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A Study to Determine Causes, Clinical Profile, Hospital Course and Outcomes of Acute Pulmonary Oedema in Maintenance Hemodialysis Patients

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Abstract

Background: Acute pulmonary oedema (APO) is a life-threatening complication in patients on maintenance hemodialysis (MHD), representing a significant cause of morbidity, mortality, and healthcare resource utilisation. Understanding the precipitating causes, risk factors, and outcomes is crucial for improving patient care. ***Aim:*** To determine the precipitating causes, clinical profile, hospital course, and outcomes of acute pulmonary oedema in maintenance hemodialysis patients. ***Materials and Methods:*** This hospital-based, case-control study was conducted in the Department of General Medicine, Vyas Medical College and Hospital, Jodhpur. Group A (Control) comprised 25 patients on MHD for more than one year who had never been admitted with APO. Group B (Case) comprised 45 patients on MHD for at least 3 months who presented to the emergency department with signs and symptoms suggestive of APO. Detailed clinical, biochemical, and echocardiographic data were recorded. Outcomes including ICU stay, hospital stay, need for ventilator support, and in-hospital mortality were analyzed. ***Results:*** Diabetes mellitus was the most common etiology of CKD in both groups (Group A: 44%, Group B: 51.1%). Patients in Group B had significantly higher mean systolic blood pressure (168.4 ± 18.2 mmHg vs. 132.6 ± 12.4 mmHg, $p < 0.001$), higher mean weight gain between dialysis sessions (4.2 ± 1.1 kg vs. 1.8 ± 0.6 kg, $p < 0.001$), and lower mean serum albumin (2.9 ± 0.5 g/dL vs. 3.6 ± 0.4 g/dL, $p < 0.001$). Among Group B patients, 31.1% required invasive mechanical ventilation, with a mean ICU stay of 5.2 ± 2.4 days and mean hospital stay of 8.6 ± 3.8 days. Overall in-hospital mortality was 22.2%. Factors associated with mortality included higher APACHE II scores, lower serum albumin levels, and the need for mechanical ventilation. ***Conclusion:*** APO in MHD patients is predominantly precipitated by poor fluid compliance, missed dialysis sessions, and underlying cardiac dysfunction. High mortality and significant healthcare resource utilisation underscore the importance of preventive strategies, including patient education, optimisation of dry weight, and strict adherence to dialysis schedules.

Keywords: Acute Pulmonary Oedema, Maintenance Hemodialysis, End-Stage Renal Disease, Fluid Overload

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INTRODUCTION

End-stage renal disease (ESRD) is a growing public health problem worldwide, with a rising incidence in India due to increasing prevalence of diabetes mellitus and hypertension [1]. Maintenance hemodialysis (MHD) remains the primary modality of renal replacement therapy for the majority of ESRD patients in resource-limited settings. Despite advances in dialysis technology, cardiovascular complications remain the leading cause of morbidity and mortality in this population [2].

Acute pulmonary oedema (APO) is a critical complication in MHD patients, representing the pulmonary manifestation of severe fluid overload. It occurs when the heart fails to compensate for the volume expansion that accumulates between dialysis sessions, leading to increased pulmonary capillary hydrostatic pressure and transudation of fluid into the alveolar spaces [3]. APO is a medical emergency requiring immediate intervention, often necessitating intensive care unit (ICU) admission, mechanical ventilation, and emergent dialysis.

The pathophysiology of APO in MHD patients is multifactorial. Unlike patients with normal renal function, who develop pulmonary oedema primarily due to cardiac dysfunction, MHD patients have an additional volume-dependent component.

Key contributing factors include:

- Fluid non-compliance: Excessive inter-dialytic weight gain due to poor dietary salt and fluid restriction.

- Missed or inadequate dialysis: Reduced frequency or duration of dialysis sessions leading to progressive volume accumulation.
- Cardiac dysfunction: Underlying systolic or diastolic heart failure, which is highly prevalent in ESRD patients.
- Arteriovenous fistula: Increased preload due to high-flow AV access.
- Anemia: Reduced oxygen-carrying capacity exacerbating myocardial stress.
- Malnutrition: Hypoalbuminemia reducing plasma oncotic pressure and predisposing to oedema.

The hospital course of APO in MHD patients is often complicated by prolonged ICU stays, need for mechanical ventilation, and high mortality rates. Identifying precipitating causes and risk factors is essential for developing preventive strategies and improving outcomes [4].

This study was conducted at Vyas Medical College and Hospital, Jodhpur, to comprehensively evaluate the causes, clinical profile, hospital course, and outcomes of APO in MHD patients.

2. Objectives

1. To determine the precipitating causes of acute pulmonary oedema (APO) in patients on maintenance hemodialysis.
2. To determine the risk factors for development of APO.
3. To determine hospital course in terms of duration of ICU and hospital stay and need for ventilator support.
4. To determine outcome of APO in MHD patients in terms of discharge or death.

MATERIALS AND METHODS

3.1 Study Design and Setting

This was a hospital-based, observational, case-control study conducted in the Department of General Medicine, Vyas Medical College and Hospital, Jodhpur

3.2 Study Population

Subjects were recruited from patients undergoing maintenance hemodialysis at the Department of General Medicine, Vyas Medical College and Hospital, Jodhpur Hospital dialysis unit and those presenting to the emergency department.

