



Assessment of clinical profile of pulmonary hypertension in patients identified with the chronic kidney disease

Dr. Abhay Kumar¹, Dr. Govind Prasad^{2*}, Dr. Om Kumar³

^{1,2} Senior Resident, Department of Nephrology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India

³ Professor & Head of Department, Department of Nephrology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India.

Abstract

Pulmonary hypertension (PH) has been commonly found in patients undergoing renal replacement with haemodialysis via an arterio-venous graft or fistula, and is characterized by elevations in the pulmonary arterial pressure and pulmonary vascular resistance (PVR) that eventually result in right ventricular failure and even premature death. Chronic renal failure (CRF) is most often seen with patients with PH.

Hence based on above observations this study was planned to know the prevalence of PH in patients who are on conservative management, haemodialysis or continuous ambulatory peritoneal dialysis.

The study was conducted on 80 patients diagnosed with chronic kidney disease and on conservative or maintenance haemodialysis were included in the present study. The patients above 12 years of age and suffering from chronic kidney disease on dialysis were included in the study. The pregnant females and patients having other diseases were excluded from the study.

From the above data it can be concluded that Pulmonary hypertension is positively correlated with clinical parameters like RRT-haemodialysis, A-V fistula, duration of dialysis and biochemical parameters like low haemoglobin, low sr. bicarbonate, high levels of uric acid, BUN, creatinine.

Keywords: CKD with PH, haemodialysis, Prevalence study, pulmonary arterial pressure etc.

Introduction

Pulmonary hypertension is a type of high blood pressure that affects the arteries in lungs and the right side of the heart. In one form of pulmonary hypertension, tiny arteries in the lungs, called pulmonary arterioles, and capillaries become narrowed, blocked or destroyed. This makes it harder for blood to flow through the lungs, and raises pressure within the lungs' arteries. As the pressure builds, the heart's lower right chamber (right ventricle) must work harder to pump blood through the lungs, eventually causing the heart muscle to weaken and fail.

Pulmonary hypertension (PH or PHTN) is a condition of increased blood pressure within the arteries of the lungs. Symptoms include shortness of breath, syncope, tiredness, chest pain, swelling of the legs, and a fast heartbeat. The condition may make it difficult to exercise. Onset is typically gradual. The cause is often unknown. Risk factors include a family history, prior blood clots in the lungs, HIV/AIDS, sickle cell disease, cocaine use, COPD, sleep apnea, living at high altitudes, and problems with the mitral valve. The underlying mechanism typically involves inflammation of the arteries in the lungs. Diagnosis involves first ruling out other potential causes [1]. Treatment depends on the type of disease. A number of supportive measures such as oxygen therapy, diuretics, and medications to inhibit clotting may be used. Medications specifically for the condition include epoprostenol, treprostinil, iloprost, bosentan, ambrisentan, macitentan, and sildenafil. A lung transplant may be an option in certain cases [2].

While the exact frequency of the condition is unknown, it is

estimated that about 1,000 new cases occur a year in the United States. Females are more often affected than males. Onset is typically between 20 and 60 years of age. It was first identified by Ernst von Romberg in 1891 [3].

Treatment of pulmonary hypertension is determined by whether the PH is arterial, venous, hypoxic, thromboembolic, or miscellaneous. If it is caused by left heart disease, the treatment is to optimize left ventricular function by the use of medication or to repair/replace the mitral valve or aortic valve. Patients with left heart failure or hypoxemic lung diseases (groups II or III pulmonary hypertension) should not routinely be treated with vasoactive agents including prostanoids, phosphodiesterase inhibitors, or endothelin antagonists, as these are approved for the different condition called primary pulmonary arterial hypertension. To make the distinction, doctors at a minimum will conduct cardiac catheterization of the right heart, echocardiography, chest CT, a six-minute walk test, and pulmonary function testing. Using treatments for other kinds of pulmonary hypertension in patients with these conditions can harm the patient and wastes substantial medical resources [4].

