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CASE REPORT

Oral manifestations of papular-purpuric 'gloves and socks' syndrome due to parvovirus B19 infection: the first case presented in Greece and review of the literature

A Sklavounou-Andrikopoulou[1], M Iakovou[1], S Paikos[2], V Papanikolaou[3], D Loukeris[4], M Voulgarelis[4]

[1]*Department of Oral Pathology and Medicine, School of Dentistry, University of Athens, Athens;* [2]*Dental Department, 'LAIKON' General Hospital, Athens, Greece;* [3]*Department of ENT, St John Hospital, Mid Essex, UK;* [4]*Department of Pathophysiology, School of Medicine, University of Athens, Athens, Greece*

Papular-purpuric 'gloves and socks' syndrome (PPGSS) is a novel, rare, self-limited dermatosis initially described in 1990. It is characterized by painful, pruritic edema and erythema, rapidly evolving to papular-purpuric lesions on the distal extremities, in a gloves-and-socks distribution, accompanied by fever and oral lesions such as petechiae, vesiculopustules and small erosions. Parvovirus B19 has been implicated in most cases as the etiological factor. Herein we present the first case of PPGSS in a 42-year-old Greek man with von Willebrand disease. On admission the patient was febrile, and presented acral edema and erythema rapidly followed by purpuric lesions on the same sites, and palatal petechiae. Complete remission of the exanthem occurred 7 days after hospitalization. Clinical and laboratory evaluation including serologic tests and PCR, confirmed the presence of parvovirus B19. Review of the existing literature on this novel syndrome and its association with parvovirus B19 is also presented. *Oral Diseases* (2004) **10**, 118–122

Keywords: papular-purpuric 'gloves and socks' syndrome; parvovirus B19; acral dermatosis; oral mucosa

Introduction

In 1990, Harms, Feldmann and Saurat, observed five, young, otherwise healthy persons, who developed an acral acute papular purpuric dermatosis of unknown etiology. The dermatosis was characterized by pruritic edema and erythema followed by petechial purpura and was localized on the hands and feet in a gloves-and-socks distribution, accompanied by fever and oral lesions. They named the entity papular-purpuric 'gloves and socks' syndrome, and they suggested that it might be of infectious origin (Harms *et al*, 1990).

The syndrome occurs mainly in young adults between 20–40 years and equally affects men and women, during the spring and summer seasons (Borradori *et al*, 1994; Veraldi *et al*, 1996). There have also been reports of pediatric cases (Stone and Murph, 1993; Labbé *et al*, 1994; Morell *et al*, 1995; Larralde *et al*, 1998; Ongrádi *et al*, 2000; Petter, Rytter and Haustein, 2001). The rash is generally self-limited and clears within a period of 1 or 2 weeks with possible desquamation of the involved areas (Harms *et al*, 1990; Veraldi *et al*, 1996; Smith *et al*, 1998). Patients usually require symptomatic treatment while cases of recurrences have not been reported (Veraldi *et al*, 1996; Smith *et al*, 1998).

Patients' laboratory findings reveal mild anemia, a decrease in leukocyte and platelet counts and transient elevation of liver function (Harms *et al*, 1990; Halasz, Cormier and Den, 1992). The histopathologic findings from skin biopsies are non-specific (Harms *et al*, 1990).

To date the pathogenesis of the syndrome remains insufficient. Bagot and Revuz, were the first to report an association between the 'gloves and socks' syndrome and parvovirus B19 (B19V) (Bagot and Revuz, 1991). Since then several reports provided evidence to associate more than one half of the reported cases with B19V (Harms *et al*, 1990; Bagot and Revuz, 1991; Harms, Feldmann and Saurat, 1991, 1994; Evans, Grossman and Gregory, 1992; Halasz *et al*, 1992; Stone and Murph, 1993; Borradori *et al*, 1994; Feldman, Harms and Saurat, 1994; Labbé *et al*, 1994; Pérez-Ferriols, Martinez-Aparicio and Aliaga-Boniche, 1994; Puig *et al*, 1994; Trattner and David, 1994; Veraldi and Rizzitelli, 1994, 1995; Carrascosa *et al*, 1995; Morell *et al*, 1995; Aractingi *et al*, 1996; Gaston and Zurowski, 1996; Guibal *et al*, 1996; Menedez *et al*, 1996; Vargas-Diez *et al*, 1996; Veraldi *et al*, 1996; Drago, Parodi and Rebora, 1997; Larralde *et al*, 1998; Ruzicka

