Symmetric Peripheral Gangrene Associated with H1N1 Infection

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Case Report

ABSTRACT

More and more cases of H1N1 influenza are being detected in India and so also the variety of complications this virus can cause. Here, we report a case of symmetric peripheral gangrene following H1N1 infection.

Keywords: H1N1, symmetric peripheral gangrene, vessel obstruction

INTRODUCTION

H1N1 influenza virus infection is a condition appreciated recently to present with a variety of respiratory symptoms, and most of the complications reported have been pertaining to the respiratory system mainly. However, it has the potential to cause a variety of complications involving different systems of the body.

Symmetrical peripheral gangrene (SPG) is defined as symmetrical distal ischemic damage of two or more sites in the absence of large vessel obstruction. Although it accompanies decreased flow states, such as cardiogenic and hypovolemic shock and various infectious diseases involving bacterial viral pathogens, this is a rare case where the H1N1 infection was associated with SPG.

CASE REPORT

A middle-aged woman presented to casualty with h/o fever since 7 days and bluish discoloration of fingers and palms since 5 days. She had h/o rashes over the hands and legs for 4 days and h/o loose stools four to five episodes for 2 days, 4 days back. On examination, she was febrile with tachycardia (HR – 110/min), and tachypnoea (RR – 28/min) and was in hypotension (BP – 90/60 mmHg). JVP was normal; ecchymotic rashes were present on the forearms up to the elbow and on the legs up to the knee – greenish to purple colored nonblanching. Systemic examination revealed no abnormalities.

Day 1

Investigations revealed thrombocytopenia (platelet, 43,000): Rest of the investigations including the renal function test, liver function test, serum electrolytes, and chest X-ray showed no abnormalities.

Provisional diagnosis of viral fever with thrombocytopenia was made, and the patient was initiated on supportive management with i.v. fluids, antibiotics – piperacillin and tazobactam. However, in view of the fever with rashs the diagnosis of vasculitis was also considered and evaluated for the same. Antinuclear antibody, RA factor, dsDNA, and ANCA were negative. Serologic tests- IgM, IgG for dengue and for Chikunguya were negative as well. The coagulation parameters were within the normal limits.

Day 2

The patient developed breathlessness that was associated with bilateral basal crepitations and progressive hypoxia. Meanwhile, the bluish discoloration of tips of fingers increased [Figures 1 and 2]. Chest X-ray taken showed a Acute Respiratory Distress Syndrome-like picture.

The platelet count further dropped to 13,000. Keeping dengue hemorrhagic fever/ARDS due to H1N1 as the tentative diagnosis tab. Oseltamivir 75 mg bid was started and was given for 5 days. A throat swab was sent for H1N1 analysis.

Day 3

Fever reduced and tachycardia decreased. Gangrenous changes progressed with middle and fourth fingers of her right hand frankly turning into dry gangrene. Skin rashes progressed till mid-forearm and in legs up to knee. Over next few days, she was afebrile, but breathlessness persisted and she had extensive crepts over bilateral lung fields. In addition, blackish discoloration increased in intensity and also the rashes which rose up to groin with hands and legs turning edematous. A color Doppler study of peripheral arterial and venous system showed no evidence of large vessel obstruction. Keeping diagnosis of possible vasculitic crisis in mind, the patient was also administered inj. methylprednisolone. Meanwhile, blood culture sent on the first day before initiation of antibiotics showed no growth.

She responded well to steroids and by seventh day, her breathlessness reduced and completely disappeared by eighth day. Skin lesions also improved as evidenced by her decreased edema of hands and legs and progression of the lesions halted. In the meanwhile, the throat swab for H1N1 was positive. At around tenth day, respiratory symptoms completely subsided and the gangrenous changes became restricted to both the hands distal-to-proximal interphalangeal joints [Figures 3 and 4]. A surgical opinion was taken for the same, and she was advised amputation of the upper extremity digits. However, the patient was not willing for surgery and was lost for follow-up next 2 months. By that time, total dry gangrene of the hands distal-to-PIPs had developed [Figures 5 and 6]. This time she was willing for the amputation, and the procedure was performed.

DISCUSSION

Symmetrical peripheral gangrene

Symmetrical peripheral gangrene is a well-documented but rare clinical syndrome characterized by symmetrical distal ischemic damage leading to gangrene of two or more