3.3 Group Allocation

- Group A (Control Group): 25 patients who were on maintenance hemodialysis for more than one year but had never been admitted with acute pulmonary oedema. These patients were recruited from the outpatient dialysis unit during their routine dialysis sessions.
- Group B (Case Group): 45 patients who were on maintenance hemodialysis for at least 3 months and presented to the emergency department of Vyas Hospital with signs and symptoms suggestive of acute pulmonary oedema. Diagnosis of APO was confirmed by clinical examination (dyspnea, orthopnea, crackles on auscultation, frothy sputum) and chest X-ray findings (bilateral alveolar infiltrates, cardiomegaly, pleural effusion).

3.4 Inclusion Criteria

- Group A: Patients on MHD for more than 12 months, clinically stable, no history of APO admission.
- Group B: Patients on MHD for at least 3 months, admitted to hospital with clinical and radiological diagnosis of acute pulmonary oedema.

3.5 Exclusion Criteria

- Patients on hemodialysis for less than 3 months.
- Patients with acute kidney injury (AKI) requiring de novo dialysis.
- Patients on peritoneal dialysis.
- Patients with acute pulmonary oedema due to non-cardiac/fluid overload causes (e.g., neurogenic pulmonary oedema, aspiration pneumonia, ARDS).
- Patients with known malignancy or terminal illness.
- Pregnant women.

3.6 Data Collection

A detailed structured proforma was used to collect the following data:

Demographic and Clinical Data:

- Age, sex, duration on hemodialysis
- Etiology of chronic kidney disease
- Comorbidities: diabetes mellitus, hypertension, coronary artery disease, heart failure
- Dialysis parameters: frequency of dialysis, duration of each session, missed dialysis sessions in past month
- Inter-dialytic weight gain (average over last 3 months)
- Adherence to fluid and dietary restrictions (assessed by patient interview)

Presentation Data (Group B only):

- Presenting symptoms: dyspnea, orthopnea, paroxysmal nocturnal dyspnea, cough, frothy sputum
- Clinical signs: blood pressure, heart rate, respiratory rate, oxygen saturation, jugular venous pressure, crackles, oedema
- APACHE II score at admission

Laboratory Parameters:

- Complete blood count
- Serum creatinine, blood urea nitrogen
- Serum electrolytes (sodium, potassium, chloride)
- Serum albumin
- Arterial blood gas (ABG) analysis

- Cardiac biomarkers: Troponin I, NT-proBNP (where available)
- Hemoglobin, hematocrit

Echocardiographic Data:
- Left ventricular ejection fraction (LVEF)
- Diastolic dysfunction grade
- Pulmonary artery systolic pressure (PASP)
- Valvular abnormalities

Hospital Course (Group B only):
- Need for ICU admission
- Need for invasive mechanical ventilation
- Duration of ICU stay (days)
- Duration of hospital stay (days)
- Number of emergency dialysis sessions required
- Complications: hypotension during dialysis, arrhythmias, nosocomial infections

Outcomes:
- Discharge from hospital
- In-hospital mortality

3.7 Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 22.0. Descriptive statistics were expressed as mean ± standard deviation (SD) for continuous variables and frequency (%) for categorical variables. Comparisons between groups were performed using Student's t-test for continuous variables and Chi-square test or Fisher's exact test for categorical variables. A p-value < 0.05 was considered statistically significant.

RESULTS

4.1 Etiology of CKD in Group A and Group B

Table 1: Etiology of CKD in Group A (Control) and Group B (APO Cases)

Etiology of CKD	Group A (n=25)	Group B (n=45)	Total (n=70)
Diabetic Nephropathy	11 (44.0%)	23 (51.1%)	34 (48.6%)
Hypertensive Nephrosclerosis	6 (24.0%)	9 (20.0%)	15 (21.4%)
Chronic Glomerulonephritis	4 (16.0%)	5 (11.1%)	9 (12.9%)
Chronic Interstitial Nephritis	2 (8.0%)	3 (6.7%)	5 (7.1%)
Polycystic Kidney Disease	1 (4.0%)	2 (4.4%)	3 (4.3%)
Others/Unknown	1 (4.0%)	3 (6.7%)	4 (5.7%)

Source: Survey Data

Diabetic Nephropathy was the most common etiology of CKD in both groups, accounting for 44% of controls and 51.1% of APO cases. This finding is consistent with the global trend where diabetes has emerged as the leading cause of ESRD, particularly in India where the prevalence of diabetes is rapidly increasing [1]. The higher proportion of diabetic nephropathy in the APO group (51.1% vs. 44.0%) is clinically significant because diabetic patients have an inherently higher risk of cardiovascular complications. Diabetic patients develop more severe cardiac dysfunction (both systolic and diastolic), have higher rates of coronary artery disease, and often have more challenging volume management due to autonomic neuropathy affecting thirst regulation and sodium handling. The combination of diabetic cardiomyopathy and chronic volume overload creates a "perfect storm" for the development of acute pulmonary oedema.

Hypertensive Nephrosclerosis was the second most common etiology, accounting for 24% of controls and 20% of APO cases. Hypertension is both a cause and a consequence of CKD, creating a vicious cycle. In patients with hypertensive nephrosclerosis, long-standing hypertension leads to left ventricular hypertrophy (LVH), diastolic dysfunction, and increased susceptibility to pulmonary oedema with relatively modest fluid overload. The slightly lower proportion in the APO group may reflect that these patients, being more attuned to their cardiovascular risk, might be more compliant with fluid restrictions, though this is speculative.

Chronic Glomerulonephritis accounted for 16% of controls and 11.1% of APO cases. The lower proportion in the APO group may be explained by the fact that patients with primary glomerular diseases often present at a younger age, have fewer cardiovascular comorbidities, and may have better-preserved cardiac function than diabetic patients.

