Simultaneous Occurrence of Varicella Zoster Virus-Induced Pancreatitis and Hepatitis in a Renal Transplant Recipient: A Case Report and Review of Literature

Puneet Chhabra, MD, DM; Priyadarshi Ranjan, MS, MCh; Deepak K Bhasin, MD, DM, FASGE, AGAF, FAMS

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ABSTRACT

Introduction: Gastrointestinal complications are common after renal transplantation, including oral lesions, esophagitis, gastritis, diarrhea, and colon carcinoma. The differential diagnosis is difficult in this scenario because multiple factors such as drugs, infections, and preexisting gastrointestinal disease come into play.

Case Presentation: We report a case of varicella zoster virus-induced pancreatitis and hepatitis in a renal transplant recipient. The patient underwent renal transplantation 3 years earlier and now presented with severe pain in the epigastrium radiating to his back and had raised serum lipase levels and skin lesions characteristic of varicella. Liver enzyme levels were also elevated. He was started on a regimen of acyclovir. His pain improved in 24 hours, and liver enzyme levels returned to normal in 48 hours.

Discussion: There is a paucity of literature on the simultaneous occurrence of varicella zoster virus-induced hepatitis and pancreatitis in both immunocompetent and immunocompromised patients. Our case highlights the gastrointestinal complications of varicella infection in immunocompromised patients that may precede the characteristic dermatologic manifestations, and the fact that rarely both hepatitis and pancreatitis may be seen.

INTRODUCTION

Varicella zoster virus (VZV), also known as human herpes virus 3, causes varicella in childhood and herpes zoster in adults. Primarily it is a neurotropic virus, but it can infect multiple organs and can cause disseminated infection, especially in immunocompromised patients.[1,2] Primary varicella infection starts with a prodrome followed by appearance of skin lesions, which are more commonly seen in older children and adults.[3] The initial lesions of varicella often involve the face, trunk, and extremities. The lesions begin as erythematous macules followed by appearance of vesicles, which later show crusting. However, these lesions may be seen in various stages simultaneously in a patient.

Infections are a major cause of concern after organ transplantation. Infections are one of the commonest causes of death in allograft recipients.[4] Availability of potent immunosuppressive drugs has reduced the incidence of graft rejection but simultaneously poses a risk of infections after transplantation. Early infections after transplantation are likely to be acquired from the surgical site, from the donors, and from nosocomial pathogens. Opportunistic infections occur later and reflect the impact of immunosuppressive drugs. The infection risk after transplant at any time point is determined by the status of immunosuppression, epidemiologic exposure, vaccination, and chemoprophylaxis status of the recipient.[5]

Most immunosuppressive drugs target T lymphocytes, which are the primary mediators in the immunogenic reaction against the graft, leading to rejection. Immunosuppressive regimens these days include two or more drugs that target the immune system at different levels. Primary varicella infection induces both humoral and cell-mediated immunity. T-cell immune response is detectable one to two weeks after infection and is composed of both effector and memory cells. Immune response is needed to maintain the varicella in subclinical stages in the sensory ganglia, and reactivation occurs once the immunity weans off, such as in patients who receive immunosuppressive drugs after transplantation.[6]

CASE PRESENTATION

Presenting Concerns

A 50-year-old Indo-Aryan man with diabetes mellitus and hypertension who had undergone renal transplantation 3 years earlier, now presented with severe pain in the epigastrium, which had been radiating to his back for the previous 7 days. He required intravenous analgesics for relief of pain and had multiple episodes of vomiting. His immunosuppression therapy was composed of tacrolimus, 2.5 mg twice daily; mycophenolate sodium, 360 mg twice daily; and prednisolone, 5 mg once daily. His blood tacrolimus level, measured 2 weeks before the onset of symptoms, was 7.2 ng/mL. His creatinine level at admission was 1.3 mg/dL (upper limit of normal = 1.4 mg/dL), and total leukocyte count was 4600/mm^3 (neutrophils = 74%, absolute neutrophil count = 3404/mm^3). At presentation the patient had a pulse rate of 88/min, blood pressure of 110/70 mmHg, and respiratory rate of 18/min, and he was afebrile. Results of the abdominal examination revealed no tenderness or guarding, although bowel sounds were sluggish. He had multiple vesiculopapular rashes all over the body in various morphologic stages typical of a varicella rash (Figure 1A and 1B), which he reported had developed 3 days after onset of the abdominal pain. Results of the neurologic examination did not reveal any evidence of sensorimotor

