



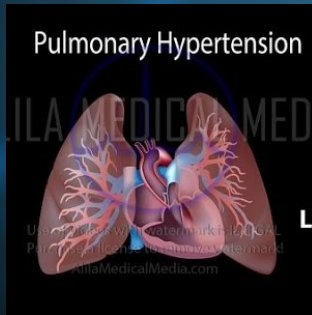
# **PULMONARY HYPERTENSION**

## **In chronic kidney disease patients**

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# Key points



1-Definition of pulmonary Hypertension

2-Clinical classification

3-Pathogenesis of pulmonary hypertension in CKD

4-Diagnosis

5-Treatment

6-Take home message



# Definition

- Pulmonary hypertension is defined as mean pulmonary artery pressure greater than or equal to 25 mmHg



Pulmonary Hypertension



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# Classification

- **Type ( I)** . Super vasoconstriction
- **Type (II)**. Left side heart disease
- **Type(III)**. Lung problems
- **Type(IV)**. Chronic pulmonary emboli
- **Type(V)**.multi factorial

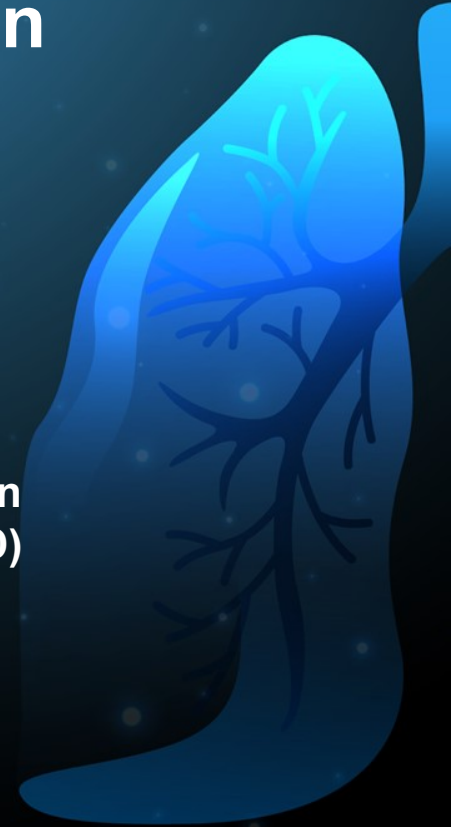
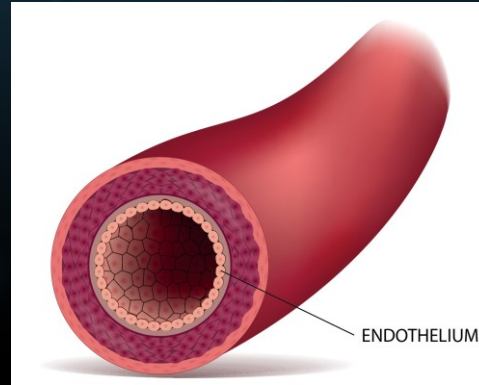
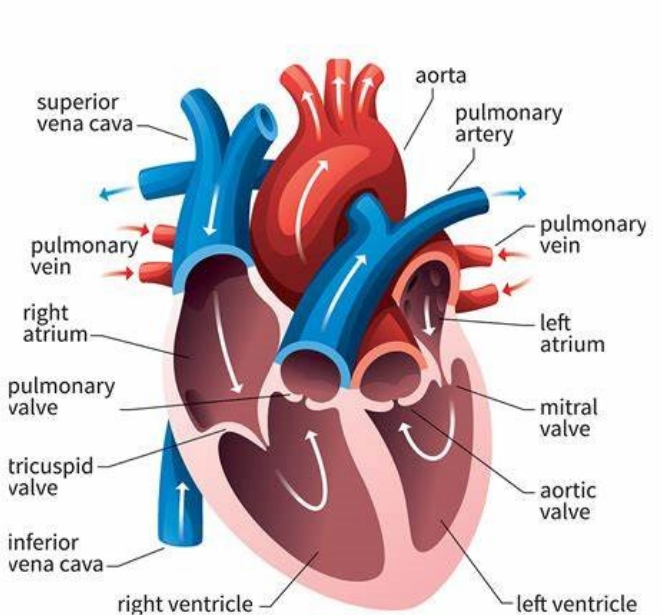