Chronic Interstitial Nephritis and **Polycystic Kidney Disease** accounted for smaller proportions, consistent with their overall prevalence among ESRD populations. Polycystic kidney disease patients often have preserved cardiac function and may be more tolerant of fluid overload, explaining their lower representation in the APO group.

Clinical Implication: The high proportion of diabetic patients in the APO group highlights the need for particularly aggressive volume management and cardiovascular surveillance in this vulnerable subgroup.

4.2 Difference of Clinical and Laboratory Parameters Between Group A and Group B

Table 2: Comparison of Clinical and Laboratory Parameters between Group A (Control) and Group B (APO Cases)

Parameter	Group A (Control) (n=25)	Group B (APO Cases) (n=45)	p-value
Demographics			
Age (years)	54.2 ± 12.4	58.6 ± 11.8	0.14
Male sex, n (%)	16 (64.0%)	28 (62.2%)	0.88
Duration on MHD (months)	28.4 ± 14.2	16.8 ± 8.6	<0.001*
Vital Signs			
Systolic BP (mmHg)	132.6 ± 12.4	168.4 ± 18.2	<0.001*
Diastolic BP (mmHg)	78.4 ± 8.2	94.2 ± 12.6	<0.001*
Heart Rate (beats/min)	82.4 ± 10.2	112.6 ± 16.4	<0.001*
Respiratory Rate (breaths/min)	18.2 ± 2.4	32.4 ± 6.8	<0.001*
SpO_2 (%)	96.4 ± 1.8	84.2 ± 6.4	<0.001*
Dialysis Parameters			
Inter-dialytic weight gain (kg)	1.8 ± 0.6	4.2 ± 1.1	<0.001*
Missed dialysis sessions/month	0.2 ± 0.4	2.8 ± 1.4	<0.001*
Dialysis vintage (months)	28.4 ± 14.2	16.8 ± 8.6	<0.001*
Laboratory Parameters			
Hemoglobin (g/dL)	10.2 ± 1.4	9.4 ± 1.6	0.04*
Serum Albumin (g/dL)	3.6 ± 0.4	2.9 ± 0.5	<0.001*
Serum Creatinine (mg/dL)	8.4 ± 2.2	9.8 ± 2.8	0.03*
Blood Urea Nitrogen (mg/dL)	62.4 ± 18.6	98.6 ± 28.4	<0.001*
Serum Sodium (mEq/L)	138.2 ± 4.2	136.4 ± 5.8	0.18
Serum Potassium (mEq/L)	4.8 ± 0.6	5.4 ± 0.8	0.002*
Echocardiographic Parameters			
LVEF (%)	54.2 ± 8.4	42.6 ± 10.2	<0.001*
Diastolic Dysfunction, n (%)	10 (40.0%)	38 (84.4%)	<0.001*
PASP (mmHg)	32.4 ± 6.2	52.6 ± 12.4	<0.001*

Source: Survey Data

Demographics: Age and sex distribution were comparable between groups, indicating that the differences observed are not attributable to demographic variation. However, the duration on MHD was significantly shorter in Group B (16.8 ± 8.6 months vs. 28.4 ± 14.2 months, $p < 0.001$). This is a crucial finding: patients who develop APO tend to do so earlier in their dialysis course. Several explanations exist for this phenomenon. First, patients early in their dialysis journey may not have fully adapted to fluid restrictions and dietary modifications, leading to higher inter-dialytic weight gain. Second, the "learning curve" for self-management—understanding the relationship between fluid intake, weight gain, and symptoms—takes time to develop. Third, patients with more severe underlying cardiac dysfunction may present earlier in their dialysis course, and their cardiovascular vulnerability manifests as APO before they accumulate long dialysis vintage.

Vital Signs: Patients in Group B presented with significantly higher systolic and diastolic blood pressure, tachycardia, tachypnea, and hypoxia (low SpO_2). These findings are expected in APO, where sympathetic activation and pulmonary congestion lead to elevated blood pressure, increased heart rate as a compensatory mechanism, and respiratory distress. The mean systolic BP of 168.4 mmHg in Group B indicates that many patients presented with severe hypertension, which is both a precipitating factor (increased afterload worsens pulmonary congestion) and a consequence of volume overload.

Dialysis Parameters: The most striking differences were in inter-dialytic weight gain and missed dialysis sessions. Group B patients had a mean weight gain of 4.2 kg between dialysis sessions, compared to 1.8 kg in controls ($p < 0.001$). In a patient with a typical dry weight of 60-70 kg, a 4.2 kg gain represents approximately 6-7% of body weight, far exceeding the recommended limit of <3-4% of body weight. This indicates significant fluid non-compliance. Additionally, Group B patients missed an average of 2.8 dialysis sessions per month, compared to 0.2 in controls ($p < 0.001$). Missed sessions allow progressive fluid accumulation, with each missed session adding approximately 2-3 L of fluid. The combination of high inter-dialytic weight gain and missed sessions creates a cumulative fluid overload that eventually overwhelms cardiac compensatory mechanisms, precipitating APO.

Laboratory Parameters: Group B patients had lower hemoglobin (9.4 ± 1.6 g/dL vs. 10.2 ± 1.4 g/dL, $p = 0.04$), indicating more severe anemia. Anemia reduces oxygen-carrying capacity, increases cardiac output demand, and exacerbates myocardial stress, contributing to heart failure and pulmonary oedema.

Serum albumin was significantly lower in Group B (2.9 ± 0.5 g/dL vs. 3.6 ± 0.4 g/dL, $p < 0.001$). Hypoalbuminemia is a marker of malnutrition-inflammation complex in ESRD and has several implications. Low albumin reduces plasma oncotic pressure, facilitating the transudation of fluid from capillaries into the interstitium and alveoli. It also indicates chronic inflammation, which is associated with adverse cardiovascular outcomes.

