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Case Series

Adenoviral infection in children with multisystem involvement: a case series from a tertiary care hospital in Eastern India

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ABSTRACT

Adenovirus infection in children commonly causes mild respiratory illness; however, severe and multisystemic forms are increasingly recognized even among immunocompetent individuals. This hospital-based observational case series included four children with PCR-confirmed adenoviral infection who presented with diverse clinical manifestations encephalitis, myocarditis, hepatitis and severe pneumonia with multiorgan dysfunction. All the 4 cases belonged to age group of below 5 years and had no known co-morbidities or immunodeficiency. The patients had life threatening complications including encephalitis, myocarditis, hepatitis, acute kidney injury and multiorgan dysfunction. Detailed clinical evaluation, laboratory investigations, and treatment outcomes were documented. Despite intensive management, one child succumbed to respiratory failure with multiorgan dysfunction, while the other three recovered completely. Early detection, supportive intensive care, and vigilant monitoring for complications are essential for decreasing the mortality and morbidity.

Keywords: Adenovirus, Encephalitis, Myocarditis, Multiorgan dysfunction, Children

INTRODUCTION

Human adenoviruses are double stranded DNA viruses which cause a range of illness, including upper respiratory tract symptoms, fever, pneumonia, gastroenteritis and conjunctivitis.[1] A total of 51 serotypes of human adenoviruses are recognized and approximately 85 genotypes have been described.[2] The clinical course of this infection is usually self-limited; however it can cause significant morbidity and mortality in young children or immunocompromised persons.[3] In some patients, adenovirus infection can cause severe pneumonia, myocarditis, hepatitis, encephalitis, nephritis and disseminated disease which may lead to acute respiratory distress syndrome (ARDS) and multiple organ dysfunction syndrome (MODS). If not treated timely and appropriately, the mortality rate can be over 50%.[4,5] Unfortunately, no effective antiviral therapy or vaccines are available for the treatment or prevention of adenovirus in children/adults and treatment is mostly supportive. We herein report 4 pediatric cases of adenovirus infections with diverse clinical presentations and multisystem involvement.

CASE SERIES

Case 1

A 1-year 6-month-old boy presented to pediatric emergency with fever for 4 days prior to admission; fever was high grade, ranging between 102-103 °F without chills and rigor and subsided on taking medications. It was associated with multiple episodes of generalized tonic clonic convulsions which developed on second day of fever, following which child became drowsy and irritable. Fever was also associated with redness of both eyes and a rash over neck and trunk. The baby also developed difficulty in breathing one day before admission. Post admission, child was drowsy, febrile (102°F) and pale with a pulse rate (PR) of 128 beats/minute, respiratory rate (RR) of 60/minute, blood pressure (BP) of 90/60 mm Hg and O_2 saturation (SpO_2) ranging between 85-89% in room air.

Head to toe examination showed bilateral conjunctival congestion and maculopapular rash over neck and trunk. On central nervous system (CNS) examination, Glasgow coma scale-E2V2M4, tone was normal in all 4 limbs, refexes were normal (2+), plantar response was flexor bilaterally, meningeal signs were absent and there was no cranial nerve involvement. Chest examination showed presence of subcostal and intercostal retractions and rhonchi were audible bilaterally on auscultation. Abdominal and cardiovascular (CVS) system examination was normal.

After admission, child had multiple episodes of convulsion and was started on anti-epileptic drugs and 3% NaCl injection (considering raised intracranial tension). Intravenous antibiotics were started. He was given supplemental oxygen via face mask and nebulized bronchodilators to relieve the respiratory symptoms. Packed red blood cells (PRBC) were given due to anaemia. The hematological and biochemical parameters revealed the following- discussed in Table 1.

Lumbar puncture was done and cerebrospinal fluid (CSF) showed pleocytosis (cell count- 152/cu.mm, cell type- lymphocytes 95%), raised protein (110 mg/dl) and low sugar 56mg/dl, gram stain and acid-fast bacilli (AFB) were negative. Blood and CSF cultures showed no growth. Dengue IgM, Typhidot IgM and blood for malarial parasites were negative. Fundoscopy and magnetic resonance imaging (MRI) brain were normal.

Chest X ray showed diffuse haziness in both lung fields. In view of fever, rash, bilateral conjunctival redness and respiratory symptoms, nasopharyngeal swab for respiratory viral polymerase chain reaction (PCR) was sent and came positive for adenovirus (Figure 1).

