

Acute appendicular perforation: A rare complication of chickenpox in an immunocompetent adult

Dharmendra Kanwaria, Porwal Y. C., Manish Kumar

ABSTRACT

Introduction: Chickenpox is a benign self-limiting disease of childhood, rarely occurs in adult. The common complications of varicella zoster infection in adults are pneumonia, diarrhea, dehydration, pharyngitis, otitis media and cutaneous secondary infection, but it may be complicated rarely with acute appendicitis in otherwise healthy patient. **Case Report:** We present a case of primary varicella zoster in adult who presented as localized peritonitis following acute appendicular perforation responded well with conservative management. **Conclusion:** Appendicular perforation may be a rare but severe complication of primary varicella infection in immunocompetent adult.

Keywords: Appendicular perforation, Chicken pox, Peritonitis, Varicella zoster infection

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INTRODUCTION

Acute appendicitis, an acute inflammation of appendix, is common in the age group of 6–18 years [1] but appendicular perforation, a complication of acute appendicitis is commonly seen in between age 30–50 years [2]. Although, varicella zoster (VZ) infection is mainly a childhood disease, it is not uncommon in adult population in tropical countries [3].

Varicella zoster is a highly infectious disease that spread from person to person by direct contact or by air from an infected person's coughing or sneezing. Typically, the disease is more severe in adult. Most people become infected before adulthood but 10% of young adults remain susceptible. The most serious complication of VZ infection is pneumonia [4]. Other common complications in both adults and children are diarrhea, pharyngitis, skin/soft tissue infection and otitis media. We report a case of an adult with VZ infection and appendicular perforation.

CASE REPORT

A 38-year-old male was admitted through emergency department with progressive distension of abdomen along with dull aching pain and breathlessness over last seven days, preceded by fever for five days, viral prodrome (nausea, vomiting, headache, diarrhea) and pleomorphic vesicular eruptions all over body (predominantly on face, neck, trunk). There was no previous history of chickenpox in the childhood or any chronic medical illness. But he had history of contact with his son having chickenpox two weeks back. On examination, he was conscious, dyspneic (respiratory rate 27 per minute) and febrile with pulse

rate of 120/minute, and blood pressure 130/70 mmHg. Multiple small hyperpigmented crusted lesions of different stages were found all over the body (Figure 1). There was mild to moderate distension of abdomen with localized guarding and tenderness over right hypochondrium and lumbar region. Bowel sounds were reduced. No lump was palpable in abdomen. All routine lab investigations were in normal limit including, CBC (hemoglobin 14 g/dl, TLC $6900/\text{mm}^3$, DLC P_{68%}, L_{25.3%}, M_{3.8%}, E_{2.9%}, platelet count 148×10^3), KFT (Blood urea 37 mg/dl, S. creatinine 1.1 mg/dl), viral markers (HIV, HBsAg, antiHCV non-reactive), Coagulation profile (PT/INR 1.12, APTT 33.2 sec), serum amylase, lipase. LFT were normal except mildly raised total bilirubin 2.3 mg/dl (conjugated 1.4 mg/dl), X-ray chest on admission showed mild pleural effusion on left side with infiltrations at lower zone, X-ray abdomen had ground glass opacity with no gas under diaphragm (Figure 2A). Even sequential X-rays did not show gas under diaphragm (Figure 2B).

Ultrasonography of abdomen showed moderate ascites with septae formation and mild hepatomegaly. Ascitic fluid analysis suggestive of inflammatory exudative picture (SAAG ratio = 0.2) (hazy appearance, absent coagulum, Total protein-3.9 g/dl, (albumin 2.5 g/dl), glucose 127 mg/dl, adenosine deaminase was 43 u/L (>60 u/L significantly positive in tuberculosis), cytology showed many RBC/HPF, WBC $10900/\text{mm}^3$ with 82% polymorphs, 16% mononuclear cells, no organism was found on Gram stain and AFB stain, no malignant cells were present). His serology done by ELISA for VZV was positive. After surgical review we managed the patient conservatively with broad-spectrum antibiotics. After 2–3 days patient clinically improved (decreased tachycardia with less fever spike and pain abdomen). Bowel sounds improved with passage of stool and flatus. Repeated ultrasonography of the abdomen, done on day-6, showed loculated ascites, analysis of which showed WBC $1800/\text{mm}^3$ (polymorphs 56%, mononuclear cells 44%) and few RBCs/HPF. Real time PCR for mycobacterium tuberculosis was negative along with negative Mantoux test (Figure 3). Computed tomography scan abdomen showed features of appendicular perforation with surrounding peritonitis (Figure 4). Patient was continued on broad spectrum antibiotics (piperacillin-tazobactam and clindamycin). All skin lesions were on healing process with crusting. Tzanck smear could not be done as the lesions already started crusting. Patient improved clinically and was discharged two weeks after surgical review with repeat ultrasonography abdomen (revealed normal finding except minimal inter bowel fluid) and routine lab test which was normal.