Correspondence: Sklavounou-Andrikopoulou Alexandra, 5 Sismanoglou Street, P. Psychico 154-52, Athens, Greece. Tel: 0030-210-7701341, 0030-210-6741930, Fax: 0030-210-7795600, E-mail: asklavou@dent.uoa.gr
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et al, 1998; Smith *et al*, 1998; Grilli *et al*, 1999; Van Rooijen, Brand and Ballmer-W, 1999; Ghigliotti *et al*, 2000; Ongrádi *et al*, 2000; Seguí *et al*, 2000; Passoni *et al*, 2001; Petter *et al*, 2001; Velez, Fernandez-de-la-Puebla and Moreno, 2001; Higashi *et al*, 2002). Herein we present another typical case of B19V-associated PPGSS in an adult Greek male, which is the first report in Greece. We also attempt a review of the existing literature on PPGSS and it's relation to B19V.

Case report

In February 2002, a 42-year-old white man, known to suffer from Von Willebrand's disease, was admitted to the Pathophysiology Department of 'LAIKON' General Hospital, with a 2-days history of pruritic erythema on the hands and feet, which rapidly evolved into acral purpura. One-day prior to admission, he experienced fever and chills, while the rash expanded on the groins, the elbows and the armpits.

On admission he was febrile (39.5°C). Results of a physical examination showed a confluent purpuric-petechial rash involving the dorsal and palmar surfaces of the distal extremities in a symmetrical gloves-and-socks distribution (Figure 1). The rash expanded on the groins, the elbows and the armpits. Multiple petechiae were present on the hard and soft palate (Figure 2). He also presented erythema on the dorsal surface of the nose, the cheeks and forehead. No enlargement of peripheral lymph nodes, liver or spleen was palpable. He had 110 pulses/min and his blood pressure was 110/90 mmHg.

Laboratory tests revealed normal values. Von Willebrand's disease was under control; factor's VIII activity was normal. A skin biopsy specimen showed no specific findings but there was evidence of leukocytoclastic vasculitis. Serology for CMV, EBV, HIV1, HIV2, HBV, HCV, HSV1, HSV2, Coxsackie viruses, and rickettsiosis were negative. At this time serum samples were also negative for anti-B19V antibodies.

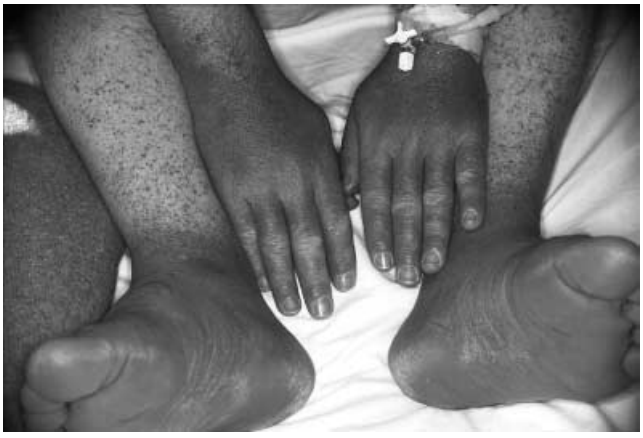

Figure 1 Confluent purpuric-petechial rash involving the dorsal and palmar surfaces of the distal extremities in a gloves-and-socks distribution

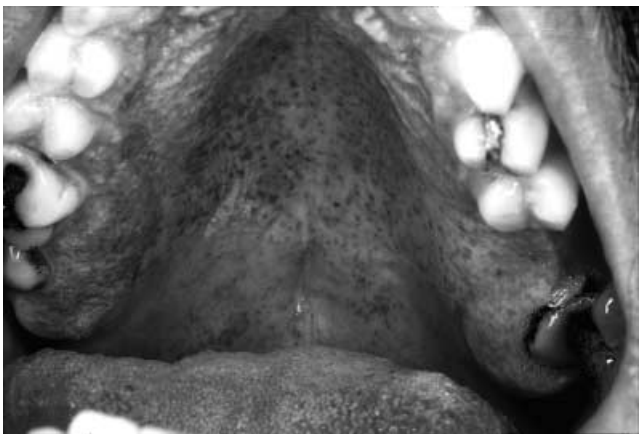

Figure 2 Multiple petechiae on the hard and soft palate

On the fourth day of admission the patient's hematocrit dropped from 41.8 to 35.2%. A myelogram showed total red cell aplasia and characteristic giant erythroblasts indicative of a viral infection. At the same time, B19V DNA was detected following PCR amplification of target sequences in skin lesion, whole blood and serum, using a VP1-specific primer set, complimentary to nucleotides 3222–3240 and 3459–3480, that amplifies a 258 bps sequence of the viral DNA (Figure 3).