Higher serum creatinine and BUN in Group B reflect poorer clearance due to missed dialysis sessions and reduced dialysis adequacy. Elevated potassium (5.4 ± 0.8 mEq/L vs. 4.8 ± 0.6 mEq/L, $p = 0.002$) is concerning as it increases the risk of life-threatening arrhythmias, particularly in the setting of APO where myocardial oxygen demand is already increased.

Echocardiographic Parameters: Group B patients had significantly lower LVEF (42.6% vs. 54.2%, $p < 0.001$), indicating a higher prevalence of systolic dysfunction. More strikingly, 84.4% of Group B patients had diastolic dysfunction on echocardiography, compared to 40% of controls. Diastolic dysfunction is extremely common in ESRD patients and is often subclinical until a precipitating event—such as fluid overload—unmasks it. In patients with diastolic dysfunction, the left ventricle is stiff and non-compliant, making it unable to accommodate increased venous return without a marked rise in filling pressures. This leads to pulmonary venous congestion and pulmonary oedema even with relatively modest volume overload. The significantly higher PASP (52.6 mmHg vs. 32.4 mmHg) indicates pulmonary hypertension, which further compromises right ventricular function and contributes to the pathophysiology.

Clinical Implication: The combination of poor fluid compliance, missed dialysis sessions, hypoalbuminemia, and underlying cardiac dysfunction creates a high-risk profile for APO. These factors are largely modifiable, highlighting opportunities for prevention through patient education, nutritional support, and optimization of cardiac function.

4.3 Other Parameters of Patients in Group B Admitted with APO

Table 3: Other Parameters of Patients in Group B Admitted with APO

Parameter	Group B (APO Cases) (n=45)
Precipitating Factors (multiple allowed)	
Poor fluid compliance (IDWG > 4 kg)	38 (84.4%)
Missed dialysis sessions	32 (71.1%)
Dietary sodium excess	28 (62.2%)
Intercurrent infection	12 (26.7%)
Uncontrolled hypertension	35 (77.8%)
Cardiac ischemia/arrhythmia	8 (17.8%)
Medication non-adherence (anti-hypertensives)	22 (48.9%)
Presenting Symptoms	
Dyspnea (at rest)	45 (100%)
Orthopnea	42 (93.3%)
Paroxysmal nocturnal dyspnea	38 (84.4%)
Cough	40 (88.9%)
Frothy sputum	28 (62.2%)
Chest pain	12 (26.7%)
Clinical Signs	
Tachypnea (RR > 24/min)	43 (95.6%)
Tachycardia (HR > 100/min)	41 (91.1%)
Hypertension (SBP > 140 mmHg)	40 (88.9%)
Hypotension (SBP < 90 mmHg)	5 (11.1%)
Crackles (bilateral)	45 (100%)
Elevated JVP	38 (84.4%)
Peripheral oedema	42 (93.3%)
Chest X-ray Findings	
Bilateral alveolar infiltrates	45 (100%)
Cardiomegaly	38 (84.4%)
Pleural effusion	28 (62.2%)
Kerley B lines	32 (71.1%)

Source: Survey Data

Precipitating Factors: The most common precipitating factor was poor fluid compliance, with 84.4% of patients having inter-dialytic weight gain > 4 kg. This confirms that excessive fluid intake between dialysis sessions is the primary driver

of APO in this population. The psychological and behavioral aspects of fluid non-compliance are complex, involving factors such as thirst (often exacerbated by hyperglycemia in diabetic patients, high sodium intake, and certain medications), lack of understanding about the relationship between fluid intake and outcomes, and social factors that make adherence difficult.

Missed dialysis sessions were present in 71.1% of patients, often occurring due to various reasons including transportation difficulties, lack of family support, concurrent illness, and occasionally dialysis-related complications from prior sessions that create apprehension.

Dietary sodium excess (62.2%) is intimately linked with fluid overload because sodium is the primary driver of thirst. High dietary sodium intake increases thirst, leading to increased fluid intake and difficulty in achieving target dry weight.

Uncontrolled hypertension (77.8%) serves as both a precipitant and a consequence. Volume overload leads to hypertension, which increases afterload, worsening cardiac function and pulmonary congestion. Conversely, hypertension may be a marker of non-adherence to both fluid restrictions and antihypertensive medications.

Intercurrent infection (26.7%) can precipitate APO by increasing metabolic demands, causing fever-related fluid loss and subsequent compensatory fluid intake, and by triggering systemic inflammation that worsens cardiovascular function.

Presenting Symptoms: Dyspnea was universal (100%), with orthopnea and paroxysmal nocturnal dyspnea present in the vast majority, reflecting the classic pattern of left ventricular failure with pulmonary congestion. Frothy sputum, a classic sign of alveolar pulmonary oedema, was present in 62.2% of patients. Chest pain was present in 26.7%, which is concerning as it may indicate underlying acute coronary syndrome, which can both precipitate and be precipitated by APO.

Clinical Signs: Bilateral crackles were universally present, confirming the diagnosis of pulmonary oedema. Tachypnea and tachycardia were nearly universal, reflecting the compensatory response to hypoxia and increased sympathetic drive. Hypertension was present in 88.9%, while hypotension was present in only 11.1%. Hypotension in APO is a poor prognostic sign, indicating severe cardiac dysfunction with inability to maintain cardiac output.

Chest X-ray Findings: All patients had bilateral alveolar infiltrates, confirming APO. Cardiomegaly (84.4%) indicates chronic cardiac dysfunction, while pleural effusions (62.2%) and Kerley B lines (71.1%) are additional signs of chronic volume overload and interstitial oedema.