DISCUSSION

Acute appendicitis is usually precipitated by obstruction of the appendicular lumen.

The most common cause of luminal obstruction is fecoliths and lymphoid follicular hyperplasia. Calcium salt



Figure 1: Patient with crusted lesions.

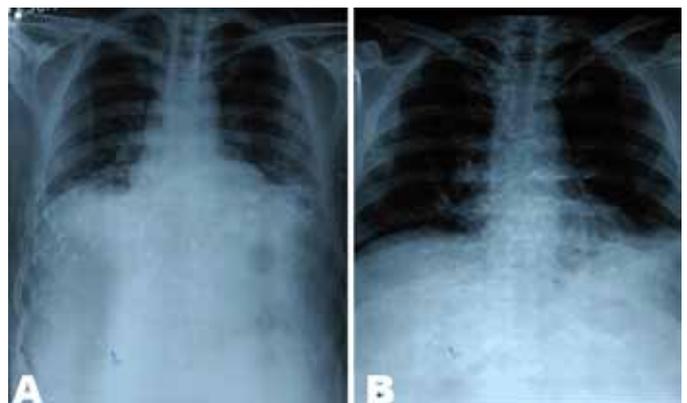


Figure 2: (A) Left side mild pleural effusion with infiltrations of lower zone with no gas under diaphragm, (B) Sequential X-ray showing no gas under diaphragm, left lower zone infiltration with mild plural effusion.



Figure 3: Negative Mantoux test.

and fecal debris deposited around a nidus of inspissated fecal material located within the appendix and form fecolith. Other less common cause of obstruction of the appendicular lumen are parasites or foreign material, tuberculosis and tumor [5]. A variety of inflammatory and infectious disorders are associated with lymphoid hyperplasia [6]. Immunocompetent adult with varicella, as compared to children, had a 2.0 times (95% CI, 1.8–2.3) higher risk of developing complications such as dehydration and pneumonia; and a 6.2 times (95% CI, 4.0–9.7) higher risk of being hospitalized [7]. Varicella may present as gastrointestinal complications such as abdominal pain, gastrointestinal bleeding, disseminated gastrointestinal infection and acute pancreatitis [8–10]. Acute appendicitis has been reported in the setting of CMV reactivation or acute CMV infection in adult patients with AIDS [11]. There are very few case reports of ‘acute appendicitis as complication of chickenpox’ in literature and they are mostly in children [12–18]. It can be a rare coincidental finding. Since acute appendicitis is a known complication of chickenpox, the possibility of VZ infection causing appendicular perforation seemed to be high. Since the patient improved on conservative

management so the specimen could not be sent for the PCR study.

Nicholas et al. found *Varicella pneumonia* as the only cause of mortality and the leading cause of morbidity and also found one case each of perforated appendicitis, suspected appendicitis and acute pancreatitis [12]. Zenon et al. reported a case of a 11-year-old boy with varicella appendicitis treated surgically and proved by positive result of VZV DNA PCR on the appendix tissue specimen [13]. Koscak reported a case of a nine-year-old girl with an acute perforated appendicitis, as a complication of varicella [14]. Camens reported a case of chickenpox encephalitis complicated by acute appendicitis and peritonitis [15]. Harvanek et al. reported a group of four children with chickenpox, acute perforated appendicitis and peritonitis [16]. Beyzaozcinar et al. reported a case of an 18-year-old immunocompetent adult with acute varicellazoster infection and appendicitis treated medically [17]. Boris et al. reported three cases of acute appendicitis with varicella infection diagnosed clinically in which two case confirmed by positive serology for VZV [18]. To the best of our knowledge we are presenting the first rare case of appendicular perforation in immunocompetent adult as a complication of chickenpox.

