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AN OBSERVATIONAL STUDY ON TYPE AND AETIOLOGY OF PLEURAL EFFUSION IN CHRONIC KIDNEY DISEASE

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Abstract

Introduction: Chronic kidney disease (CKD) encompasses a spectrum of different pathophysiologic processes associated with abnormal kidney function and a progressive decline in glomerular filtration rate (GFR). CKD resulted in 1·2 million deaths in 2017. An additional 1·4 million deaths from cardiovascular disease were attributable to impaired kidney function, representing 7·6% of deaths due to cardiovascular disease in 2017. Ranked as the 17th leading cause of death in 1990, CKD has increased in importance, ranking as the 12th leading cause of death in 2017.

Materials and Methods: CKD resulted in 1·2 million deaths in 2017. An additional 1·4 million deaths from cardiovascular disease were attributable to impaired kidney function, representing 7·6% of deaths due to cardiovascular disease in 2017. Ranked as the 17th leading cause of death in 1990, CKD has increased in importance, ranking as the 12th leading cause of death in 2017. A detailed history and physical examination was carried out. Thoracentesis was done after confirmation by clinical and radiological examination of the site. USG guided pleural fluid aspiration was done whenever required. In suspected cases of exudative effusion, pleural biopsy was performed by Cope's needle.

Results: In this study of 76 patients, there were 52 patients (68.4%) with exudative effusion, and 24 patients (31.6%) had transudative effusion. The three commonest causes of pleural effusion in our study were, cardiac failure in 22 28.9%) and uremia and tuberculosis in 14 patients (18.4%) each. Malignancy, pneumonia and hypoalbuminemia were responsible for pleural effusion in 12 (15.78%), 8 (10.52%) and 2 (2.6%) cases. The aetiology remained unknown in four (5.26%) cases. As is apparent, the most common cause of pleural effusion in CKD in this study was cardiac failure, followed by uremia, tuberculosis and malignancy.

Conclusion: Our study had majority of exudative effusion, where uremic and tubercular effusion were most common etiology among exudative effusion. Cardiac failure was the most common etiological and transudative cause of effusion.

Key Words: Chronic kidney disease, glomerular filtration rate

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INTRODUCTION

Chronic kidney disease (CKD) encompasses a spectrum of different pathophysiologic processes associated with abnormal kidney function and a progressive decline in glomerular filtration rate (GFR). In 2017 globally, there were 697·5 million cases of CKD. Almost a third of patients with CKD lived in two countries, China (132·3 million) and India (115·1 million cases). Bangladesh, Brazil, Indonesia, Japan, Mexico, Nigeria, Pakistan, Russia, the USA, and Vietnam had more than 10 million cases of CKD each. 79 of 195 countries included in Global Burden Disease had more than 1 million prevalent cases of CKD in 2017. In 2017, the prevalence of CKD was estimated as 9·1% in the world's population, with CKD stages 1–2 accounting for 5·0%, stage 3 for 3·9%, stage 4 for 0·16%, stage 5 for 0·07%, dialysis for 0·041%, and kidney transplantation for 0·011%. The age-standardised prevalence of CKD was 1·29 times higher in females than in males (7·3%). The global age-standardised incidence of dialysis and transplantation was 1·47 times greater among males (13·7 per 100000 population) than among females (8·6 per 100000 population). The global age-standardised CKD mortality rate was 1·39 times greater among males (18·9 per 100000 population) than among females (13·6 per 100000 population). The country median prevalence of CKD was 8·9%. The global age-standardised prevalence of CKD has remained stable between 1990 and 2017, with a 1·2% change, although the all-age prevalence of CKD has increased by 29·3% from 1990 to 2017. The availability of renal replacement therapy from 1990 to 2017 has grown; global all-age incidence of dialysis and kidney transplantation increased by 43·1% and 34·4%, respectively.[1]

