

# Varicella Pneumonia with Acute Kidney Injury in an Immunocompetent Host

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## ABSTRACT

Chicken pox is mostly a benign infection of childhood and is caused by Varicella Zoster Virus which generally does not lead to any serious complication in an immunocompetent host. Severe and potentially lethal complications like varicella pneumonia, encephalitis and acute kidney injury are known entity in adults but are very rare in paediatric age group. We present a case of Acute Kidney Injury (AKI) in the form of tubulointerstitial nephritis induced by varicella infection in a young immunocompetent child with varicella pneumonia.

**Key words:** Acute Kidney Injury; Tubulointerstitial nephritis; Varicella pneumonia



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## INTRODUCTION

Varicella is mostly a self-limited benign disease with a very high secondary attack rate.<sup>1</sup> A large study of Asia pacific region reported the annual incidence of varicella from 17.8 to 323 / 100,000 populations.<sup>2</sup> Varicella is known to cause various systemic and local complications like pneumonia, AKI (Acute kidney injury), vasculopathy, encephalitis, cerebellar ataxia, retinal necrosis, otitis media, myocaditis, hepatitis, pancreatitis, cellulitis and necrotising fasciitis with varying incidences and mortality as high as 6%.<sup>3,4</sup> Renal involvement in varicella was first reported by Henoch in 1884.<sup>5</sup> Varicella pneumonia with AKI is rare but a dreaded complication which proved fatal in our case.

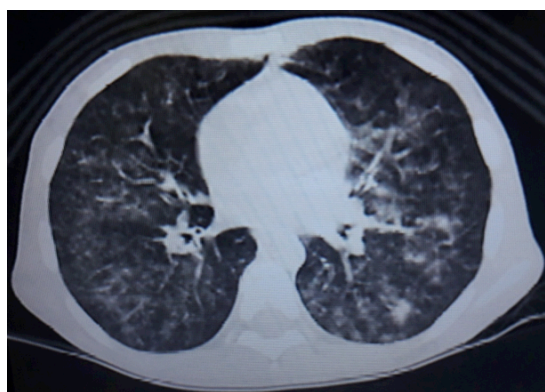
## CASE REPORT

A six year old previously healthy girl child presented on tenth day of illness with classic presentation of chicken pox related post viral pneumonia and AKI. At presentation she was febrile and anuric with altered sensorium and also had respiratory distress (Respiratory rate – 36 / minute and SpO<sub>2</sub> – 94% with oxygen by mask). BCG scar was present. Fine crackles were heard all over the lung fields. Blood examination showed neutrophilic leucocytosis (TLC - 23,300 / mm<sup>3</sup>; neutrophils - 87%; lymphocytes- 10%; monocytes - 01%). Renal functions were grossly deranged (Urea - 194 mg/dl, creatinine - 10.7 mg/dl, estimated GFR by Schwartz formula was: 5.91 ml/min/1.73m<sup>2</sup>) along with hyperkalemia (Potassium - 7.6 mEq/L). Arterial blood gas analysis revealed severe

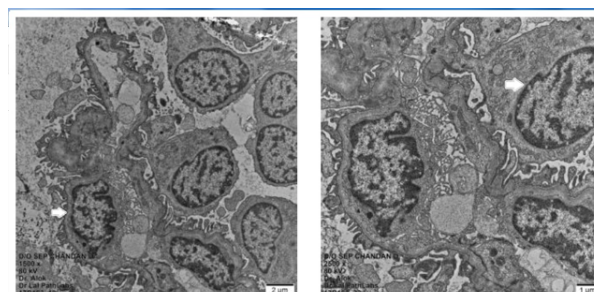
metabolic acidosis (pH- 7.008, HCO<sub>3</sub> – 5.5 mEq/L, PaCO<sub>2</sub>- 18.3 mmHg). Chest X-Ray showed bilateral non-homogenous opacities fanning two third lung parenchyma centrifugally. Bilateral widespread regions of ground glass opacities and ill-defined centrilobular nodules were seen on computed tomography (CT) of chest (Figure. 1).

Increased renal cortical echogenicity of both the kidneys with hepato-splenomegaly were noted on ultrasonogram. Blood, urine and nasal swab culture were sterile. An initial diagnosis of varicella with secondary infection causing MODS (Multiple organ dysfunction syndrome) was made. Further work up revealed low complement levels C<sub>3</sub> - 68.60 mg/dl, normal C<sub>4</sub> - 21.80 mg/dl with negative pANCA and cANCA (Perinuclear and cytoplasmic anti-neutrophil cytoplasmic antibodies respectively).

Renal functions and electrolytes improved after 57 cycles of peritoneal dialysis with 1.5% of peritoneal fluid. She required three anti-hypertensive medicines to control her blood pressure below 90<sup>th</sup> percentile. Simple microscopy of kidney biopsy revealed patchy acute tubular injury in the form of multifocal dense chronic lymphoplasmacytic interstitial inflammation and interstitial edema. Immunofluorescence examination showed linear positivity for IgG, kappa & lambda light chains along tubular basement membranes. Other parameters on immune staining pattern were negative. The glomerular basement membrane thickness ranged from 229.4 to 300.6 nm with 20% focal effacement of visceral epithelial cell foot processes (Figure. 2).