No specific treatment was instituted and the patient gradually improved. The next day he was afebrile and the rash was markedly decreased. On the seventh day of admission, the patient was discharged in a good condition, with complete regression of the rash. All laboratory test results were normal.

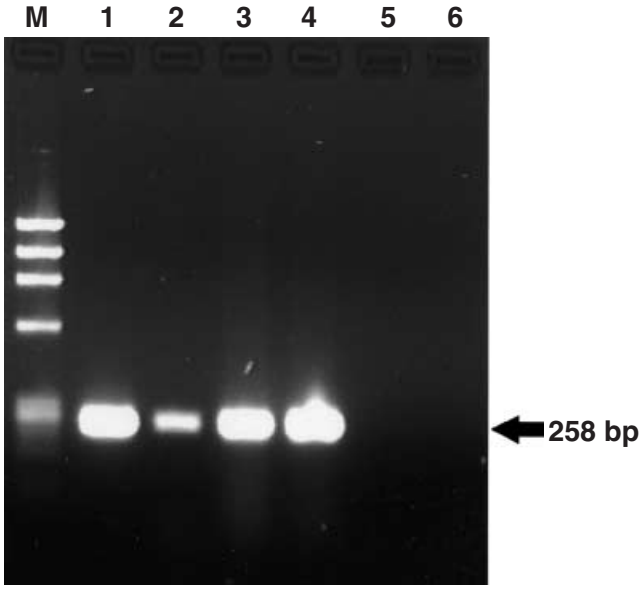

Figure 3 Polymerase chain reaction detection of B19V DNA in whole blood (lane 1), serum (lane 2) and skin lesion (lane 3). One positive and one negative control are used in lanes 4 and 5, retrospectively, as well as an unknown negative sample (lane 6). In lane M, molecular weight marker ØF 174 RF DNA/*Hae*III digest

Two months later, when the patient returned for follow-up, serologic tests showed positivity for antiparvovirus B19 IgM and IgG, supporting the initial diagnosis established by the serum PCR analysis.

Discussion

The papular-purpuric 'gloves and socks' syndrome is quite rare. Since the first description in 1990 (Harms *et al*, 1990), more than 50 cases have been reported in the literature, most of them in Europe (Harms *et al*, 1990, 1991, 1994; Bagot and Revuz, 1991; Evans *et al*, 1992; Halasz *et al*, 1992; Stone and Murph, 1993; Borradori *et al*, 1994; Feldman *et al*, 1994; Labbé *et al*, 1994; Pérez-Ferriols *et al*, 1994; Puig *et al*, 1994; Trattner and David, 1994; Veraldi and Rizzitelli, 1994, 1995; Carrascosa *et al*, 1995; Morell *et al*, 1995; Aractingi *et al*, 1996; Gaston and Zurowski, 1996; Guibal *et al*, 1996; Menedez *et al*, 1996; Vargas-Diez *et al*, 1996; Veraldi *et al*, 1996; Drago *et al*, 1997; Larralde *et al*, 1998; Ruzicka *et al*, 1998; Smith *et al*, 1998; Grilli *et al*, 1999; Van Rooijen *et al*, 1999; Ghigliotti *et al*, 2000; Ongrádi *et al*, 2000; Seguí *et al*, 2000; Passoni *et al*, 2001; Petter *et al*, 2001; Velez *et al*, 2001; Higashi *et al*, 2002).

The syndrome is characterized initially by edema and erythema of the dorsal and palmar surfaces of the distal extremities with sharp margins at the wrisks and ankles, followed by pruritic and sometimes painful erythematopapular and purpuric lesions, of a few millimeters in diameter, symmetrically localized on the same sites (Puig *et al*, 1994; Veraldi *et al*, 1996; Smith *et al*, 1998). Similar lesions occur less frequently on other sites of the body such as the cheeks, elbows, knees, trunk, buttocks and the inner aspects of the thighs (Harms *et al*, 1990). Oral lesions and general symptoms such as fever, asthenia, anorexia, arthralgia, myalgia and lymphadenopathy usually accompany the purpuric exanthema of the hands and feet (Veraldi *et al*, 1996; Smith *et al*, 1998; Grilli *et al*, 1999). Harms *et al* (1990), also reported one case of PPGSS accompanied by conjunctivitis.

