CASE REPORT

MEASLES WITH THE COMPLICATION OF NON-OBSTRUCTIVE HYPERTROPHIC CARDIOMYOPATHY; CASE REPORT

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ABSTRACT

Measles is a highly contagious disease caused by a viral infection. It is transmitted through the respiratory route, and the illness begins with fever, cough, coryza, and conjunctivitis, followed by a distinctive rash. It is best prevented through vaccination. Complications that can be seen in measles are otitis media, diarrhea, pneumonia, encephalitis, and myocarditis, and a long-term complication is subacute sclerosing panencephalitis. We present a case of a 6-month-old girl who came with measles-like symptoms and later developed complications of non-obstructive hypertrophic cardiomyopathy.

Key Words: Pneumonia, Cardiac arrest, Pneumothorax, ARDS

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INTRODUCTION

Measles is a significant concern with approximately 10 million people infected annually causing over 100,000 deaths worldwide [1]. It is an extremely infectious illness caused by the measles virus, and it continues to cause over 100,000 fatalities annually. There is a significant decrease from the previous annual toll of over 2 million deaths,due to the implementation of the measles vaccine [2]. Four decades after the introduction of effective vaccines, measles still poses a threat, leading to fatalities and severe illnesses in children globally. Complications arising from measles can affect nearly every organ system, with pneumonia, croup, and encephalitis being frequent contributors to mortality. Encephalitis stands out as the primary cause of long-term consequences [3].

Hypertrophic cardiomyopathy (HCM), a genetic disease caused by mutations in sarcomeric contractile proteins, is characterized by left ventricular (LV) hypertrophy, myocardial fibrosis, and myocyte disarray. As a result, patients may experience functional limitations [4]. Despite its rare prevalence in pediatric age, HCM carries a relevant risk of mortality and morbidity in both infants and children. Pediatric HCM is a large heterogeneous group of disorders [5].

CASE REPORT

A 6-month-old baby girl, fully immunized up to her age and weighing 7.5 kg, was brought to the emergency room with a 7-day history of fever, generalized tonic-clonic fits for one day, shortness of breath, and an inability to feed for one day.

On a general physical examination, the child was pale, sick-looking, and had rashes all over her face and body. Capillary refill time: < 3 seconds. Her vitals were BP 109/50, pulse 140 beats per minute,

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Fahrenheit, and oxygen saturation 82% at room air. The central nervous system (CNS) examination revealed a Glasgow Coma Scale (GCS) of 15/15. Upon auscultation of the chest, harsh vesicular breathing and crepitations were audible. Examination of the abdomen and cardiovascular system (CVS) did not reveal any abnormalities. Clinical laboratory investigation (tables 01, 2, & 3) showed increased neutrophils (72%), decreased PO2 (49.3 mmHg), decreased oxygen saturation (85.9%), increased C-reactive protein (21.3 mg/L), and decreased potassium (2.5 mEq/L). Based on the clinical presentation, further imaging investigations were done. Chest x-rays show typical signs of pneumonia. An echocardiogram (ECHO) was done, which showed hypertrophic cardiomyopathy (non-obstructive); no cardiac medication was required; only follow-up was needed after three months. Her management includes B-CPAP 6/6, intravenous fluids, intravenous antipyretics, intravenous antibiotics, and vitamin A.

Table 01: Abgs showing Hypoxia with O2 Saturation of 85%

Variables	Findings
pH	7.439
PCO3	1.8
PO2	49.3
Bicarbonate	21
Bases excess	-2.0
O2 SAT	85.9

Table 02: Complete Blood Count showing Anemia and Neutrophil

Varaibles	Findings
Hemoglobin	11
Red cell count	4.87
H.C.T (P.C.V)	33
M.C.V	67
M.C.H	23
M.C.H.C	34
R.D.W	17.0
Total WBC count	6.9
Neutrophils	72
Lymphocytes	21
Monocytes	06
Eosinophils	01
Platelet count	185

Table 03: Clinical Chemistry Showing Hypokalemia and Raised CRP

Variables	Findings
Urea	21
Creatinine	0.4
C-reactive protein	21.3
Na	134
K	2.5
Cl	98
HCO3	20

She was under observation in the pediatric intensive care unit, and every day her labs and chest x-rays were sent, which didn't show any improvement. However, despite these management measures, the patient's condition did not improve and continued to deteriorate, and she was transferred to a ventilator. The patient developed generalized edema gradually along with sepsis on her fourth day of admission. On the ninth day, she developed an apical pneumothorax (Figure 01 & 02) on the right side of her lung with homogenous opacity in both lungs.