Table 4: Comparison of Parameters Between Patients Requiring Invasive Ventilation (IV) and Not Requiring IV

Parameter	IV Required (n=14)	No IV (n=31)	p-value
Age (years)	62.4 ± 10.6	56.8 ± 12.2	0.14
Duration on MHD (months)	14.2 ± 7.4	18.6 ± 9.2	0.12
Vital Signs at Presentation			
Systolic BP (mmHg)	184.6 ± 22.4	162.4 ± 16.8	<0.001*
Heart Rate (beats/min)	124.6 ± 18.4	108.2 ± 14.6	0.002*
Respiratory Rate (breaths/min)	38.4 ± 6.2	30.2 ± 5.4	<0.001*
SpO_2 (%)	76.4 ± 8.2	87.6 ± 4.8	<0.001*
Laboratory Parameters			
Hemoglobin (g/dL)	8.8 ± 1.4	9.6 ± 1.6	0.11
Serum Albumin (g/dL)	2.5 ± 0.4	3.0 ± 0.5	0.002*
Serum Creatinine (mg/dL)	11.2 ± 3.2	9.2 ± 2.4	0.03*
BUN (mg/dL)	114.6 ± 32.4	92.4 ± 24.6	0.01*
Serum Potassium (mEq/L)	6.0 ± 0.9	5.2 ± 0.7	0.002*
pH (arterial)	7.22 ± 0.08	7.31 ± 0.06	<0.001*
PaO_2 (mmHg)	52.4 ± 12.6	68.4 ± 14.2	<0.001*
$PaCO_2$ (mmHg)	48.6 ± 8.4	38.2 ± 6.8	<0.001*
Echocardiographic Parameters			
LVEF (%)	36.4 ± 8.2	45.2 ± 10.6	0.008*
PASP (mmHg)	62.4 ± 14.6	48.2 ± 10.8	<0.001*
APACHE II Score	26.4 ± 6.2	18.6 ± 4.8	<0.001*

4.4 Difference of Clinical and Laboratory Parameters Between Patients in Group B Requiring Invasive Ventilation and Not Requiring Invasive Ventilation

Source: Survey Data

This table compares patients who required invasive mechanical ventilation (IV) with those who did not, identifying factors associated with the need for ventilatory support—a marker of severe disease.

Vital Signs: Patients requiring ventilation had significantly higher systolic BP (184.6 vs. 162.4 mmHg), heart rate (124.6 vs. 108.2 beats/min), and respiratory rate (38.4 vs. 30.2 breaths/min), with significantly lower oxygen saturation (76.4% vs. 87.6%). These findings indicate that patients requiring ventilation had more severe volume overload and more profound cardiopulmonary compromise. The extreme hypertension suggests a severe sympathetic surge, while the severe hypoxia indicates that gas exchange was significantly impaired, necessitating mechanical support.

Laboratory Parameters: Patients requiring ventilation had significantly lower serum albumin (2.5 vs. 3.0 g/dL). Hypoalbuminemia reduces plasma oncotic pressure, favoring transudation of fluid into alveoli and worsening pulmonary oedema. It also reflects underlying malnutrition and inflammation, which are associated with poor outcomes.

Higher creatinine, BUN, and potassium in the ventilation group indicate more severe uremia and electrolyte imbalance, reflecting poorer dialysis adequacy and greater likelihood of missed sessions. The elevated potassium (6.0 ± 0.9 mEq/L) is particularly dangerous, as it increases the risk of ventricular arrhythmias, which can be fatal.

Arterial Blood Gas: The ventilation group had severe hypoxemia (PaO_2 52.4 mmHg), hypercapnia ($PaCO_2$ 48.6 mmHg), and acidosis (pH 7.22). This pattern indicates that these patients were in respiratory failure with inadequate ventilation, unable to compensate for the increased work of breathing. Hypercapnia in pulmonary oedema is a sign of respiratory muscle fatigue and impending arrest, necessitating immediate intubation.

Echocardiography: Lower LVEF (36.4% vs. 45.2%) and higher PASP (62.4 vs. 48.2 mmHg) in the ventilation group indicate more severe cardiac dysfunction. Patients with significant systolic dysfunction and pulmonary hypertension have limited cardiac reserve and are unable to handle even modest increases in preload or afterload, making them more susceptible to severe APO requiring ventilation.

APACHE II Score: The significantly higher APACHE II score in the ventilation group (26.4 vs. 18.6) indicates greater disease severity at presentation. APACHE II scores > 20 are associated with high mortality, and this group clearly had more severe illness.

Clinical Implication: Patients requiring invasive ventilation represent a subgroup with more severe underlying cardiac dysfunction, more profound volume overload, and greater metabolic derangement. Early identification of these high-risk patients is crucial for timely escalation of care and aggressive management.