CKD resulted in 1·2 million deaths in 2017. An additional 1·4 million deaths from cardiovascular disease were attributable to impaired kidney function, representing 7·6% of deaths due to cardiovascular disease in 2017. Ranked as the 17th leading cause of death in 1990, CKD has increased in importance, ranking as the 12th leading cause of death in 2017. The rank of CKD among all causes of death was especially prominent in Central and Andean Latin America, ranking second and fifth, respectively. The global all-age CKD mortality rate increased by 41·5% from 1990 to 2017. The agestandardised mortality rate remained stable, with a 2·8% change from 1990 to 2017. The agestandardised CKD mortality rate, however, has increased by 60·9% in central Latin America, 60·9% in central Asia, and 57·3% in high-income North America.[2]

In India, given its population of more than one billion, the rising incidence of CKD is likely to pose major problems for both healthcare and the economy in future years. Indeed, it has been recently estimated that the age-adjusted incidence rate of ESRD in India to be 229 per million population (pmp), and more than 100,000 new patients enter renal replacement programs annually in India. Ironically, only 10% of these ESRD patients receive any renal replacement therapy (RRT) because of scarce resources.[3]

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The prevalence of CKD in India cohort is 17.2%. The prevalence of CKD stages 1, 2, 3, 4 and 5 was 7%, 4.3%, 4.3%, 0.8% and 0.8%, respectively. Hypertension, anemia and diabetes were the most common risk factors and associated characteristics associated with CKD.[4]

Patients of chronic kidney disease have alterations in the host immune response. In these patients, the function of polymorpho nuclear white blood cells, lymphocytes, and monocytes is altered, resulting in an impaired host response to infection. Malnutrition, increased intracellular calcium, iron overload, dialysis membranes, and uremic toxins (*i.e.*, circulating factors that inhibit granulocytes) contribute to impaired polymorph nuclear leukocyte function. In the setting of kidney failure, T lymphocyte, monocyte, and monocyte-derived dendritic cell function is also impaired.

Numerous risk factors predispose patients with CKD and ESRD to infection. Potential risk factors for infection among patients with CKD or ESRD include advanced age, high burden of coexisting illnesses, hypoalbuminemia, immunosuppressive therapy), nephrotic syndrome, uremia, anemia, and malnutrition. Once maintenance dialysis is initiated, additional potential risk factors for infection include vascular access used for dialysis, the dialysis procedure itself, and iron overload.

Because of altered impaired host response the incidence of the commonly seen infectious complications is approximately three times greater among CKD patients who have not yet initiated dialysis than in the general population, with UTI, pneumonia, and sepsis in descending order of prevalence. The higher UTI susceptibility in the CKD group may be explained, in part, by a greater incidence of urinary obstructions, which in turn leads to infectionscommonly seen in those with benign prostatic hypertrophy, kidney stones, and urinary tract cancers. Patients with ESRD treated by dialysis have higher annual mortality rates caused by sepsis compared with the general population, even after stratification for age, race, and diabetes.Overall, the annual percentage of mortality secondary to sepsis is approximately 100- to 300-fold higher in dialysis patients.

Compared with the non-CKD population, the rates of pneumonia are 3 times greater in the CKD population and 5 times greater in the dialysis population. The length of hospital stays for pneumonia in the CKD and dialysis populations are very similar and 4 to 6 times longer than those in the non-CKD population. In fact, pneumonia as a complication in the CKD population appears to be more severe than previously appreciated. Bacteremia/sepsis patterns are also quite different when comparing the non-CKD, CKD, and dialysis populations. CVD represents the leading cause of mortality in CKD patients, and the prevalence and burden of this complication increases with declining kidney function.