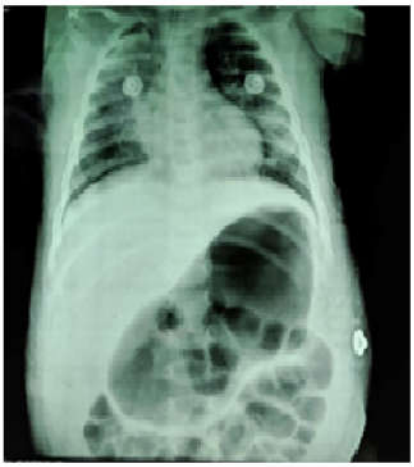

Figure 1 : 6 Month-Old Baby Girl's Chest X-Ray of Pneumonia

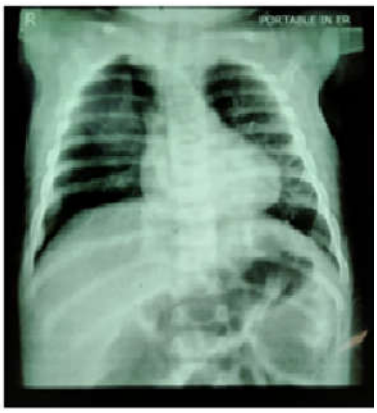

Figure 2 : Chest Ray Showing Right Sided Pneumothora

Despite the management plan involving the multidisciplinary team to effectively treat measles with complications, the patient's condition continued to deteriorate. On the 10th day of her admission, she died due to cardiopulmonary arrest. (Primary cause: Acute Respiratory Distress Syndrome (ARDS), secondarycause: measles with complications)

DISCUSSION

Measles, a ribonucleic acid (RNA) virus infection primarily observed in children, persists as a health concern despite the existence of a highly effective vaccine. While the disease is generally self-limiting, it can progress to a severe stage in individuals who are undernourished or immunocompromised.The immunosuppressive effects of the measles virus contribute to secondary infections by opportunistic bacteria in those affected [6]. In both genders, the major complications were pneumonia; lower respiratory tract infection (LRTI), acute diarrhea, diarrhea and LRTI, pneumonia and diarrhea, otitis media and pneumonia, myocarditis and LRTI, and pneumothorax. The majority of the infected children under 12 months of age had associated complications [7].

Hypertrophic cardiomyopathy is caused by to genetic mutation in which the heart muscle becomes thickened, making it harder for the heart to pump blood efficiently. It's two types; in obstructive hypertrophic cardiomyopathy there is obstruction due to which blood can't flow but in non-obstructive hypertrophic cardiomyopathy there is no obstruction so blood flow is normal.

In this case, the child presented with non-obstructive hypertrophic cardiomyopathy as a complication of measles. There was no family history of any heart disease and the parents had a non-consanguineous marriage. She was getting treatment but her condition was not improving which led to the spread of sepsis in her body along with apical pneumothorax. After 10 days she died because of cardiopulmonary arrest with ARDS as a primary cause and measles with complication as a secondary.

The presentation of non-obstructive cardiomyopathy as a measles complication is unique on its own with unknown aetiology and to consider it as a new complication of measles, we will need more research on it whether it was due to genetic mutation or it is because of measles.

CONCLUSION

Although measles has proven to be the cause of mortality or the trigger for other associated morbidities, its association with non-obstructive cardiomyopathy is still of unknown origin. Further studies can help us determine the basis for the condition.

The best option that we can apply right now is to vaccinate the children and hold campaigns to educate parents about the possible outcomes with examples of such scenarios that would make them aware of its importance.

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Author's Contribution

RS: Case presentation, data collection
SH:Manuscript writing and editing
MK:Data collection, manuscript writing and editing
TA:Editing and review the manuscript
SS:Editing and review the manuscript
ZYA:Final approval of the version to be published

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