Case Report

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Herpes Zoster as the Presenting Manifestation of Systemic Lupus Erythematosus (SLE): A Rare Case Report

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ABSTRACT

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease and is usually diagnosed with the SLICC criteria. Here we report a case of SLE presenting as Herpes Zoster (HZ). She had presented with painful vesicular eruptions from 8th thoracic nerve to 10th thoracic nerve segments and oliguria. There were no clinical manifestations suggestive of SLE. However, on further workup, haematological and immunologic laboratory profiles were suggestive of SLE. A diagnosis of lupus nephropathy was confirmed by renal biopsy and final diagnosis of SLE as the underlying systemic illness associated with HZ was established. We report this case because this patient had none of the manifestations of SLE, as a result of which this would have been an incomplete diagnosis.

Keywords: Autoimmune disease, Lupus nephropathy, SLICC criteria

CASE REPORT

A 38-year-old female presented with breathlessness on exertion for one month which was neither associated with orthopnoea nor with paroxysmal nocturnal dyspnea. She also had complains of abdominal pain since 20 days localized to periumblical region with radiation to right iliac fossa. Pain was unrelated to food intake but was associated with nausea and vomiting. There was history of nonprogressive dysphagia to both solids and liquids for last 20 days. However, there was no odynophagia. She had developed painful vesicular rash on right half of abdomen four days prior to hospitalization. There was no past history or family history of appearance of vesicular lesions. There was history of oligomenorrhea & fetal death at 21/2 months of gestation. There was no history of substance abuse. On general physical examination her blood pressure was 150/90 mmHg, pulse rate-92/min, regular. She looked pale. Dermatologic examination revealed presence of painful erythaematous vesicular bullous lesions on right side from 8th thoracic nerve to 10th thoracic nerve segments. Systemic examination revealed non tender hepatomegaly of 4-5cm. There was no splenomegaly or free fluid in the abdomen. Rest of the physical examination was unremarkable. During hospital stay patient had progressive oliguria.

On admission routine investigation revealed leukocytosis (TLC-12×10^3/µL), thrombocytopaenia (Platelets-75,000/µL) and normal haemoglobin (Hb-13gm/dl) but later on she developed anaemia (Hb-8gm/dl) and leukopaenia (TLC-1.2×10^3/µL). Kidney function test showed evidence of azotaemia (Blood urea-168, S.creat-3mg %). Electrolytes were deranged, hyponatraemia, normokalaemia were present initially followed by hyperkalaemia (K-5.5meq/l) later on, hypocalcaemia (7.7mg/dl) and hyperphosphataemia (5 meq/l) were also present. Urine examination revealed albuminuria with 24 hour urinary protein excretion being 1515 mg/24 h. Other routine investigations e.g. liver function tests, Arterial blood gases, Chest X-ray PA view, electrocardiogram, etc. didn't show any abnormality. Special investigations were carried out including immunological tests e.g. Direct coombs test - positive, Antinuclear antigen – positive (2+), dsDNA-27/IU (Positive), SSA/RO60 (4+), RO52 (3+) and TSH was increased (30.40mIU/L). She underwent Kidney biopsy in view of significant proteinuria associated with ANA and dsDNA positivity, which revealed diffuse lupus nephritis (WHO Class IV). Thus a final diagnosis of primary hypothyroidism, systemic lupus, lupus nephritis with herpes zoster was made. Patient was started on methyl prednisone 1000mg/kg i/v infusion stat for 3 days followed by oral prednisone 0.5mg/kg/day (30mg/day) for 4 weeks tapered gradually over a period of 8 weeks to 0.25mg/kg/day and the patient was maintained on the same dose of prednisone, following which she had dramatic improvement in proteinuria and azotaemia and was discharged later with an advice to follow up in Medical OPD.

DISCUSSION

Herpes zoster (HZ) is a manifestation of underlying immuno-suppressed states like malignancy, transplant recipients, cytotoxic drug intake, SLE, etc [1-3]. It is a distinctive syndrome caused by reactivation of varicella zoster virus (VZV) which presents as a painful, blistering skin eruption along one or more dermatomal segments, thoracic and lumbar being the most common. Increasing age and altered cell mediated immunity as seen in patients with neoplastic diseases (especially lymphoproliferative cancers), those receiving immunosuppressive drugs (including corticosteroids), and organ-transplant recipients, are other candidates who are at the risk of developing HZ. The risk of HZ in SLE patients varies from 13.5 to 70% in various case series [4,5]. The incidence is related to abnormal T-cell-mediated cytotoxicity which is aggravated by the simultaneous use of glucocorticoids and immuno-suppressants. It has been cited in literature that other than an increased risk of HZ infection, SLE patients are at increased risk of persistent post-zoster pain which in itself increases the morbidity of the disease [6]. HZ usually manifests following corticosteroid or immunosuppressive therapy in patients with SLE [2,3]. Diagnosis of SLE is established by SLICC criteria [7]. Our case presented with Herpes Zoster (HZ) without any clinical manifestation of underlying systemic illness including SLE. However, due to coincidental laboratory profile of unexplained azotaemia, leukopaenia and dyselectrolytaemia, the patient was further investigated to ascertain the cause of abnormalities in haematologic and renal function profile. In view of immunologic profile highly suggestive of SLE and subsequent kidney biopsy confirming the diagnosis of diffuse lupus nephritis, a final diagnosis of SLE complicated with lupus nephropathy and HZ was entertained.

SLE can rarely produce a diagnostic dilemma, as in our case. Literature describes that patients develop HZ as a complication of SLE. Hsin-Hua Chen et al., reported increased incidence of Herpes in SLE [8]. Kahl LE et al., also reported increased frequency of HZ in SLE patients as compared to general population with risk being increasing with severity of disease [1]. Harriet J Forbes et

al., described SLE as the greatest risk factor of HZ [9]. In SLE, HZ has been described after treatment with steroid/cytotoxic drugs which was also reported by Manzi S et al., [2]. But our patient was treatment naive. No such case report of SLE presenting as HZ in the absence of other common manifestations of the disease is available in literature till date.

Our patient was atypical in the sense that she didn't have any clinical features suggestive of Systemic lupus. Moreover, herpes zoster typically comes after administration of steroids but in our case herpes zoster was a presenting feature rather than a complication of treatment of SLE with steroids or cytotoxic drugs. She didn't have typical findings of SLE including arthritis, morning stiffness and photosensitivity. Absence of these features would have led to erroneous diagnosis. Though the clinical picture was completely atypical but due to awareness on the part of treating physicians to find out the cause of azotaemia in this young female, further laboratory investigations were carried out and the diagnosis of SLE was finally established.

CONCLUSION

HZ usually heralds some underlying immunocompromised state. Possibility of those conditions including SLE should be kept in mind whenever there is a slightest of doubt. Thus, presence of HZ should alert the clinician to the probability of some underlying serious immunosuppressed state especially in younger patients.

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