Puneet Chhabra, MD, DM, is an Attending Consultant in the Department of Gastroenterology and Hepatology at the Fortis Hospital Mohali in Chandigarh, Mohali, Punjab, India. E-mail: puneet.pgi@gmail.com. **Priyadarshi Ranjan, MS, MCh,** is the Director of the Urology and Kidney Transplantation Department at the Fortis Hospital Mohali in Chandigarh, Mohali, Punjab, India. E-mail: drp.ranjan1978@gmail.com. **Deepak K Bhasin, MD, DM, FASGE, AGAF, FAMS,** is the Director of Gastroenterology, Hepatology and Interventional Endoscopy at the Fortis Hospital Mohali in Chandigarh, Mohali, Punjab, India. E-mail: deepakkbhasin@gmail.com.

Figure 1A. Skin lesions in their various morphologic stages over the forehead.

Figure 1B. Skin lesions in their various morphologic stages over the abdomen.

deficit, his gait was normal, and there were no meningeal signs. Two days after admission to the hospital, his serum creatinine level was 1.2 mg/dL (upper limit of normal = 1.4 mg/dL). At presentation he had a serum lipase level of 886 IU/mL (286 IU/mL). His aspartate aminotransferase and alanine aminotransferase levels were 127 IU/mL and 193 IU/L, respectively. Serum bilirubin, alkaline phosphatase, and serum amylase levels were within normal limits. The chest x-ray film was normal and did not show any evidence of pneumonitis. He denied history of alcohol consumption or consumption of any over-the-counter preparations before the onset of illness. Hepatitis B surface antigen and antibodies to hepatitis C, A, and E viruses were negative. Autoimmune markers for liver disease (antinuclear antibodies, antismooth-muscle antibodies, and antiliver kidney microsomal antibodies) were also negative. An ultrasound scan of the abdomen did not reveal any gall stones or any feature suggestive of underlying liver disease. Contrast-enhanced computed tomography of the abdomen revealed a normal pancreas.

A diagnosis of mild interstitial edematous pancreatitis was made according to the revised Atlanta classification for acute pancreatitis. Metabolic workup to rule out other causes of pancreatitis revealed a serum calcium level of 8.6 mg/dL and fasting triglycerides level of 198 mg/dL. A diagnosis of disseminated varicella infection leading to pancreatitis and hepatitis was considered.

Therapeutic Intervention and Treatment

The patient was treated for acute pancreatitis with intravenous fluids, and a 50-mg injection of diclofenac was given as needed. Acyclovir, 10 mg/kg of body weight, was injected for treatment of varicella infection, and his immunosuppression regimen was withheld until all lesions nearly healed.

Follow-up and Outcomes

Renal functions were monitored every week after immunosuppression therapy was withheld. Tacrolimus and mycophenolate regimens were reintroduced gradually starting at 0.1 mg/kg and 720 mg/day, respectively, in divided doses. Serum creatinine levels remained stable and within normal limits during the period that immunosuppression was withheld and thereafter. The patient reported relief from pain in 24 hours. Liver enzyme levels returned to normal in 48 hours. His skin lesions after 48 hours of treatment with acyclovir showed crusting, and there was regression of skin lesions (Figure 2). He was gradually started on enteral feeding after 48 hours, which he tolerated well. He was discharged, and 4 weeks later, his liver enzyme levels were normal and his skin lesions had nearly healed. A timeline of the case is shown in Figure 3. Written, informed consent was obtained from the patient for this case report.