The heart and the vascular tree undergo major structural and functional changes when kidney function declines and renal replacement therapy is required. The heart undergoes adaptive changes that include left ventricular hypertrophy and dilatation with concomitant systolic and diastolic dysfunction. Myocardial fibrosis is the consequence of impaired angio-adaptation, reduced capillary angiogenesis, myocyte-capillary mismatch, and myocardial micro-

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arteriopathy. The vascular tree can be affected by both atherosclerosis and arteriosclerosis with both lipid rich plaques and abundant media calcification. The major cause of left ventricular hypertrophy and failure and the most common problem directly affecting myocardial function is fluid overload and, usually, hypertension. In situations of stress, such as intradialytic hypotension and hypoxaemia, the hearts of these patients are more vulnerable to developing cardiac arrest, especially when such episodes occur frequently. As a result, cardiac and vascular mortality are several times higher in dialysis patients than in the general population.

Pleural disease is a common problem in patients with chronic renal insufficiency. There are several reasons why pleural disease may be common in patients with chronic kidney disease. These include congestive heart failure, fluid overload, an increased risk of infection (Especially tuberculosis), the presence of diseases associated with renal and pleural manifestations (e.g., systemic lupus erythematosus). Uremic pleurisy results from an unknown putative agent, and therefore uremic pleuritis is a diagnosis of exclusion, that persists or recurs despite aggressive haemodialysis.

Ninety percent cases of pleural effusion in the western countries are reported to result from only five diseases: CCF, pneumonia, malignancy, pulmonary embolism, and viral infections. Twenty to forty percent of hospitalized patients with bacterial pneumonia develop pleural effusion. In India, unlike the western countries, tuberculous pleural effusion is common. The pleural cavity is involved in approximately 5% of all patients with tuberculosis, which is next only to lymph node tuberculosis.

As patients with CKD have immune dysfunction manifested by depressed cell-mediated immunity (CMI). Compromised immunity makes these patients prone forchronic infections like tuberculosis. Some studies have suggested that CKD patientsmay be at increased risk for certain malignancies, such as non-Hodgkin’s lymphoma and renal, prostate, and uterine Cancer. Increased risk for ischemic heart disease and potential for dilated cardiomyopathy makes these patients especially prone to problems with fluid balance leading to fluid overload.[5]

AIM AND OBJECTIVES

To study the type and aetiology of pleural effusion in Chronic Kidney Disease.

METHODOLOGY

The observational study was carried out at Pulmonary Medicine department of tertiary care hospital in western Maharashtra. Ethical committee approval was taken. Data of total 76 patients of CKD with pleural effusion was analysed.

Inclusion criteria: Adult patients of more than 18 years with CKD (stage 3 to 5) showing clinico-radiological evidence of pleural effusion.

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Exclusion criteria: Patients with age less than 18 years, patients having bleeding diathesis, patients on anticoagulant, acute renal failure, and patients not willing to participate in the study were excluded.

A detailed history and physical examination was carried out.Thoracentesis was done after confirmation by clinical and radiological examination of the site. USG guided pleural fluid aspiration was done whenever required.In suspected cases of exudative effusion, pleural biopsy was performed by Cope's needle.

Investigations done on pleural fluid were as follows:

1. Protein
2. LDH
3. ADA
4. Gram stain, Zn stain
5. Aerobic culture
6. Cytology
7. Total cells
8. Differential cells
9. AFB culture and sensitivity

Other investigations:

I. Serum protein
II. Serum LDH
III. Serum Pro- BNP
IV. Sputum for gram stain, Zn stain, aerobic culture
V. 2D ECHO
VI. CT Thorax
VII. Pleural biopsy

RESULTS

In this study of 76 patients, there were 52 patients (68.4%) with exudative effusion, and 24 patients (31.6%) had transudative effusion. The three commonest causes of pleural effusion in our study were, cardiac failure in 22 28.9%) and uremia and tuberculosis in 14 patients (18.4%) each. Malignancy, pneumonia and hypoalbuminemia were responsible for pleural effusion in 12 (15.78%), 8 (10.52%) and 2 (2.6%) cases. The aetiology remained unknown in four (5.26%) cases. As is apparent, the most common cause of pleural effusion in CKD in this study was cardiac failure, followed by uremia, tuberculosis and malignancy.