Pulmonary Hypertension



# Type I Pulmonary hypertension

- Pathophysiology: increase vasoconstrictor process.
- Etiology:
  - 1-idiopathic
  - 2-connective tissue disease
  - 3-hereditary( BMPR2)
  - 4-HIV infection
  - 5-portopulmonary hypertension
  - 6- left to right shunts(VSD-ASD)



# Type II pulmonary hypertension

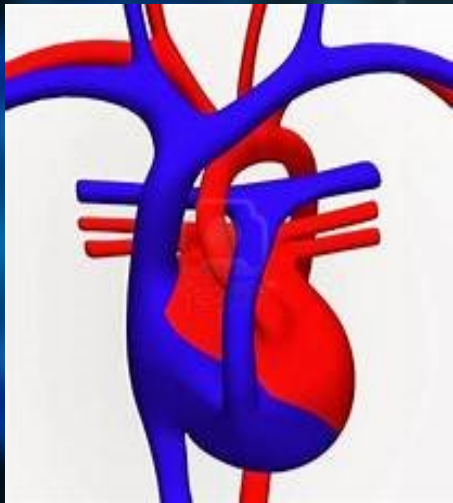
Pathophysiology : pulmonary veins congestion

Etiology :

Left side heart disease

Lt heart failure .

Valvular heart disease



## Pulmonary Hypertension



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# Type III pulmonary hypertension

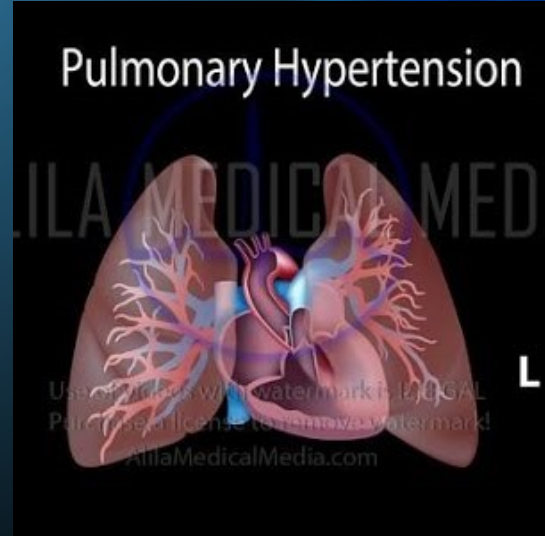
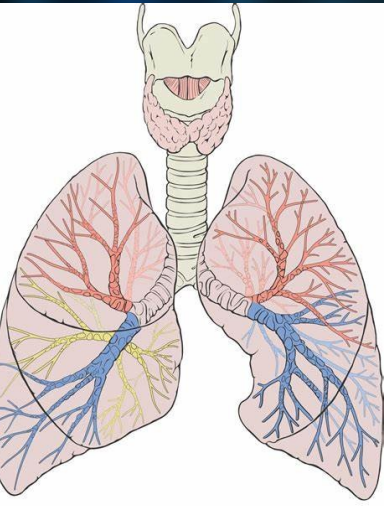
Pathophysiology : hypoxia induce vasoconstriction

Etiology : chronic lung disease

Chronic obstructive air way disease .

Sleep apnea .