DISCUSSION

We describe a case of a disseminated VZV infection in an immunocompromised man with visceral involvement. Our patient primarily presented with pancreatitis and dermatologic manifestations after the pancreatic pain. He simultaneously had hepatitis as well. Although varicella virus can involve the pancreas and the liver, simultaneous hepatitis and pancreatitis are rare in primary varicella infection even if the patient is immunocompromised. This case also highlights that prompt identification and institution of therapy can prevent morbidity and mortality even if the patient presents with visceral involvement. Apart from this, the case has potential teaching opportunities regarding varicella immunization and management of such cases in solid-organ transplant recipients.

Visceral involvement in VZV infection is seen infrequently and is usually diagnosed with asymptomatic derangement in biochemical parameters. Two cases have been described in the literature in which gastrointestinal manifestations preceded the dermatologic manifestations of VZV. Both patients had gastric ulcers; one patient was receiving chemotherapy for B-cell lymphoma,[6] and the other had common variable immunodeficiency.[7] In a prospective study by Locksley et al[8] on VZV infection in bone marrow transplant recipients, 21% had visceral involvement before skin manifestations. Chitasombat et al[9] conducted a retrospective study on disseminated VZV infection after kidney transplantation. The prevalence of infection was 2% (22/1032), with most of the infections occurring 1 year after

transplantation. Half of the patients had vesicular rash, half had a dermatomal distribution, and liver involvement was seen in 9.1%. All patients improved with acyclovir treatment.[9] In a prospective study by Mustapic et al[10] of 40 renal allograft recipients, 33 patients (82.5%) had a dermatomal distribution and 5 (12.5%) had disseminated herpes zoster. Yagi et al[11] reported 2 cases of varicella infection after bone marrow transplant. Both of these patients presented with acute abdominal pain without any cutaneous signs. One patient had a diagnosis of gastrointestinal bleed in the small bowel, which settled only with acyclovir treatment, and the other patient had fulminant liver failure: varicella infection was diagnosed on postmortem liver biopsy.[11] A series of 10 patients who had undergone bone marrow transplant had abdominal pain as the initial presenting symptom of VZV infection, and only 4 of them had the characteristic varicella rash.[12] In a prospective study conducted in immunocompetent adults with chicken pox, it was seen that alanine aminotransferase levels were raised in 51.9% of patients and acute hepatic failure was seen in 1.9%.[13]