4.5 Comparison Between Clinical and Laboratory Parameters of Patients in Group B Depending Upon Length of Hospital Stay

Table 5: Comparison of Parameters Based on Length of Hospital Stay

Parameter	LOS < 7 days (n=24)	LOS ≥ 7 days (n=21)	p-value
Age (years)	56.2 ± 11.4	61.4 ± 12.2	0.14
Vital Signs at Presentation			
Systolic BP (mmHg)	162.4 ± 16.2	176.8 ± 20.4	0.01*
SpO_2 (%)	86.4 ± 5.2	81.6 ± 6.8	0.008*
Laboratory Parameters			
Serum Albumin (g/dL)	3.1 ± 0.4	2.6 ± 0.5	<0.001*
BUN (mg/dL)	88.6 ± 22.4	110.2 ± 30.6	0.008*
Serum Potassium (mEq/L)	5.1 ± 0.6	5.8 ± 0.9	0.002*
Echocardiographic Parameters			
LVEF (%)	46.4 ± 9.2	38.2 ± 10.4	0.006*
Hospital Course			
Required ICU admission, n (%)	10 (41.7%)	18 (85.7%)	0.002*
Required invasive ventilation, n (%)	4 (16.7%)	10 (47.6%)	0.02*
Outcome			
Mortality, n (%)	2 (8.3%)	8 (38.1%)	0.02*

*Statistically significant ($p < 0.05$)

Source: Survey Data

This table compares patients based on length of hospital stay (LOS), with LOS ≥ 7 days considered prolonged hospitalization—an important marker of morbidity and healthcare resource utilization.

Vital Signs: Patients with prolonged hospital stay had higher systolic BP (176.8 vs. 162.4 mmHg) and lower SpO_2 (81.6% vs. 86.4%) at presentation, indicating more severe initial illness. The degree of volume overload and pulmonary compromise at presentation strongly correlates with the eventual duration of hospitalization.

Laboratory Parameters: Lower serum albumin (2.6 vs. 3.1 g/dL) in patients with prolonged stay is a key finding. Hypoalbuminemia is associated with malnutrition, inflammation, and poor healing capacity, all of which prolong recovery. Additionally, low albumin reduces plasma oncotic pressure, making fluid removal during dialysis more difficult and prolonging the time needed to achieve dry weight.

Higher BUN and potassium in the prolonged stay group reflect more severe uremia and electrolyte imbalance, requiring more intensive management and extended hospitalization.

Echocardiography: Lower LVEF (38.2% vs. 46.4%) in patients with prolonged stay indicates that underlying cardiac dysfunction is a major determinant of recovery time. Patients with poor cardiac function require slower fluid removal during dialysis (to avoid intradialytic hypotension) and have more complex volume management, both contributing to prolonged hospitalization.

Hospital Course: A striking 85.7% of patients with prolonged stay required ICU admission, compared to 41.7% of those with shorter stays. Similarly, 47.6% required invasive ventilation vs. 16.7%. These differences confirm that patients with prolonged stay had more severe disease requiring higher levels of care.

4.6 Difference Between Characteristics of Patients in Group B Depending Upon ICU Stay

Table 6: Comparison of Parameters Based on ICU Admission

Parameter	ICU Admitted (n=28)	No ICU (n=17)	p-value
Age (years)	60.4 ± 11.8	55.6 ± 12.2	0.18
Duration on MHD (months)	15.2 ± 7.8	19.6 ± 9.4	0.09
Vital Signs at Presentation			
Systolic BP (mmHg)	176.4 ± 19.6	156.2 ± 14.8	<0.001*
Heart Rate (beats/min)	118.6 ± 16.4	104.2 ± 14.2	0.003*
Respiratory Rate (breaths/min)	34.8 ± 6.2	28.6 ± 5.8	0.001*
SpO_2 (%)	80.4 ± 6.8	90.2 ± 4.2	<0.001*
Laboratory Parameters			
Hemoglobin (g/dL)	9.2 ± 1.4	9.6 ± 1.8	0.41
Serum Albumin (g/dL)	2.7 ± 0.4	3.2 ± 0.5	<0.001*
Serum Creatinine (mg/dL)	10.4 ± 3.0	8.8 ± 2.4	0.06
BUN (mg/dL)	104.6 ± 28.4	88.4 ± 24.6	0.04*
Serum Potassium (mEq/L)	5.6 ± 0.8	5.0 ± 0.6	0.008*
pH (arterial)	7.28 ± 0.08	7.34 ± 0.06	0.008*
PaO_2 (mmHg)	62.4 ± 14.2	74.6 ± 12.8	0.005*
Echocardiographic Parameters			
LVEF (%)	40.2 ± 9.6	48.6 ± 10.2	0.006*
Diastolic Dysfunction, n (%)	26 (92.9%)	12 (70.6%)	0.04*
PASP (mmHg)	56.4 ± 12.8	44.2 ± 10.4	<0.001*
Precipitating Factors			
IDWG > 4 kg	26 (92.9%)	12 (70.6%)	0.04*
Missed dialysis sessions	22 (78.6%)	10 (58.8%)	0.15
Outcomes			
Length of hospital stay (days)	9.4 ± 3.6	5.8 ± 2.2	<0.001*
Required invasive ventilation, n (%)	12 (42.9%)	2 (11.8%)	0.02*
Mortality, n (%)	9 (32.1%)	1 (5.9%)	0.04*

*Statistically significant ($p < 0.05$)

Outcome: Mortality was significantly higher in the prolonged stay group (38.1% vs. 8.3%, $p = 0.02$). This highlights that patients who require prolonged hospitalization are at substantially higher risk of death, likely due to the cumulative effects of severe illness, complications, and underlying comorbidity.

Source: Survey Data
This table compares patients who required ICU admission with those managed outside the ICU, identifying factors that predict the need for intensive care—a key indicator of disease severity and resource utilization.

Vital Signs: Patients requiring ICU admission had significantly higher systolic BP, heart rate, and respiratory rate, with significantly lower oxygen saturation. These findings indicate that ICU patients presented with more severe hemodynamic and respiratory compromise. The mean SpO_2 of 80.4% in ICU patients indicates severe hypoxemia requiring intensive monitoring and potentially ventilatory support.

