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CASE REPORT

BURKHOLDERIA SEPTICAEMIA IN A CHILD WITH ATRIAL SEPTAL DEFECT AND PULMONARY HYPERTENSION: A RARE CLINICAL ASSOCIATION

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ABSTRACT

A congenital cardiac condition known as atrial septal defect (ASD) may not cause any symptoms until adulthood. It can cause serious problems, such as pulmonary arterial hypertension (PAH), which greatly deteriorates prognosis if treatment is not received. In order to prevent irreversible pulmonary vascular disease, early detection and prompt intervention are essential. We describe the case of a 7-year-old girl who had coryza, fever, exhaustion, and progressive dyspnoea. She had previously visited the hospital several times due to LRTI, and a local general practitioner recommended that a 2D-echo be performed. The results suggested that she had "large OS-ASD (20X21) mm, mild RV systolic dysfunction, mild mitral regurgitation, mild tricuspid regurgitation, and moderate pulmonary arterial hypertension." Patient was started on inj. Lasix and total fluid input and urine output was monitored. The patient was shifted for Paediatric- ICU for hourly monitoring and since was having moderate PAH, a blood culture was done, result positive for Burkholderia cepacia infection and CONS Patient was thus started on inj. Ceftazidime for 11 days.

KEYWORDS

Atrial Septal Defect, Pulmonary Arterial Hypertension, Congenital Heart Disease, Right Heart Catheterization, Burkholderia cepacia infection.

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INTRODUCTION

About 10% of adult congenital cardiac anomalies are caused by atrial septal defects (ASD), one of the most prevalent congenital heart defects. The secundum type is the most common of the different types of ASD. Larger ASDs can cause severe left-to-right shunting, which can result in chronic volume overload of the right heart chambers and pulmonary circulation, whereas smaller ASDs may remain asymptomatic and undetected until later in life. Persistent shunting can eventually result in pulmonary arterial hypertension (PAH) due to increased pulmonary blood flow and vascular remodelling. The coexistence of ASD with PAH presents a diagnostic and therapeutic challenge, as the development of elevated pulmonary vascular resistance (PVR) may render surgical or percutaneous closure of the defect high-risk or even contraindicated in certain scenarios, particularly when Eisenmenger physiology has developed. The following are risk factors for ASD with pulmonary hypertension: (1) Large or untreated ASD (2) Prolonged left-to-right shunt (which gradually increases pulmonary blood flow); (3) Female sex; (4) Genetic factors (e.g., BMPR2 mutations in idiopathic PAH can overlap with congenital heart disease). (5) underlying connective tissue diseases, such as lupus and systemic sclerosis (6) Coexisting pulmonary conditions, such as COPD or interstitial lung disease. The main risk of infection for people with ASD is infectious endocarditis (IE), particularly if the defect is large. A very uncommon type of infective endocarditis (IE) that usually affects vulnerable populations is an atrial septal defect (ASD) infected with Burkholderia. Treatment for Burkholderia is difficult because the bacterium is resistant to many drugs.

PATIENT INFORMATION

A 7- year old female, hindu by religion, resident of Wakad, Taluka Niphad is a child of 1st birth order of a Grade 3 consanguineous marriage, brought to the OPD by her father on Aug 29, 2025. The child's father reported c/o progressive dyspnoea, fatigue, fever and coryza since 2days. She was apparently alright 2 days back when she developed a low grade- intermittent type of fever, acute in onset associated with an acute- intermittent type of cough and coryza. It was not associated with burning micturition, loose stools, vomiting, headache, blurring of vision, yellowish discoloration of eyes. Cough not associated with expectoration, post- tussive vomiting. There was 1 episode of vomiting present, non- projectile type, containing undigested food and blood. There is no significant family history of any congenital heart disorder(s) or no known exposure of mother to toxins during pregnancy.

CLINICAL PRESENTATION

On systemic examination, she had a precordial bulge, trachea midline, apex beat at 5th Inter-costal space with thrill present at mitral area. S1 S2 heard, with S2 loud and grade 3 systolic murmur present . Increased JVP and parasternal heave present.