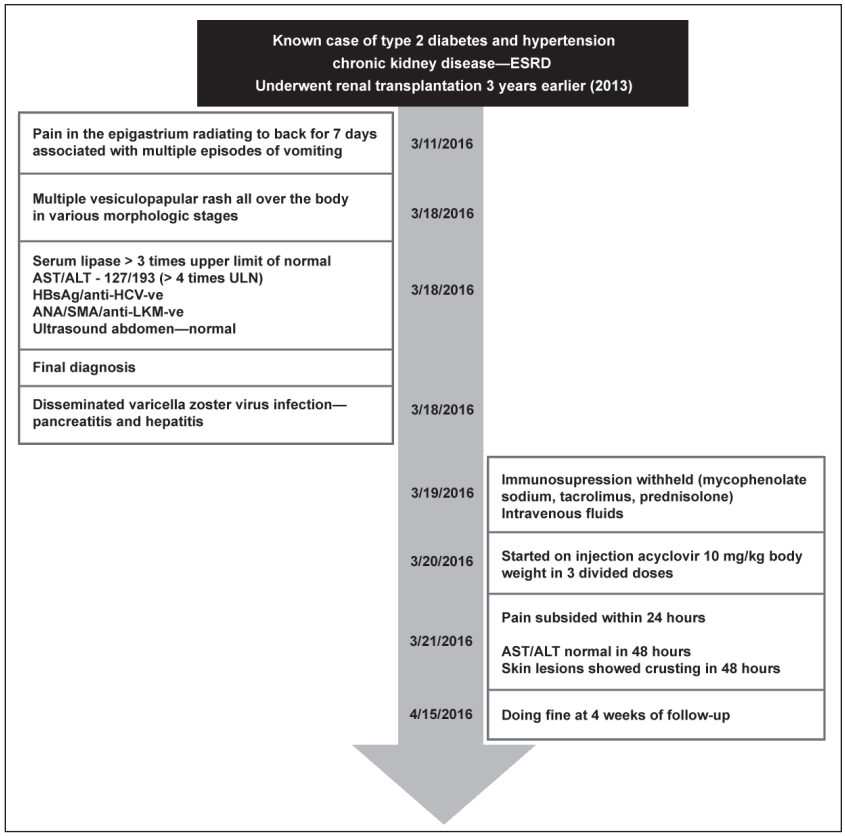

Figure 3.Timeline.

ANA/SMA/anti-LKM-ve = antinuclear antibodies/smooth-muscle antibodies/antiliver kidney microsomal antibodies; AST/ALT= aspartate aminotransferase/alanine aminotransferase (IU/L); ESRD = end-stage renal disease; HBsAg/anti-HCV-ve = hepatitis B surface antigen/antihepatitis C virus; ULN = upper limit normal.

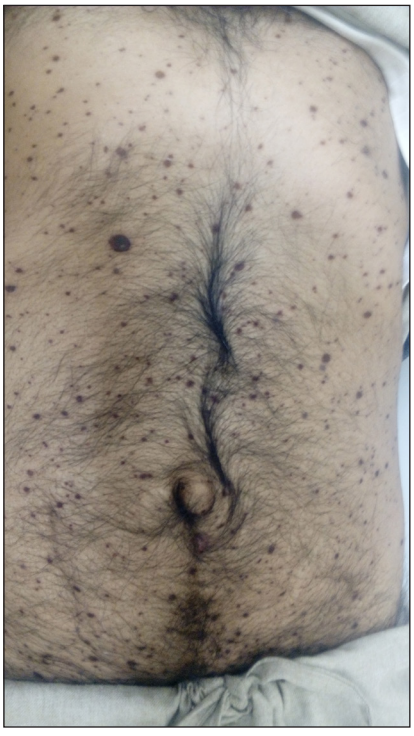

Figure 2. Crusting and regressing lesions 48 hours after initiation of acyclovir therapy.

Systemic manifestations were more common in older men who smoked and who had varicella pneumonia.[13]

Risk factors for disseminated varicella infection include an immunosuppressed state whether caused by drugs such as in posttransplant patients[14] or because of human immunodeficiency virus (HIV) infection, especially when the primary infection occurs after HIV infection.[15] Newborns who acquire infection perinatally are also at risk for dissemination.[16] The role of mycophenolate mofetil as a risk factor for disseminated varicella infection is a topic of debate.[17,18] Various risk factors for poor outcomes after disseminated VZV infection in renal transplant recipients include visceral involvement, use of azathioprine as an immunosuppressant, and longer time between transplantation and infection.[19,20]

Varicella vaccination is recommended before transplant in all seronegative individuals starting as early as 9 months of age. A live attenuated vaccine is recommended with 2 doses given 4 weeks apart. Although the vaccine is efficacious,[21,22] seronegative adults should be checked for immunity against varicella with varicella serology after 1 dose, and if they remain seronegative, the vaccine dose should be repeated. The Centers for Disease Control and Prevention recommends varicella vaccination to all children between 12 and 15 months of age, with a second dose to be given between 4 to 6 years of age.[23] Immunity to varicella after vaccination is long-lasting and is permanent in the majority. Among healthy adolescents and adults, antibodies develop in 78% after 1 dose and in 99% after the second dose. Breakthrough infection can occur with wild-type virus infection after the vaccination. It is defined as an infection with wild-type virus, which occurs 42 days after the

vaccination. The infection is milder compared with primary varicella infection.[23]