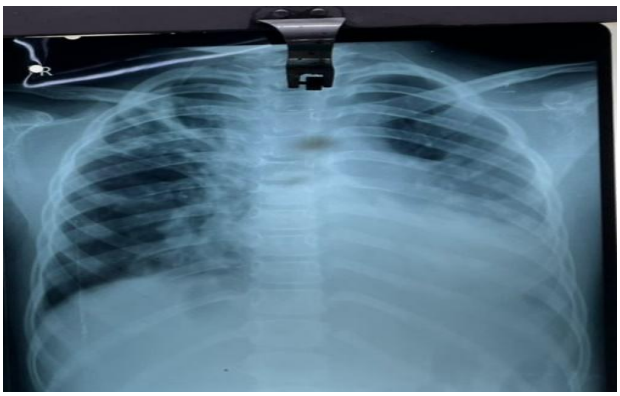

Figure 1: Chest X-ray (case 1).

With the above-mentioned management, the child responded well; he became afebrile, convulsions stopped and respiratory distress subsided. He was allowed to feed orally, once the child became asymptomatic and hemodynamically stable, he was discharged from the hospital.

Case 2

A 8-month-old baby boy presented with a history of high-grade intermittent fever for 3 days, he also developed cough and shortness of breath for past 2 days. There was no history of rash. On examination, baby was irritable, febrile (103 °F), tachypneic (RR-74/minute), tachycardic (PR-176/minute) with visible intercostal and subcostal recessions and had an SpO_2 of 85% in room air. Respiratory system examination showed presence of bilateral crepitations on auscultation, auscultation of heart revealed gallop rhythm and per abdomen there was a soft palpable hepatomegaly (4 cm below right costal margin).

After admission, baby developed hypotension and was started on intravenous dopamine infusion. Arterial blood gas (ABG) analysis showed presence of metabolic acidosis (pH-7.1) with high lactate levels (12.1 mmol/l). An initial fluid bolus @10 ml/kg was given followed by maintenance fluids. Respiratory distress worsened further and baby was put on Heated humidified high flow nasal cannula (HHHFNC). Bedside echocardiography could not be done due to non-availability in our centre. Antibiotics were started empirically.

Chest X ray showed cardiomegaly with perihilar infiltrates. Hematological investigations revealed- described in Table 1. C-reactive protein (CRP) and procalcitonin were negative. Blood for dengue NS1, malarial parasites were negative. Creatine kinase- MB (CK-MB) was sent, came positive in high titres (680 U/l). Nasopharyngeal swab for respiratory viral PCR was also positive for adenovirus (Figure 2).

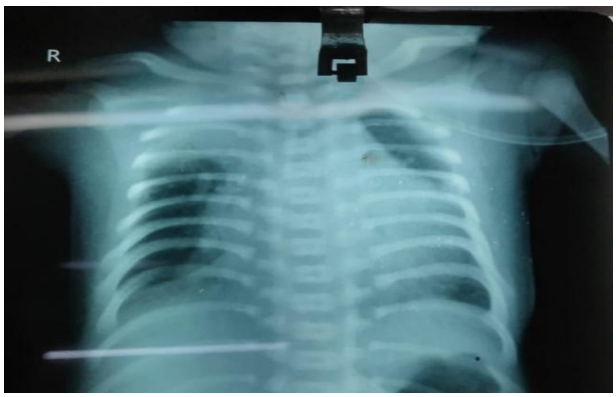

Figure 2: Chest X-ray (case 2).

Suspecting adenoviral myocarditis, intravenous immunoglobulin (IvIg) was given @1 gm/kg/day for 2 days. As blood cultures were negative, antibiotics were stopped after 48 hours. With this management, baby slowly improved clinically and was weaned off from HHHFNC to face mask and then to room air. He became asymptomatic 12 days after admission and was discharged in a hemodynamically stable condition. On follow up, CK-MB levels normalized and Echocardiography was normal.

Case 3

A 4 years old girl presented to the pediatric out-patient department with a history of fever for 8 days which was high-grade ranging between 101-102 ºF, not associated with chills and rigour and subsiding with anti-pyretics. It was associated with runny nose and dry cough for same duration and diarrhoea for 4 days. The stools were loose watery in consistency, not mixed with mucus and blood and rectal symptoms were also absent. On admission, the child was alert and active, afebrile, and had stable vitals (PR-90/minute, RR-32/minute, BP-100/60 mm Hg and SpO_2 – 98% in room air). 2 days after admission, child developed jaundice with multiple episodes of vomiting and abdominal pain. It was associated with loss of appetite and pale yellow-coloured stools. There was no evidence of bleeding or drowsiness or behavioural abnormalities.

On examination, she was icteric, her abdomen was soft and liver was palpable 3 cm below the right costal margin, it was soft in consistency and non-tender. There was no splenomegaly or ascites. Respiratory system examination showed presence of fine crepitations bilaterally. CNS and CVS examination were normal.