ANTHROPOMETRY

Parameter	Observed	Expected	Inference
Weight	14.5	24.3	<-3 SD
Height	109.4	125.1	-2 to -3 SD
BMI	12.2	15.6	-2 TO -3 SD

ON EXAMINATION

Vitals on Admission:

Temp:98.6 F

Heart rate:118 BPM

Respiratory rate:47 CPM

SP02: 99 % in room air

Blood pressure: 100/60 MMHG

Child is active, conscious, coherent and active

Cardio-Vascular System examination

Inspection

precordial bulge present

trachea in midline

apex beat seen at appro 4-5 th intercoastal space

no other visible pulsations seen

Palpation

no local rise of temperature:

no local swelling trachea in midline

apex beat is present at 5th intercostal space

thrill is present at the mitral area

Auscultation

mitral- s1 heard, loud systolic murmur grade 3

Respiratory examination

Inspection

Trachea midline
B/ l chest not symmetrical bulge present on left side
No engorging veins
No pallor, icterus, cyanosis, edema, lymphadenopathy: absent
No local rise of temperature
No tenderness
Visible pulsations present at midline

Percussion

AREAS	RIGHT	LEFT
Supraclavicular	Resonant	Resonant
Mammary	Resonant	Resonant
Infra-mammary	Resonant	Resonant
Supra-mammary	Resonant	Resonant
Axillary	Resonant	Resonant
Infra-axillary	Resonant	Resonant

Auscultation

B/L air entry present, reduced crepitation present on both sides

P/a: soft, splenomegaly present

CNS: Conscious Oriented

Activity NORMAL

Tone: NORMAL

Power: NORMAL

Reflexes: NORMAL

COURSE IN THE HOSPITAL/THERAPEUTIC INTERVENTION:

The child was admitted in view of fever, cough and cold for 2 days. On examination patient had precordial bulge, trachea midline apex beat seen at 5th intercostal space, thrill present at mitral area. S1 present, S2 loud, grade 3 systolic murmur. Patient was shifted to PICU from ward. Counts were done suggestive of HB-11.6, TLC-12.94, Platelet-257, CRP- 32. Was started on injection Augmentin and was given for 5 days. Patient was also started on tab. sildenafil 75 mg and is continued till discharge. 2-D Echo was done suggestive of large-OS-ASD 20x21 mm. Mild right ventricular systolic dysfunction, mild tricuspid regurgitation, mild mitral regurgitation, moderate pulmonary arterial hypertension. After this patient was started on injection Lasix and total fluid input and urine output was monitored. Patient had complaints of cough so syrup mucolyte was started and was given

for 5 days. Patient had moderate pulmonary arterial hypertension so was started on tab. envas. Patients' blood culture was positive for Burkholderia cepacia and cons so inj. certazidime was started and was given for 11 days. Patient needs cardiac surgery hence referring this patient for cardiac surgery to higher center.

DIAGNOSIS

The findings are in accordance with atrial septal defect with pulmonary arterial hypertension with blood culture result positive for Burkholderia cepacia infection and CONS

INFORMED CONSENT

The patient's father was first informed about the study, and then informed consent was obtained

DISCUSSION

This case demonstrates the intricate relationship between pulmonary hypertension and atrial septal defect. In patients with atrial septal defects, the development of pulmonary arterial hypertension frequently indicates a delayed diagnosis or delayed treatment, which may raise concerns about the viability and safety of defect closure (5). A comprehensive haemodynamic evaluation is necessary to direct the course of treatment. Improved clinical outcomes may result from the combination of targeted pulmonary arterial hypertension therapy and atrial septal defect closure in certain situations. Certain conditions, such as large defects, coexisting structural abnormalities, or the presence of bacteremia from dental, skin, or surgical sources, can cause infective endocarditis in patients with ASDs (6). In this instance, Burkholderia species, a rare and extremely resistant gram-negative pathogen associated with healthcare settings and immunocompromised states, caused infective endocarditis in a patient with a known ASD (7). Because of their innate resistance to antibiotics and capacity to create biofilms on endovascular surfaces, Burkholderia infections are difficult to treat (8).

CONCLUSION:

Diagnosing and treating ASD with concomitant pulmonary hypertension is difficult. This case demonstrates an uncommon but clinically significant case of infective endocarditis in a patient with an untreated atrial septal defect, complicated by infection with Burkholderia species. For positive results, early detection, a suitable microbiological diagnosis, and focused antimicrobial treatment are essential. Even in the absence of typical risk factors, clinicians should keep a high index of suspicion for endocarditis in patients with congenital heart disease who present with persistent fever. They should also take atypical organisms into consideration, particularly in the context of exposures related to healthcare.

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