High dose calcium channel blockers are useful in only 5% of IPAH patients who are vasoreactive. Unfortunately, calcium channel blockers have been largely misused, being prescribed to many patients with non-vasoreactive PAH, leading to excess morbidity and mortality. The criteria for vasoreactivity have changed. Only those patients whose mean pulmonary artery pressure falls by more than 10 mm Hg to less than 40 mm Hg with an unchanged or increased cardiac output when challenged with adenosine, epoprostenol, or nitric oxide are

considered vasoreactive. Of these, only half of the patients are responsive to calcium channel blockers in the long term [5].

Chronic kidney disease (CKD) is a type of kidney disease in which there is gradual loss of kidney function over a period of months or years. Early on there are typically no symptoms. Later, leg swelling, feeling tired, vomiting, loss of appetite, or confusion may develop. Complications may include heart disease, high blood pressure, bone disease, or anemia. [6].

Causes of chronic kidney disease include diabetes, high blood pressure, glomerulonephritis, and polycystic kidney disease. Risk factors include a family history of the condition. Diagnosis is generally by blood tests to measure the glomerular filtration rate and urine tests to measure albumin. Further tests such as an ultrasound or kidney biopsy may be done to determine the underlying cause. A number of different classification systems exist. [7].

Screening at-risk people is recommended. Initial treatments may include medications to manage blood pressure, blood sugar, and lower cholesterol. NSAIDs should be avoided. Other recommended measures include staying active and certain dietary changes. Severe disease may require hemodialysis, peritoneal dialysis, or a kidney transplant. Treatments for anemia and bone disease may also be required [8].

PH has been commonly found in patients undergoing renal replacement with haemodialysis via an arterio-venous graft or fistula, and is characterized by elevations in the pulmonary arterial pressure and pulmonary vascular resistance (PVR) that eventually result in right ventricular failure and even

premature death. Chronic renal failure (CRF) is most often seen with patients with PH [9]. Chronic haemodialysis patients are continuously exposed to multifactorial pulmonary insults. Hence based on above observations this study was planned to know the prevalence of PH in patients who are on conservative management, haemodialysis or continuous ambulatory peritoneal dialysis.

Methodology

The study was planned in IGIMS to assess the prevalence of pulmonary hypertension in patients suffering from chronic kidney disease. The patients visited to Out Patient Department (OPD) and in-patient department (IPD) of IGIMS were considered in the study. All the patients were informed consents. The entire patient's clinical history was collected.

The study was conducted on 80 patients diagnosed with the chronic kidney disease and on conservative or maintenance haemodialysis were included in the present study.

The patients above 12 years of age and suffering from chronic kidney disease or on dialysis were included in the study. The pregnant females and patients having other diseases were excluded from the study.

Results and discussion

The data from the total 80 patients diagnosed with chronic kidney disease and on conservative or maintenance haemodialysis were collected and presented as below. The table 1 indicates the number of chronic kidney disease patients with the treatment type.

Table 1: Number of chronic kidney disease patients with the treatment type

Treatment Type	No. of Chronic Kidney Disease Patients	No. of Chronic Kidney Disease Patients with Pulmonary Hypertension
Maintenance Haemodialysis	60	21
Conservative Treatment	20	6

Table 2: Biochemical Parameters in Chronic Kidney Disease Patients with Pulmonary Hypertension

Sr No.	Biochemical parameter (units)	With Pulmonary Hypertension	Without Pulmonary Hypertension
	Number of Cases	27	53
1	Haemoglobin (g %)	6.7-9.1	7.3-9.6
2	Uric Acid (mg %)	4.8-8.1	4.5-7.4
3	Serum Bicarbonate (mmol/L)	6.7-10.5	9.2-12.8
4	Blood urea nitrogen (BUN) (mg %)	45 – 103	34-95
5	Serum Creatinine (mg %)	5.7-11.6	4.5-11.2

Our study revealed a positive association between the duration of dialysis and the prevalence of PH. A similar study by Patel *et al* [10], showed that as duration of renal failure increased so did the chance of developing PH.