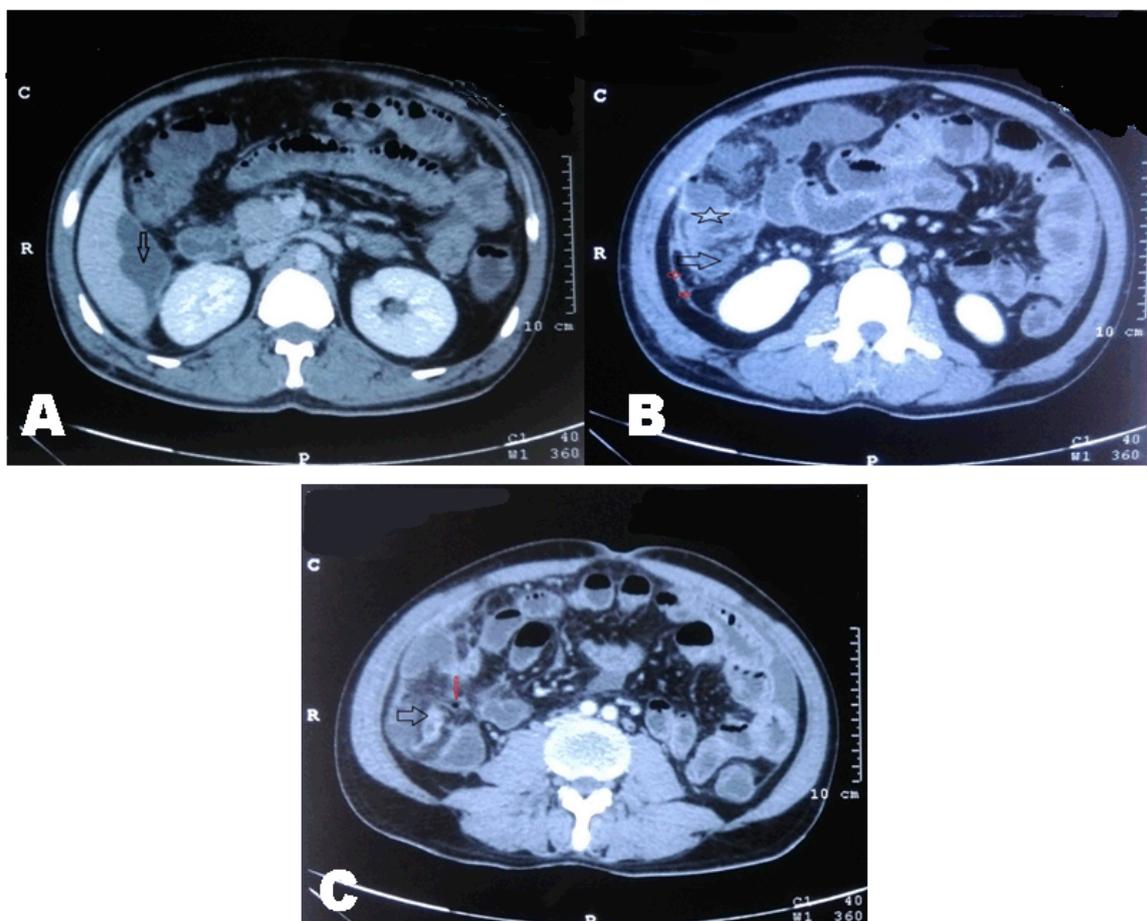


Figure 4: Signs indicative of perforation. (A) Arrow showing collection in Morrison's pouch, (B) Big arrow showing inflamed mesentery and surrounding fats. Star showing inter-bowel fluid. Small red arrow shows extraluminal air pockets, (C) Big arrow shows little part of appendix with enhancing wall. Small red arrow showing extraluminal air pocket.

CONCLUSION

Appendicular perforation may be a rare but severe complication of primary varicella infection in immunocompetent adult. The exact mechanisms causing appendicitis and perforation with activation of varicella zoster virus (VZV) and role of VZV in pathogenesis of appendicitis and appendicular perforation has to be investigated further.

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Author Contributions

Dharmendra Kanwaria – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Y. C. Porwal – Acquisition of data, Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Manish Kumar – Acquisition of data, Drafting the article, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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