Case Study

International Journal of Current Innovations in Advanced Research

Content Available at www.ijciar.com ISSN (O) 2636-6282 ISSN (P) 2659-1553

Case study on Eisenmenger Syndrome

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Received: 11 Aug 2021 Revised: 05 Sept 2021 Accepted: 25 Oct 2021

Abstract

The most severe type of pulmonary arterial hypertension, known as Eisenmenger syndrome, is caused by congenital heart disease with systemic-to-pulmonary shunt. Patients with Eisenmenger syndrome experience a complex and multisystemic disorder that includes coagulation disorders (bleeding complications and paradoxical embolisms), renal dysfunction, hypertrophic osteoarthropathy, heart failure, reduced quality of life, and premature death due to the chronic slow progressive hypoxemia with central cyanosis. The present case of 31-year-old female patient who was admitted in the cardiology department of a tertiary care teaching hospital with complaints of shortness of breath, increased excretion, cough with mucoid expectoration and discolouration of eye, tongue and fingers. Treatment includes antibacterial, diuretics, mucolytic agents, phosphodiesterase (PDE) inhibitors. After the continuous treatment for a period of 7 days the patient was found to be normal with no major complaints and so, the pharmacist when performed discharge counselling and patient was discharged with a 2week drug regimen after discharge.

Keywords: Pulmonary arterial hypertension, renal dysfunction, phosphodiesterase (PDE) inhibitors.

Introduction

Eisenmenger syndrome results from untreated congenital cardiac defects featuring intracardiac communication, leading to pulmonary hypertension, reversed blood flow, and cyanosis. The initial left-to-right shunt transforms into a right-to-left shunt due to elevated pulmonary artery pressures and associated vascular disease [1]. This syndrome, often arising from large septal defects, signifies a stage where pulmonary hypertension becomes irreversible, indicating that surgical intervention is likely infeasible. Late complications involve cardiac arrhythmias and sudden cardiac death. Conservative management involving medications or lung and cardiac transplantation can enhance the quality of life for individuals with Eisenmenger syndrome [2]. It is most commonly found in children and young adults with an untreated ventricular septal defect (VSD).

Congenital Heart Disease Subtypes Contributing to Eisenmenger Syndrome

Increased pulmonary arterial flow: Atrial septal defects (ASD), systemic arteriovenous fistulae, total anomalous pulmonary venous return.

Increased pulmonary arterial pressure and flow: Large ventricular septal defect (VSD), large patent ductus arteriosus (PDA), truncus arteriosus, single ventricle with unobstructed pulmonary blood flow.

Elevated pulmonary venous pressure: Mitral stenosis, cortriatriatum, obstructed pulmonary venous return [3].

Epidemology

Eisenmenger syndrome typically arises before puberty or during adolescence and early adulthood, affecting around 8% of patients with congenital heart disease. While symptoms limit the quality of life, individuals can survive into their third and fourth decades. The prevalence of Eisenmenger syndrome is declining in developed countries due to improved identification and surgical correction of congenital heart conditions, with late presentations more common in underdeveloped regions [4]. The likelihood of pulmonary hypertension and reversed shunting varies depending on the

specific heart defect and surgical intervention. For instance, large ventricular septal defects or patent ductus arteriosus can lead to pulmonary hypertension in about 50% of infants, while common atrioventricular canal patients almost universally develop severe pulmonary hypertension by the second year of life. Surgical procedures, such as the Blalock-Taussig anastomosis, may result in pulmonary hypertension in approximately 10% of cases, and palliative procedures like Waterston or Potts shunts lead to pulmonary hypertension in about 30% of patients [5].

Signs and symptoms

- Angina
- Arrhythmias
- Clubbing
- Coughing up blood.
- Cyanosis
- Dizziness or fainting.
- Oedema
- Heart palpitations
- Lethargy
- Dyspnoea

Causes /Aetiology

Eisenmenger Syndrome is primarily caused by untreated congenital heart defects such as ventricular septal defects (VSD), patent ductus arteriosus (PDA), atrial septal defects (ASD), atrioventricular septal defects (AVSD), and truncus arteriosus. These defects create abnormal blood flow in the heart, leading to increased blood flow to the lungs and eventual damage, causing pulmonary hypertension. Over time, irreversible pulmonary hypertension develops, converting the initial left-to-right shunt into a right-to-left shunt, resulting in cyanosis. The progression is influenced by defect size and nature. Early detection and intervention to correct these defects are essential in preventing Eisenmenger Syndrome.