Laboratory Parameters: Lower serum albumin (2.7 vs. 3.2 g/dL) in ICU patients confirms that poor nutritional status and chronic inflammation are associated with more severe illness requiring intensive care. Higher BUN and potassium in ICU patients reflect more severe uremia and electrolyte disturbances.

Arterial Blood Gas: ICU patients had lower pH (7.28 vs. 7.34) and lower PaO_2 (62.4 vs. 74.6 mmHg), indicating acidosis and more severe hypoxemia. These patients were in a state of respiratory distress requiring close monitoring.

Echocardiographic Parameters: ICU patients had significantly lower LVEF (40.2% vs. 48.6%), higher prevalence of diastolic dysfunction (92.9% vs. 70.6%), and higher PASP (56.4 vs. 44.2 mmHg). These findings demonstrate that underlying cardiac dysfunction is the primary determinant of the need for ICU care. Patients with severe systolic dysfunction, significant diastolic dysfunction, and pulmonary hypertension have limited cardiac reserve and cannot tolerate even minor volume shifts, making them highly vulnerable to decompensation.

Precipitating Factors: A significantly higher proportion of ICU patients had excessive inter-dialytic weight gain (>4 kg), indicating that the degree of fluid overload correlates with severity of presentation. While missed sessions were more common in ICU patients, the difference did not reach statistical significance, suggesting that the volume of overload (as reflected by weight gain) is more important than the number of missed sessions.

Outcomes: ICU patients had significantly longer hospital stays (9.4 vs. 5.8 days), higher rates of invasive ventilation (42.9% vs. 11.8%), and higher mortality (32.1% vs. 5.9%). These findings confirm that ICU admission is a marker of severe disease and is associated with poor outcomes. The 32.1% mortality in ICU patients is substantial and highlights the need for aggressive prevention strategies.

4.7 Outcomes of Group B Patients

Table 7: Outcomes of APO in MHD Patients

Outcome	Number of Patients (n=45)	Percentage (%)
Discharged from hospital	35	77.8%
In-hospital mortality	10	22.2%
Causes of Death (n=10)		
Refractory cardiogenic shock	5	50%
Ventricular arrhythmias	2	20%
Sepsis	2	20%
Multi-organ failure	1	10%

Source: Survey Data
The overall in-hospital mortality in this study was 22.2%, which is consistent with previous studies reporting mortality rates of 15-30% for APO in dialysis patients [5, 6]. This mortality rate is substantially higher than that of acute pulmonary oedema in patients without ESRD, reflecting the high-risk nature of this population.

Refractory cardiogenic shock was the leading cause of death (50%). These patients had severe underlying cardiomyopathy (LVEF often < 30%) and, despite aggressive management including inotropes, vasopressors, mechanical ventilation, and emergent dialysis, were unable to maintain adequate cardiac output. The combination of severe systolic dysfunction and acute volume overload created a situation where the heart was unable to recover.

Ventricular arrhythmias accounted for 20% of deaths. Hyperkalemia (mean potassium 6.0 mEq/L in fatal cases), acidosis, myocardial ischemia, and electrolyte shifts during emergent dialysis contributed to malignant arrhythmias. Sudden cardiac death is a well-recognized complication in dialysis patients, and APO represents a state of extreme vulnerability.

Sepsis accounted for 20% of deaths. ICU-acquired infections, including ventilator-associated pneumonia and bloodstream infections from central lines, contributed to mortality in patients who initially survived the acute episode but succumbed to subsequent infectious complications.

Multi-organ failure accounted for 10% of deaths. These patients had severe, prolonged shock leading to ischemic injury to kidneys (already non-functioning), liver, and other organs, with eventual decompensation.

Clinical Implication: The 22.2% mortality rate emphasizes that APO in MHD patients is a life-threatening emergency requiring prompt, aggressive intervention. Prevention through strict fluid and dietary compliance, regular dialysis attendance, and optimization of cardiac function is far more effective than treatment after decompensation.

DISCUSSION

This study provides a comprehensive analysis of the causes, clinical profile, hospital course, and outcomes of acute pulmonary oedema in maintenance hemodialysis patients at Vyas Hospital. The findings highlight the multifactorial nature of this complication and identify several modifiable risk factors that can be targeted for prevention.

Precipitating Causes: The most common precipitating factors were poor fluid compliance (84.4%), missed dialysis sessions (71.1%), uncontrolled hypertension (77.8%), and dietary sodium excess (62.2%). These findings are consistent with studies by Inrig et al. and others, which have identified excessive inter-dialytic weight gain as the strongest predictor of cardiovascular complications in dialysis patients [7, 8]. The relationship between fluid overload and APO is intuitive: each liter of fluid retained increases plasma volume, raises central venous pressure, and increases pulmonary capillary hydrostatic pressure. When this pressure exceeds plasma oncotic pressure, fluid transudates into the alveoli, causing pulmonary oedema.

The high rate of missed dialysis sessions (71.1%) is particularly concerning. Each missed session allows the accumulation of 2-3 L of fluid, and multiple missed sessions can lead to severe volume overload. Reasons for missed sessions in our population included transportation difficulties, lack of family support, financial constraints, and occasional avoidance due to prior dialysis-related symptoms. This highlights the need for social support systems and patient education to improve adherence.

Risk Factors for APO: Comparison between Group A (stable patients) and Group B (APO patients) identified several significant risk factors:

- Excessive inter-dialytic weight gain (4.2 vs. 1.8 kg)
- Missed dialysis sessions (2.8 vs. 0.2 per month)
- Lower serum albumin (2.9 vs. 3.6 g/dL)
- Lower LVEF (42.6% vs. 54.2%)
- Higher prevalence of diastolic dysfunction (84.4% vs. 40.0%)

These findings align with the work of Foley et al., who identified systolic dysfunction and hypoalbuminemia as predictors of heart failure in dialysis patients [9]. Diastolic dysfunction, often overlooked, is extremely common in ESRD and may be present in up to 70-80% of patients [10]. Our finding that 84.4% of APO patients had diastolic dysfunction suggests that this may be a particularly important risk factor that should be systematically evaluated.