**Figure 1.** Computed Tomography of chest showing bilateral widespread regions of ground glass opacities



**Figure 2.** The ultrastructural examination showed glomerular basement membrane thickness with focal effacement of visceral epithelial cell foot

**Table 1.** Laboratory parameters during the course of hospitalisation

Investigations	On day of admission	9th day of admission (Kidney biopsy was done)	17th day of admission (Required ventilator support)
Haemoglobin (gm/dl)	10.1	8.4	7.9
Total Leucocytes count	23,300 / mm <sup>3</sup>	11,600 / mm <sup>3</sup>	19,700 / mm <sup>3</sup>
Neutrophils	87%	59%	88%
Lymphocytes	10%	35%	06%
Monocytes	01%	02%	02%
Eosinophils	02%	04%	04%
Platelets	833 x 10 <sup>9</sup> / L	479 x 10 <sup>9</sup> / L	395 x 10 <sup>9</sup> / L
Urea / Creatinine (mg/dl)	194 / 10.7	119 / 3.7	64 / 1.6
Sodium / Potassium (mEq/L)	134 / 7.6	140 / 4.5	141 / 5.3
Calcium (mg/dl)	8.4	7	8.4
Alkaline Phosphatase (IU/L)	238	119	-
Protein /Albumin (gm/dl)	5.9/2.1	-	6.1/2.1
Procalcitonin (PCT)	-	-	0.12 ng/mL
C- reactive protein (CRP)	-	-	5 mg/L
<i>Compliment C<sub>3</sub>: 68.60 mg/dl (low)</i>			
<i>Compliment C<sub>4</sub>: 21.80 mg/dl (Normal)</i>			
<i>pANCA (perinuclear Anti-neutrophil cytoplasmic antibodies) - Negative</i>			
<i>cANCA (cytoplasmic Anti-neutrophil cytoplasmic antibodies) - Negative</i>			
<i>Urine routine and microscopic examination - Albumin detected, 10 - 12 pus cells, 6 - 8 Red blood cells and granular cast seen</i>			
<i>Blood culture, urine culture, peritoneal dialysis fluid culture and nasopharyngeal swab culture were sterile on all occasions</i>			

The raised leucocytes along with its differential counts returned to normal but after 17<sup>th</sup> day of management it started rising (Table 1).

The child remained febrile and oxygen dependent. Graded oxygen therapy was instituted and acyclovir along with antibiotics in renal adjusted doses was administered as per hospital policy. Her respiratory parameters continued to worsen. She did not respond while on ventilator support and succumbed to her illness.

## DISCUSSION

Complications of varicella are severe in immunocompromised individuals like pregnant women, elderly and infants.<sup>6</sup> Healthy children are less prone for have serious and potentially lethal complications but multi organ involvement irrespective of immune status is not a rarity in paediatric age group. Similar to this case renal

involvement leading to AKI with varicella pneumonia has been described in adults.<sup>7</sup>

Varicella pneumonia is one of the serious complications presenting in a healthy child.<sup>8</sup> Mortality rate among children with varicella pneumonia varies and has considerably decreased in post vaccination era.<sup>9</sup> In our case, clinical and laboratory findings at presentation rendered the possibilities of septicaemia, varicella pneumonia, or pulmonary edema due to AKI. The raised leucocyte count returned to normal but started rising after 17<sup>th</sup> day of hospitalisation (with negative C reactive protein and procalcitonin) while on adequate antibiotic cover, refuting the possibility of secondary bacterial affection as the blood and other body fluid cultures also remained sterile. CT chest was also convictive of a viral etiology.

Various associations of kidney injury have been reported in varicella infection. In this case, though there was neutrophilic leucocytosis but negative culture reports disfavour sepsis causing AKI, more so the child was hypertensive at presentation. Renal biopsy showing a lymphoplasmacytic infiltration instead of a mononuclear response along with negative blood culture also contradicted sepsis related renal damage. Acute tubulointerstitial nephritis is known to happen by many virus infections including herpes group of viruses. It can be caused by immunological response even in absence of direct invasion of viruses. About 1% cases of acute interstitial nephritis may progress to acute renal insufficiency. Nephrotoxic drugs like Acyclovir may cause acute tubulointerstitial nephritis but in this case the child had not received it prior to onset of AKI. The possibility of renal tubular damage secondary to varicella infection is high on cards in this case, though the mechanism remains arguable. AKI caused by acute tubular necrosis secondary to hypovolemia in varicella infection is known in adult patients.<sup>10</sup> Presence of plasma cells in interstitium rears a doubt of drug

induced allergic nephritis of acyclovir but eosinophil counts were always within the normal range in our case. The values of complement levels suggested immune complex mediated renal injury. However, immunofluorescence examination of kidney biopsy did not show glomerular immune deposition of IgG or C3. Microscopic examination of urine showed the presence of granular cast, characteristic of glomerular basement membrane injury but the histopathological findings of kidney biopsy suggested tubulointerstitial nephritis, possibly caused by a viral etiology.

## CONCLUSIONS

We conclude that varicella infection in paediatric age group may present with utmost severity regardless of immune status of host. Acute tubular injury induced by varicella infection leading to AKI is a rare entity. Early recognition and prompt institution of appropriate management can restore normal renal function.

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