Laboratory investigations revealed the following- liver function tests showed direct hyperbilirubinemia (direct bilirubin- 9.4 mg/dl) and total bilirubin – 12 mg/dl, SGPT-365 U/l, SGOT-296 U/l and ALK 432 U/l. GGT and coagulation profile were normal.

Abdominal ultrasound showed hepatomegaly with normal echotexture. Hepatitis workup including hepatitis A, B, C and E, Ebstein Barr virus, cytomegalovirus and herpes simplex viral panels were negative. Blood for malarial parasites, dengue IgM, Scrub typhus IgM and Widal tests were negative. Work up for probable non-infectious causes of hepatitis including anti-nuclear antibody, anti- smooth muscle antibody, serum ceruloplasmin were negative. Since the onset of hepatitis followed after the respiratory symptoms and diarrhoea, and also the persistence of respiratory symptoms prompted us to send nasopharyngeal swab for viral respiratory PCR and it was positive for adenovirus. Stool samples were also sent for adenovirus nucleic acid detection by PCR and came positive.

Our patient was given supportive treatment; gradually her liver enzymes and serum bilirubin returned to baseline. She was discharged 10 days after admission in a stable condition.

Case 4

A 2-month-old girl was brought with a history of cough and cold with runny nose for 4 days; 1 day before admission she developed respiratory distress which worsened very fast and she also had 1 episode of generalized tonic clonic convulsion and was taken to our hospital. There was no associated history of fever or rash. On admission the baby was pale and had severe respiratory distress with RR of 86/minute, SpO_2 of 70% with oxygen via face mask @6 l/minute and PR of 142/minute. Auscultation of chest revealed bilateral coarse crepitations, abdominal palpation showed soft 2 cm hepatomegaly. Cardiovascular and CNS examination were normal.

She was immediately transferred to pediatric intensive care unit (PICU), intubated and started on invasive mechanical ventilation with synchronised intermittent mandatory ventilation mode with FiO_2 - 80%, peak inspiratory pressure – 15mm Hg, Positive end expiratory pressure- 5 mmHg and RR-45/minute. ABG analysis showed presence of respiratory acidosis (partial pressure of CO_2 -94 mm Hg and pH-7.02) and hypoxemia (partial pressure of O2 -60 mmHg); ventilator parameters were increased.

Chest X ray showed presence of bilateral diffuse infiltrated in both lung fields (Figure 3). Bedside Echocardiography couldn't be done due to non-availability. Baby was started on injectable antibiotics, antivirals (oseltamivir) and antiepileptics. Complete hemogram (Table 1) showed severe anaemia (Hb-6.8 g/dl); PRBC was transfused @10 ml/kg. Nasopharyngeal swab for viral respiratory PCR was positive for adenovirus. Blood cultures were sterile; antibiotics and oseltamivir were stopped after 48 hours.

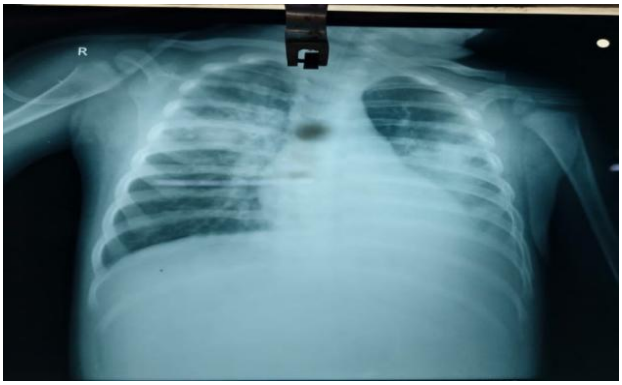

Figure 3: Chest X-ray (case 4).

On day 4 of admission, hemodynamic parameters worsened further and injection Dobutamine was started at a dose of 5 µg/kg/minute, later increased to 7.5 µg/kg/minute. She also developed facial puffiness and abdominal distension with diminished urine output. Renal function tests showed elevated serum urea and creatinine with dyselectrolytemia (serum sodium-146 mmol/l and potassium-6.8 mmol/l).

She underwent several cycles of peritoneal dialysis; however, the baby failed to respond to the above management and eventually succumbed to respiratory failure with multi-organ dysfunction secondary to severe adenoviral infection.

Laboratory investigations carried out in the 4 cases have been described in Table 1.

Table 1: Investigations carried out in the 4 cases.