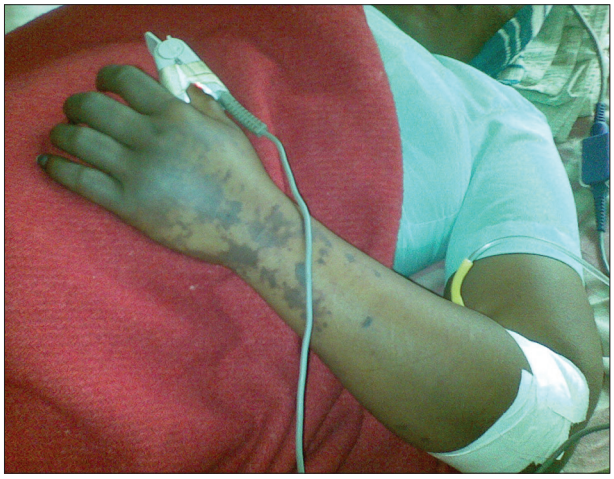

Figure 1: Evolving initial ischemic lesions in upper limbs - day 2

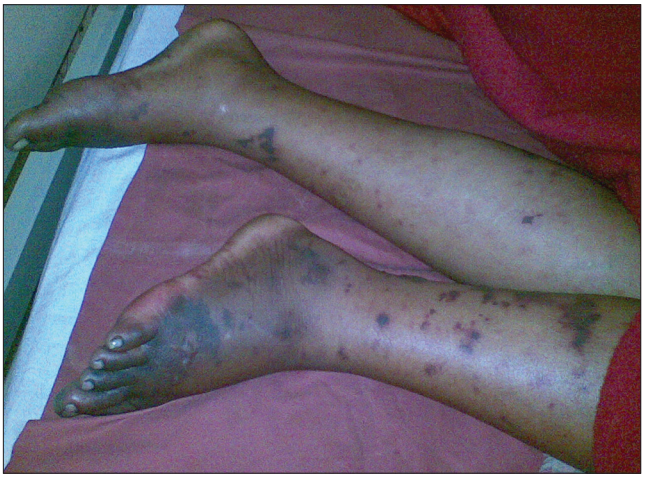

Figure 2: Initial ischemic skin lesions in lower limbs - day 2

Figure 3: Gangrenous finger tips –day 10

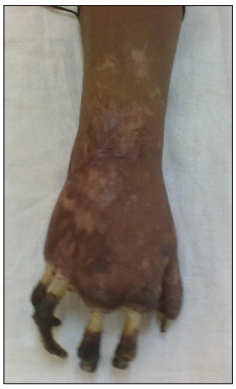

Figure 5: Dry gangrene of fingers - 2 months later

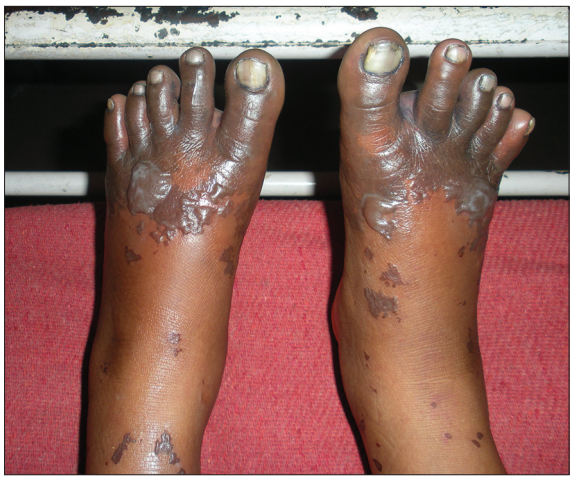

Figure 4: Resolving lower limb lesions - day 10

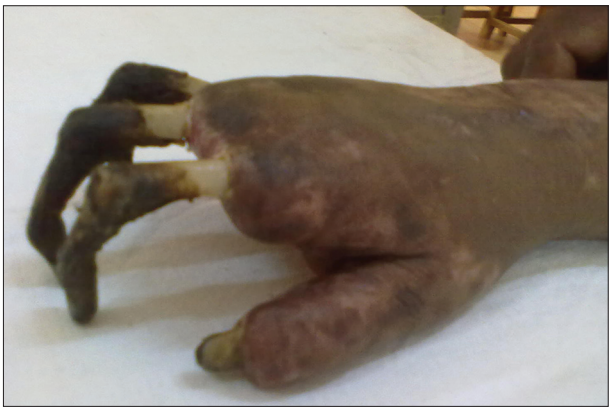

Figure 6: Dry gangrene of fingers - 2 months later

sites in the absence of large vessel obstruction or vasculitis.[1-3] It was initially described in 1981 by Hutchison[4] It is a manifestation of many systemic disorders and accompanies infectious disease of various etiologies.[5] Noninfective causes of SPG include myocardial infarction, cardiac failure,[1] hypovolemic shock,[1] hypertension,[1] pulmonary embolism, supraventricular tachycardia, Hodgkin's lymphoma, systemic lupus erythematosus, polymyalgia rheumatica, decreased levels of protein C and protein S, antiphospholipid antibodies, cryoglobulinemia, acute lymphatic leukemia, dog bite,[3] appendicitis, extracorporeal shock wave lithotripsy, suprapubic prostatectomy, cholecystectomy, sickle cell disease, hyperosmolar coma, hypernatremic dehydration, and small cell lung cancer. Drugs such as adrenaline, noradrenaline,[6] and dopamine[7] may also be important causative factors. Drugs such as ergotamine and vasopressin can cause vasospastic Raynaud's phenomena and are better excluded from the etiology of SPG. Asplenia, immunosupression, diabetes mellitus, and renal failure are among the other aggravating factors.[8] Infective organisms such as Pneumococcus,[9] *Staphylococcus aureus, Neisseria meningitides, Streptococcus pyogenes, Klebsiella pneumoniae*, *Escherichia coli*, *Salmonella paratyphi, Proteus vulgaris, Proteus mirabilis, Pasteurella multocida, Pseudomonas*,[3] *Enterococcus faecalis, Capnocytophaga, Plasmodium falciparum*, *Mycobacterium tuberculosis*, *Rubeola* virus, Dengue virus,[10] and *Varicella zoster* virus[1] have been implicated as the causative agents of SPG. Viral gastroenteritis has also been described as a causative factor of SPG.

Disseminated Intravascular Coagulation, infections, myocardial infarction, congestive cardiac failure, dog bite, pulmonary embolism, appendicitis, Hodgkin's disease, polymyalgia rheumatica, extracorpeal shock wave lithotripsy, use of vasopressors, SLE, small cell lung cancer,[11] ergotism, metastatic carcinoma of the colon, acquired hemolytic anemia, reaction to drugs (like penicillin), pulmonary embolism, pneumonia, etc., SPG can also occur as a complication of malignant disease (paraneoplastic syndrome), ergotism, or protein C deficiency. In this rarest

of the rare case, the causative organism turned out to be H1N1. This is probably the first case report of such an association between H1N1 and SPG. The cellular degeneration inflammatory mediators released during viral replication could be the contributing factors for its causation. Further studies are needed to explore other factors related to it.

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