vacuolar interface changes
2011	Santoja[34]	Day Unknown	Lymphocytic perivascular infiltrate and RBC extravasation
2011	Santoja[34]	Day Unknown	Lymphocytic perivascular infiltrate, exocytosis of lymphocytes into epidermis, scattered interstitial eosinophils, focal features of vasculitis, and apoptotic keratinocytes
2011	Gutermuth et al[9]	Day Unknown	Dermal lymphocytic inflammation, sub-epidermal edema, intraepidermal cleft formation, acanthosis, necrotic keratinocytes, and extravasated RBCs
2015	Ohshita[35]	Day 4	Mild perivascular lymphocytic infiltrate with scattered neutrophils and dermal edema
2017	Vazquez-Osorio[36]	Day unknown	Superficial and deep perivascular lymphohistiocytic infiltrate, lymphocytic exocytosis, papillary dermal edema, and extravasated RBCs
2018	Phuan[37]	Day 14	Spongiotic dermatitis with eosinophils and extravasated RBCs
2019	Tidman and Fatima[38]	Day unknown	Superficial chronic inflammation with eosinophils and papillary dermal edema
2019	Bou-Prieto[39]	Day 1	Superficial perivascular mixed infiltrate with lymphocytes and eosinophils and dermal hemorrhage

Overall, PPGS, although rare in both children and adults, should be considered in cases of rapidly onset painful and/or pruritic lesions on the hands and feet. It is imperative that we raise awareness of this unique clinical presentation of PPGSS associated with influenza, as well as the classic histopathologic findings, as it will assist in establishing the diagnosis and guiding appropriate management.

CONFLICT OF INTEREST

The authors declared no conflict of interest.

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