Investigations	Case 1	Case 2	Case 3	Case 4
Hemoglobin level (g/dl)	7.1	10.2	6.8	10.6
Total leucocyte count (/cu.mm)	9800	10,400	50,200	9650
Differential count –neutrophil (N) lymphocyte (L)	N-24% L-68%	N-32% L-64%	N-56% L-40%	N-40% L-58%
Platelet count (lacs/cu.mm)	2.1	1.98	0.65	2.5
Serum urea (mg/dl)	32	19	121	26
Serum creatinine (mg/dl)	0.4	0.5	2.3	0.3
Serum sodium (mmol/l)	136	142	146	138
Serum potassium (mmol/l)	3.7	4.2	6.8	3.9
Serum calcium (mg/dl)	9.4	8.9	8.6	9.8
Liver function tests				
SGPT (U/l)	Normal	Normal	Normal	365
SGOT (U/l)				296
ALK (U/l)				432

DISCUSSION

Adenovirus accounts for at least 5-10% of pediatric respiratory tract infections.[6] While majority of these infections are mild and go unrecognized without requiring hospitalization, some may lead to complications as certain adenovirus species exhibit tissue-specific tropism.[7] Serotypes 1, 2, 3 and 5 mainly cause respiratory illness, serotypes 40 and 41 cause gastroenteritis; certain serotypes such as HAdV-7 have been associated with severe disease manifestations like respiratory failure, shock and hepatitis. Multisystem involvement in adenovirus infection is uncommon; although severe and disseminated presentations are typically reported in immune-compromised hosts.[8,9] The children in our study had no known comorbidities or underlying immunodeficiency.

The first case presented with fever, rash, conjunctival congestion, seizures, and respiratory distress, suggestive of a multisystem inflammatory response due to adenoviral infection. The second case had clinical and biochemical evidence of myocarditis, with elevated CK-MB and cardiomegaly on chest X-ray, which responded to intravenous immunoglobulin therapy. The third case had acute hepatitis following a febrile respiratory and diarrhoeal illness.

The fourth case demonstrated the most severe form, with adenovirus-induced pneumonia progressing to ARDS, renal failure, and multi-organ dysfunction leading to death. These cases highlight that adenoviral infection, although often mild, can result in severe complications such as encephalitis, myocarditis, hepatitis and acute kidney injury (AKI) in previously healthy children.

A hospital-based observational study from South India 10 involving 130 children with PCR-confirmed adenovirus infection found that multisystem involvement was present in approximately 75% of cases, and non-respiratory manifestations in about 20%. Common complications included AKI, left ventricular dysfunction, acute liver failure, ARDS, and multiorgan dysfunction with an overall mortality of 13%. These findings are consistent with our cases: our case 3 displayed many of those severe features (respiratory failure, AKI, multi-organ dysfunction), whereas cases 1 and 2 had multisystem or non-respiratory involvement but eventually recovered. A case series of infection with adenovirus type 7 in South India reported very high mortality (57%), with frequent myocarditis, CNS involvement, and haemophagocytosis among young children needing mechanical ventilation.[11] Our findings are also in line with a retrospective study from North India 12 including 85 children with adenoviral pneumonia that reported a mortality rate of 22%, with frequent complications such as ARDS, MODS, shock, encephalopathy, and AKI; low admission pH, myocardial dysfunction, ARDS, and MODS were independent predictors of death.

There is currently no specific antiviral therapy approved for adenovirus infection in immunocompetent children. While cidofovir has shown activity against adenovirus in vitro and in immunocompromised hosts, their use is limited by toxicity and lack of pediatric data. Ribavirin and oseltamivir are generally ineffective. Hence, management remains largely supportive. Oxygen therapy, mechanical ventilation, vasoactive support, and renal replacement therapy are the mainstays of treatment in severe cases. Intravenous immunoglobulin (IVIG) and corticosteroids have been used empirically with variable success, as observed in our second case, but evidence supporting their routine use is limited. Early detection through viral PCR and rational discontinuation of unnecessary antibiotics are essential to avoid antimicrobial overuse and secondary infections.

Mortality in adenoviral pneumonia complicated by multi-organ dysfunction remains high; the outcome depends on the virulence of the viral strain, host immune response, age, and availability of intensive care interventions such as mechanical ventilation, continuous renal replacement

therapy, and extracorporeal membrane oxygenation (ECMO). In resource-limited settings, the unavailability of these advanced supports, further worsens prognosis.

CONCLUSION

Adenoviral infections in children can range from mild respiratory illness to life-threatening multisystem disease even in previously healthy individuals. Early recognition, supportive intensive care, and vigilant monitoring for complications such as myocarditis, renal dysfunction, and ARDS are crucial for improving outcomes.

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