Varicella vaccine also protects against herpes zoster. In a study of children with leukemia, 4.1 years after vaccination with varicella zoster vaccine, herpes zoster developed in 2% of vaccine recipients vs 15% of controls with varicella infection.[24] Cases of disseminated varicella with visceral involvement as well as mortality have been described with the vaccine strain of varicella after vaccine administration.[25] Varicella vaccine is recommended for postexposure prophylaxis in unvaccinated persons without evidence of immunity. Vaccination within 3 days of exposure to the rash gives more than 90% protection, and within 5 days the vaccine is more than 70% effective in preventing varicella. Most studies of postexposure prophylaxis with vaccination have been done in children[26] and are lacking among adults. For immunocompromised patients including solid-organ transplant recipients receiving immunosuppression therapy who have no immunity to varicella, varicella zoster immunoglobulin is recommended. Varicella zoster immunoglobulin provides maximum benefit within 96 hours of exposure. It is recommended for transplant recipients receiving immunosuppression therapy who are exposed to family members who carry the infection.[23]

Herpes zoster vaccine is a live attenuated vaccine and is recommended for healthy adults who are older than 60 years of age.[27] Safety of zoster vaccine has been demonstrated in patients with end-stage renal disease who are older than 60 years of age. The risk of herpes zoster after vaccination is reduced in patients receiving hemodialysis, and it decreases further if the vaccine is administered within 2 years of initiation of dialysis.[28] However, in patients receiving immunosuppressive drugs, there is a modest increase in the risk of herpes zoster developing within 6 weeks of vaccination, as demonstrated by Cheetham et al.[29] Herpes zoster vaccine is not routinely recommended for use in transplant recipients and is an area of further research.[22]

Immunosuppressive drugs after infections in renal transplant recipients are modified or withheld depending on the type of infection. For BK virus infection the guidelines recommend that immunosuppressive medications should be reduced if the BK virus plasma nucleic acid test shows a viral load that is persistently greater than 10,000 copies per milliliter.[30] Similarly for cytomegalovirus infection, the immunosuppression is to be reduced if the patient has life-threatening cytomegalovirus disease or the disease persists despite treatment. For VZV infection, a temporary reduction in amount of immunosuppressive medication and continued treatment until all lesions have scabbed is recommended.[30]

Pancreatitis is a rare complication associated with VZV. Multiple cases of VZV-induced pancreatitis have been described in immunocompetent patients.[31-36] VZV is one of the etiologic agents known to trigger autoimmune hepatitis.[37] There are multiple reports of VZV-induced fulminant hepatic failure. Cases have been reported in which immunocompetent patients have required liver transplantation after VZV-induced acute liver failure and have died because of multiorgan dysfunction after fulminant hepatic failure.[38-40]

Gastrointestinal complications of varicella are common in immunocompromised patients. Gastrointestinal complications can manifest before the hallmark dermatologic manifestations of varicella, especially in immunocompromised individuals. To avoid such complications after transplant operations, pretransplant serologic evaluation should be done in all solid-organ recipients. If the recipients have a negative serologic result against varicella, they should be vaccinated. In case of exposure to varicella, unvaccinated solid-organ recipients should be given varicella zoster immunoglobulin, preferably within 96 hours of exposure.

In immunocompromised patients, especially solid-organ transplant recipients, both hepatitis and pancreatitis rarely can develop before the appearance of typical skin lesions of varicella. These patients should be asked about and checked for exposure to varicella and their vaccination status against varicella whenever they present with pancreatitis and/or an asymptomatic rise in aminotransferase levels after exclusion of the common causes. ❖

Disclosure Statement

The author(s) have no conflicts of interest to disclose.

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