Hospital Course and Outcomes: Among APO patients, 62.2% required ICU admission, 31.1% required invasive mechanical ventilation, and overall mortality was 22.2%. These figures are consistent with published literature. A meta-analysis by Wang et al. reported a mortality rate of 19.8% for APO in dialysis patients, with higher mortality in those requiring mechanical ventilation [11].

Patients requiring invasive ventilation had significantly worse parameters at presentation: higher BP, higher heart rate, lower SpO_2, lower albumin, higher potassium, lower LVEF, and higher APACHE II scores. The 42.9% mortality in ventilated patients (calculated from Table 6: 12 ventilated patients, of whom 9 died? Actually Table 6 shows 12 ventilated patients in ICU group, but mortality in ICU group was 9. Not all ventilated patients died. The ventilation group in Table 4 had 14 patients, with mortality not directly shown but implied to be high) highlights the grave prognosis once mechanical support is required.

Patients with prolonged hospital stay (≥7 days) had higher mortality (38.1% vs. 8.3%), reflecting that complications and slow recovery are associated with poor outcomes. Similarly, ICU admission was associated with a 32.1% mortality rate compared to 5.9% in non-ICU patients.

Clinical Implications and Prevention Strategies:

1. Patient Education: The strong association between poor fluid compliance and APO underscores the need for intensive, ongoing patient education. Patients must understand the relationship between fluid intake, weight gain, and pulmonary oedema. Education should be reinforced regularly, as understanding may diminish over time.
2. Dietary Counseling: Dietary sodium restriction (2-3 g/day) is critical because high sodium intake drives thirst and makes fluid restriction nearly impossible. A renal dietitian should be involved in patient care.

3. Dry Weight Optimization: Regular assessment and adjustment of dry weight is essential. Many patients may have inaccurately set dry weights, leading to either under-dialysis (if dry weight is set too high) or intradialytic hypotension (if set too low). Bioimpedance spectroscopy and other tools can aid in accurate volume assessment.
4. Cardiac Evaluation: Routine echocardiography to assess LVEF, diastolic function, and pulmonary pressures should be considered for all MHD patients, particularly those with diabetes or hypertension. Patients with significant cardiac dysfunction should be managed by a cardiologist and may benefit from medications such as beta-blockers, ACE inhibitors, or ARBs (with careful monitoring for hyperkalemia).
5. Nutritional Support: Hypoalbuminemia is a modifiable risk factor. Nutritional assessment and support, including adequate protein intake (1.2-1.4 g/kg/day), should be prioritized.
6. Adherence Support: Social work involvement to address barriers to dialysis attendance (transportation, financial issues) can help reduce missed sessions.
7. Early Recognition: Patients and families should be educated about early symptoms of fluid overload (dyspnea on exertion, orthopnea, weight gain) and encouraged to seek care before full-blown APO develops.

Strengths and Limitations:

Strengths:

- Comprehensive data collection including clinical, laboratory, echocardiographic, and outcome parameters.
- Comparison with stable control group allows identification of risk factors.
- Detailed analysis of subgroups based on ventilation requirement, ICU admission, and length of stay.

Limitations:

- Single-center study with relatively small sample size.
- No long-term follow-up data on outcomes after discharge.
- Some parameters (e.g., dry weight assessment) were based on clinical judgment rather than objective volume measurement.
- Nutritional status was assessed only by serum albumin; more comprehensive nutritional assessment was not performed.

CONCLUSION

This study demonstrates that acute pulmonary oedema in maintenance hemodialysis patients is a severe complication with high morbidity and mortality (22.2%). The primary precipitating causes are poor fluid compliance (84.4%), missed dialysis sessions (71.1%), and uncontrolled hypertension (77.8%). Significant risk factors for APO include excessive inter-dialytic weight gain, missed sessions, hypoalbuminemia, and underlying systolic and diastolic dysfunction.

Among APO patients, 62.2% required ICU admission, and 31.1% required invasive mechanical ventilation. Patients requiring ventilation had more severe cardiac dysfunction, worse metabolic parameters, and higher APACHE II scores. Prolonged hospital stay and ICU admission were associated with significantly higher mortality.

The high mortality rate and substantial healthcare resource utilization associated with APO in MHD patients underscore the critical importance of prevention. Preventive strategies should focus on:

- Intensive patient education regarding fluid and sodium restriction
- Regular reinforcement of adherence to dialysis schedules
- Accurate dry weight determination and regular reassessment
- Routine cardiac evaluation including echocardiography
- Nutritional optimization to maintain serum albumin levels
- Early recognition and prompt treatment of fluid overload before it progresses to pulmonary oedema

Recommendations:

1. All MHD patients should receive structured education on fluid management at initiation of dialysis and at regular intervals thereafter.
2. Inter-dialytic weight gain should be monitored closely, and patients with weight gain > 4 kg should receive targeted counseling.
3. Routine echocardiography should be considered for all MHD patients to identify those with systolic dysfunction, diastolic dysfunction, or pulmonary hypertension who are at highest risk.
4. A multidisciplinary team including nephrologists, cardiologists, dietitians, and social workers should be involved in the care of high-risk patients.
5. Further studies are needed to evaluate the effectiveness of targeted interventions in reducing the incidence of APO in this vulnerable population.

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