Various oral mucosa manifestations have been described including multiple petechiae of a few millimeters in diameter on both the hard and soft palate and the buccal mucosal, which mimic oral manifestations of blood disorders. Erythema, vesicles, pustules, small erosions and shallow ulcerations on both the hard and soft palate and the buccal mucosa, erythema and swelling of the lips, aphthous ulcers on the labial mucosa, angular cheilitis, sore throat and tongue, erythema of the pharynx and pharyngitis, even Köplik spots have also been reported (Harms *et al*, 1990; Bagot and Revuz, 1991; Evans *et al*, 1992; Halasz *et al*, 1992; Puig *et al*, 1994; Veraldi *et al*, 1996; Smith *et al*, 1998). The lesions are usually painful and they appear in more than one half of the so far presented cases (Smith *et al*, 1998). The genital mucosa may also be affected with painful edema, erythema and sometimes small ulcerations of the glans penis and vagina (Harms *et al*, 1990; Grilli *et al*, 1999).

Grilli *et al* (1999), reported some additional clinical features, which had not been previously described, namely dysuria with vulvar edema and erythema, and unilateral petechial rash on the breast. Passoni *et al* (2001), also reported atypical clinical features of PPGSS. Their patient presented hemorrhagic bullae associated with purpuric lesions on the feet that progressed into cutaneous necrosis with superficial ulcerations. Healing with thick black eschars had not been previously described in PPGSS. Higashi *et al* (2002), in a recent study described a case with several bloody bullae particularly on the toes. They could not however determine, whether mechanical pressure or PPGSS had caused the formation of the bullae.

Our patient presented the classic triad of manifestations including fever, edema and erythema of the hands and feet, which rapidly evolved into pruritic confluent purpuric-petechial rash and expanded on the groins, elbows and armpits. Finally, multiple petechiae on both the hard and soft palate were noticed. It is noteworthy that our patient revealed unconjucated hyperbilirubinemia at presentation, a finding that has been reported in another case by Passoni *et al* (2001).

Histopathologic findings from skin biopsies taken in a few reported cases were non-specific. They included epidermal lymphocytic exocytosis, perivascular lymphocytic infiltrate, extravasation of erythrocytes, edema of the dermis, slide acanthosis, necrosis and vacuolization of the basal keratinokytes with a lichenoid reaction (Harms *et al*, 1990; Trattner and David, 1994; Aractingi *et al*, 1996; Grilli *et al*, 1999). In our case, the skin biopsy specimen showed some findings of leukocytoclastic vasculitis. Menedez *et al* (1996), also reported a case of B19V-induced PPGSS with leukocytoclastic vasculitis.

Harms *et al*, suggested that PPGSS could be of infectious origin and most of the published reports so far implicate a B19V-infection (Harms *et al*, 1990; Bagot and Revuz, 1991; Harms *et al*, 1991; Evans *et al*, 1992; Labbé *et al*, 1994; Puig *et al*, 1994; Veraldi and Rizzitelli, 1995; Larralde *et al*, 1998; Smith *et al*, 1998; Grilli *et al*, 1999; Ghigliotti *et al*, 2000; Passoni *et al*, 2001).

Parvovirus B19 is a small, non-enveloped, single stranded DNA virus, member of the erythrovirus family, and the only parvovirus known to cause disease in humans (Katta, 2002). The virus's receptor is present on the bone marrow erythroid precursor cells. *In vitro* studies have shown the lytic B19V infection of erythroid precursor cells (Anderson *et al*, 1985; Guibal *et al*, 1996; Sabella and Goldfarb, 1999). The infection is transmitted via the respiratory tract (Anderson *et al*, 1985; Sabella and Goldfarb, 1999).

The virus was firstly discovered in 1975, in the serum of healthy blood donors (Cossart *et al*, 1975). Infection with B19V is usually brief and self-limited in healthy individuals but in cases of immunosuppressed patients or patients with hematologic disorders, the infection becomes chronic and persistent. Many clinical entities have been strongly associated with infections caused by B19V such as erythema infectiosum (fifth disease-'slapped cheeks'), transient aplastic crisis, arthralgias,

rheumatoid-like arthritis, asymptomatic infection, abortions and hydrops fetalis, chronic anemia-bone marrow suppression in immunocompromized hosts, and lately B19V-induced PPGSS (Garcia-Tapia *et al*, 1995; Veraldi and Rizzitelli, 1995; Sabella and Goldfarb, 1999; Brown *et al*, 2001; Katta, 2002).

Anderson *et al* (1985), studied the results of the B19V infection by inoculating the virus intranasally, in human volunteers. One or 2 weeks after inoculation, an intense viremia developed followed by the production of IgM (10–12 days after exposure) and IgG (approximately 2 weeks after exposure) antibodies.