PH is positively correlated with clinical parameters like RRT-haemodialysis, A-V fistula, duration of dialysis and biochemical parameters like low haemoglobin, low sr. bicarbonate, high levels of uric acid, BUN, creatinine.

Our study result demonstrated high mean Haemoglobin indicating a role of anaemia in the pathogenesis of PH in CKD. This correlation is supported by C. J. Rhodes *et al*. [11]. Showing the role of iron in the natural history of PH. Iron availability influences the pulmonary vasoconstrictor response to hypoxia and accumulating evidence indicates that iron

deficiency is prevalent in idiopathic and heritable forms of PH. The mean serum uric acid level found in our study is supported by Norotoshi Nagaya *et al*. [12]. Showing that serum UA increases in proportion to the clinical severity of PH and has independent association with long-term mortality of patients with Primary PH indicating it as a predictor of morbidity & mortality in PH.

The findings of mean serum bicarbonate levels are comparable to the study done by Patel *et al*. [10] and indicate a positive correlation between PH and low bicarbonate level, leading to metabolic acidosis. The results of BUN & serum creatinine levels in our study are comparable to that of Patel *et al*. [10]. Indicating PH in CKD and correlates with high levels of both.

Conclusion

From the above data it can be concluded that Pulmonary hypertension is positively correlated with clinical parameters like RRT-haemodialysis, A-V fistula, duration of dialysis and biochemical parameters like low haemoglobin, low sr. bicarbonate, high levels of uric acid, BUN, creatinine.

References

1. Pulmonary Arterial Hypertension – NORD (National Organization for Rare Disorders). NORD. 2015. Archived from the original on, 2017. Retrieved 30 July 2017.
2. How Is Pulmonary Hypertension Treated? – NHLBI, NIH. NHLBI. 2011. Archived from the original on 2017. Retrieved 30 July 2017.
3. Von Romberg, Ernst. ÜberSklerose der Lungenarterie. Dtsch Arch Klin Med (in German). 1891-1892; 48:197-206.
4. American College of Chest Physicians; American Thoracic Society Five Things Physicians and Patients Should Question, Choosing Wisely: an initiative of the ABIM Foundation, American College of Chest Physicians and American Thoracic Society, archived from the original on, 2013. Retrieved 6 January 2013, which cites.
5. Sitbon O, Humbert M, Jaïs X, Ioos V, Hamid AM, Provencher S, *et al.* Long-term response to calcium channel blockers in idiopathic pulmonary arterial hypertension. *Circulation*. 2005; 111(23):3105-11. doi:10.1161/CIRCULATIONAHA.104.488486.PMID 15939821.
6. Kidney Failure. Medline Plus. Retrieved, 2017.
7. Summary of Recommendation Statements. *Kidney International Supplement*. 2013 3(1):5-14. doi:10.1038/kisup.2012.77.
8. Anemia in Chronic Kidney Disease. National Institute of Diabetes and Digestive and Kidney Diseases, 2016. Retrieved 19 December 2017.
9. Yigla M, Azzam Z, Rubin AHE, *et al.* Background disease in 714 patients with moderate to severe pulmonary hypertension. *Med Assoc J*. 2000; 2:501-506.
10. Patel P, *et al.* Clinical and biochemical parameter in chronic kidney disease. *Indian journal of nephrology*. 2007; (17):1. [IP: 202.177.228.90]
11. Rhodes CJ, Wharton J, Howard L, Gibbs JS, Vonk-Noordegraaf A, Wilkins MR. Iron deficiency in pulmonary arterial hypertension: a potential therapeutic target. *Eur. Respir J*. 2011; 38(6):1453-60.
12. Nagaya N, Uematsu M, Satoh T, *et al.* Serum uric acid levels correlate with the severity and the mortality of primary pulmonary hypertension. *Am J Respir Crit. Care Med*. 1999; 160(2):487-92.