Complications

- Brain abscess.
- Transient cerebral ischemia.
- Paradoxical embolism.
- Thrombotic stroke.
- Intracerebral haemorrhage.
- Hyperbilirubinemia.
- Hyperuricemia
- Hypertrophic osteoarthropathy.
- Dysrhythmia.
- Pulmonary infarction and haemorrhage.
- Infective endocarditis.
- Risk of sudden death [6].

Case Study

A 31-year-old female patient was admitted in the cardiology department with the complaints of shortness of breath, increased excretion,cough since 3 days with mucoid expectoration and discolouration of eye,tongue and fingers. She has a past medical history of large ostium seconded with Eisenmenger syndrome with left respiratory tract infection and on diuretics andphosphodiesterase (PDE) inhibitors medication and having family history of cardiac arrest.

Results

Table: 01 Blood Pressure,pulse rate and respiratory rate data:

Days	D1	D2	D3	D4	D5	D6	D7
BP(mm hg)	150/100	120/90	110/70	100/70	110/70	120/90	130/90
PR(bpm)	110	112	95	110	90	90	93
RR(bpm)	28	29	28	28	24	25	22
O_2 saturation	81	91	91	80	85	84	90

Table 02: Complete blood picture

Haemoglobin (11-16)	18.2 g	15.3 g
WBC(4000-11000)	12000 Cells/cumm	11000 cells/cumm

Table 03: Biochemical investigations

Serum creatinine (0.6-1.4)	0.6mg/dl	1mg/dl
Blood urea (14-45)	30mg/dl	28mg/dl

Table 04: Lipid profile tests

Total cholesterol(140-250)	87mg/dl	95mg/dl
Triglycerides (25-160)	70mg/dl	85mg/dl
HDL(30-165)	60mg/dl	65mg/dl
LDL(80-180)	83mg/dl	98mg/dl
VLDL(5-45)	27mg/dl	25mg/dl

Table 05: Serum electrolytes

Serum sodium(135-155)	135meq/L
Serum potassium(3.6-5.5)	4meq/L

Table: 03Drug chart

S.no	Trade name	Generic name	Dose	Route	frequency	D1	D2,3	D4	D5,6	D7
1.	T.Lasix	Furosimide	20mg	Po	Bd	**	**	**	**	**
2.	T.Sildenafil	Sildenafil	20mg	Po	Bd	**	**	**	**	**
3.	T.Pantop	Pantoprazole	40mg	Po	Bd	**	**	**	**	**
4.	T.Aldactone	Siprinolactone	25mg	Po	Od				*	*
5.	Syp.Ambroxol	Ambroxol	10ml	Po	Od	*	*	*	*	
6.	T.Amoxiclav	Amoxillin+ Calvulinic acid	625mg	Po	Tid	***	***	***		
7.	T.Azitral	Azithromycin	500mg	Po	Od	*	*	*	*	*

Discussion

The case study illustrates successful multidisciplinary management of Eisenmenger syndrome through a seven-day treatment regimen, emphasizing early detection and collaborative healthcare. The positive outcome highlights the effectiveness of pharmacological interventions, particularly phosphodiesterase inhibitors, in improving symptoms and

quality of life. The discussion covers the syndrome's complex nature, including coagulation disorders and renal dysfunction, along with its epidemiology and impact on different congenital heart disease subtypes. Overall, the study provides insights into treatment strategies and underscores the importance of prompt medical care in optimizing outcomes for individuals with Eisenmenger syndrome.

Conclusion

The case study illuminates the successful management of Eisenmenger syndrome through a comprehensive, multidisciplinary treatment approach involving antibacterial agents, diuretics, mucolytic agents, and phosphodiesterase inhibitors. Early detection and timely intervention were pivotal in achieving positive outcomes, emphasizing the critical role of prompt medical care. The effectiveness of phosphodiesterase inhibitors in improving pulmonary circulation underscores their significance in treating Eisenmenger syndrome. The case underscores the ongoing need for therapeutic advancements and a deeper understanding of the condition's pathophysiology to enhance patient quality of life. Collaboration among healthcare professionals is integral in navigating the complexities of Eisenmenger syndrome. The positive outcome reflects strides in improving prognosis and management, offering hope and an enhanced life quality for affected individuals.

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