Patients, who are unable to mount an immune response to the virus, are left with a continuous destruction of their erythroid precursor cells leading to a chronic transfusion-depended anemic state (Sharma *et al*, 2000). Ghigliotti *et al* (2000), reported three cases of PPGSS as a result of B19V infection in HIV-positive women. Because of inadequate immune response, one of them had prolonged cutaneous lesions and pruritus, and the other two had persistent anemia.

Bagot and Revuz (1991), were the first investigators to link PPGSS with B19V infection after performing viral serologic tests, in a young woman with PPGSS. Since then the association of PPGSS with this virus was demonstrated by seroconversion in many cases (Evans *et al*, 1992; Puig *et al*, 1994; Veraldi and Rizzitelli, 1995; Aractingi *et al*, 1996; Smith *et al*, 1998; Grilli *et al*, 1999; Passoni *et al*, 2001). It is important to note that in many B19V-associated PPGSS, specific IgM antibodies have not been demonstrated in the serum at the time of the initial presentation. This supports the hypothesis that the development of mucocutaneous lesions in PPGSS parallels viremia (Smith *et al*, 1998; Passoni *et al*, 2001; Katta, 2002).

B19V-DNA can also be identified by PCR in the patients' sera and in cutaneous biopsy specimens (Aractingi *et al*, 1996; Grilli *et al*, 1999). Similarly in our case, the use of PCR helped to establish the diagnosis of a B19V-associated PPGSS, by demonstrating B19V-DNA in the patient's skin biopsy specimen, serum and whole blood. Two months after illness, during the patient's follow-up period, serologic tests showed positivity for both antiparvovirus B19 IgM and IgG confirming the initial diagnosis.

The use of viral-serologic tests implicated other viruses as causative agents for PPGSS, including measles virus in two cases (Pérez-Ferriols *et al*, 1994; Veraldi *et al*, 1996), Epstein–Barr virus in one case (Drago *et al*, 1997), human herpesvirus type 6 in one case (Ruzicka *et al*, 1998), simultaneous infection by human herpesvirus type 7 and B19V in one case (Ongrádi *et al*, 2000), rubella virus in one case (Seguí *et al*, 2000), cytomegalovirus in one case (Carrascosa *et al*, 1995) and hepatitis B virus in two cases (Guibal *et al*, 1996; Velez *et al*, 2001). Finally, there is one report in which complement fixation demonstrated the causative role of coxsackie B6 virus (Feldman *et al*, 1994), and another in which a throat swab cultured on 5% human blood agar yielded *Arcanobacterium haemolyticum* (Gaston and Zurowski, 1996). Drugs such as trimethoprim/sulfamethoxazole have also been proposed as PPGSS-causative agents (Van Rooijen *et al*, 1999). In the remaining cases, the results of serologic tests for B19V were either negative or not performed.

The pathogenesis of this syndrome is still not completely understood. Aractingi *et al* (1996), performed an immunohistochemical study with specific anti-B19V antibodies in skin biopsies and showed the presence of viral antigens on the endothelial cells of the dermal vessel walls, on the epithelial cells of the sweat glands and ducts, and on the epidermal keratinocytes. These findings suggest that both epidermal and endothelial cells are infected. They also detected B19V DNA by PCR in three skin biopsies and in one serum sample, indicating that B19V DNA is present in skin cells as well as in the lumen of vessels. The authors could not however determine whether B19V replicates in the skin or the viral DNA and proteins were the result of viral load (Aractingi *et al*, 1996).

The differential diagnosis of cutaneous and oral mucosa manifestations of this novel syndrome should include blood disorders (e.g. thrombocytopenic-purpura, aplastic anemia, agranulocytosis, cyclic neutropenia), viral infections (e.g. herpetic infections, infectious mononucleosis, herpangina, hand-foot-mouth disease, atypical measles), Gianotti–Crosti syndrome, adult-onset Kawasaki disease, allergic reaction to medication, Rocky Mountain spotted fever or other rickettsial diseases, Behçet's disease, syphilis, etc (Harms *et al*, 1990; Halasz *et al*, 1992; Borradori *et al*, 1994; Smith *et al*, 1998).

In conclusion, PPGSS is an acute self-limited acral dermatosis of unknown pathogenesis, which affects young adults. It consists of pruritic edema and erythema of the hands and feet followed by purpuric-petechial rash on the same sites, and is accompanied by fever and oral lesions, which mimic the clinical picture of blood and other disorders. More than half of the reported cases have implicated B19V as the causative agent, although other viruses should not be excluded. Herein we presented the first reported case of PPGSS in Greece, in which B19V was the etiologic agent.

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