DISCUSSION

Type of Effusions:

In present study 52 patients (68.4%) had exudative effusion and 24 patients (31.6%) transudative. The similar findings were seen in study by Prem Kumar et al, where in

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transudative pleural effusions were found in 31.42% and exudative in 68.57%cases. However, in study by Kundu et al, exudates and transudates were found in equal frequencies. In a study Bakirci et al, 64.3% CKD patients on hemodialysis transudative effusion**.**6

In present study, the most common cause of exudative effusion was uraemia and tuberculosis.

Etiology of pleural fluid:

Cardiac Failure:

In present study, cardiac failure was seen in majority of the cases that is 22(28.9%). The study was comparable to the study conducted by Kundu et al, in which the commonest aetiology of pleural effusion was cardiac failure (13(41.9 %) out of 31 cases). [(35)]

Tubercular effusion:

In present study 14(18.4%) cases were of tubercular origin. Eight cases were diagnosed by pleural biopsy and remaining cases diagnosed by pleural fluid (lymphocytic predominance with increased ADA levels (>40 U/L).Kundu et al found 28.5% cases of tubercular effusion out of 31 cases with all unilateral effusion. Erkoc et al reported 30 tuberculosis patients among 287 dialysis patients and 65.4% of these cases had extra pulmonary TB. Only nine cases had tubercular effusion.

In another study by T. Manmadha Rao, R. Ram et al, out of 1237 patients on hemodialysis, 131 diagnosed as TB. Extra pulmonary (69 patients, 52.6%) was more than pulmonary TB (60 patients, 45.8%). in extra pulmonary TB, pleural effusion (N-31) was most common.[7]

Malignant pleural effusion:

In present study, malignant pleural effusion was found in 12(15.8%) cases. Out of which all were paramalignant effusion, primarily of lung.

In other study by Prem Kumar et al, malignant pleural effusion was present in 3(8.57 %) cases. Out of which 2 were paramalignant effusion one was due to malignant pleural mesothelioma.[8]

Parapneumonic effusion:

In present study parapneumonic effusions were found in 8(10.5%). In 3 cases klebsiella and in 1 case pseudomonas pathogens were isolated from pleural fluid.

In a similar study by Kundu et al, 2(6.5%) cases were found of parapneumonic effusion. Staphylococcus aureus and Pseudomonas aeruginosa were recovered on pleural fluid culture.

In a study by Guitti et al, in patients on hemodialysis 23.7% patients on hemodialysis had parapneumonic effusions and uremic effusion out of 76 patients on hemodialysis.[9]

Uremic effusion:

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Uremic effusion was found in 14(18.4%) cases which is one of the most common cause of exudative effusion. 2 patients were diagnosed by pleural biopsy. All patients were on hemodialysis.

In other study by Kundu et al, uremic effusion was found in 6(19.4%) out of 31cases.Bilateral effusion was present in 2 cases.

Present study was comparable to study by Bakirci et al, the most frequent cause of exudative effusion in CKD patients on hemodialysis was uremic effusion around 40%.[10]

Uremic effusion closely mimic to tubercular effusions. But latter differentiated from former etiology by doing all necessary investigations.

Unknown etiology:

In present study, 4(5.26%) cases were undiagnosed. Pleural fluid was suggestive of exudative, polymorphs predominance with normal ADA. Due to patient's financial problems, thoracoscopy was not possible.

Hypoalbuminemia:

2(2.6%) case of hypoalbuminemia was seen, which had bilateral effusion on chest x ray. Hypoalbuminemia was secondary to the underlying CKD. Patient was conservatively managed with parenteral albumin.

CONCLUSION

Our study had majority of exudative effusion, where uremic and tubercular effusion were most common etiology among exudative effusion. Cardiac failure was the most common etiological and transudative cause of effusion.

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