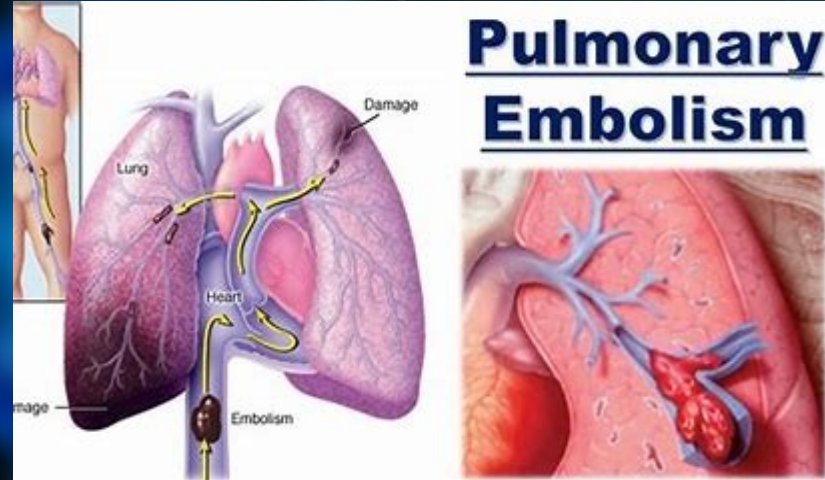
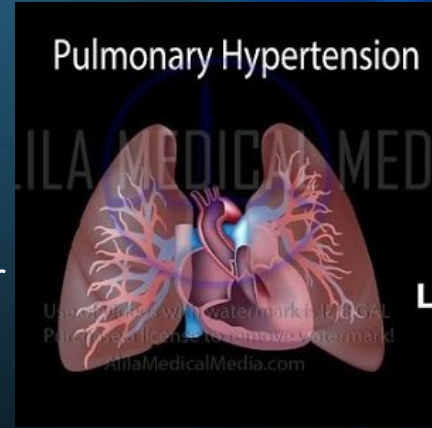
interstitial lung disease.



# Type IV pulmonary hypertension

Pathophysiology: increase resistance to the blood flow .

Etiology : chronic pulmonary emboli(recurrent shower of lower limb DvT).



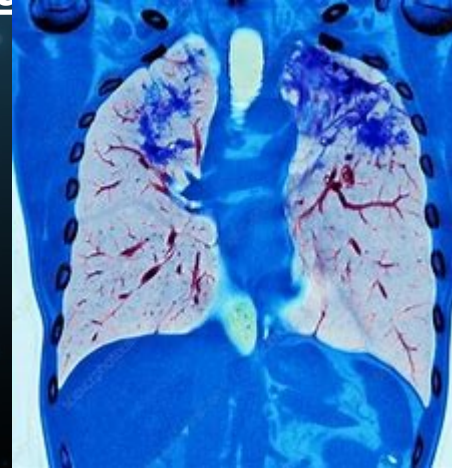


# Type V pulmonary hypertension

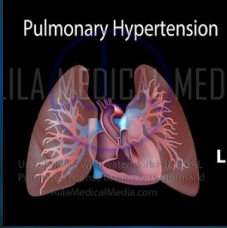
Multifactorial

Etiology:

Sarcoidosis, lymphoma, tracheal tumor, mediastinal tumor, *Chronic kidney disease*



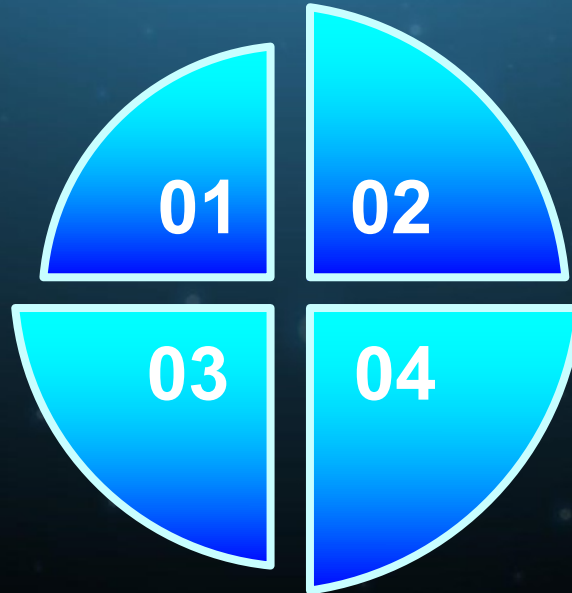
# Pathogenesis of PAH in CKD patients



**Cardiac dysfunction, endothelial dysfunction**

Air bubbles, septic and fibrin emboli

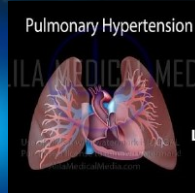
Obstructive sleep apnea



**Vascular calcification**

**A-V Fistula**

**Significant inflammatory response to unbiocompatible membrane**

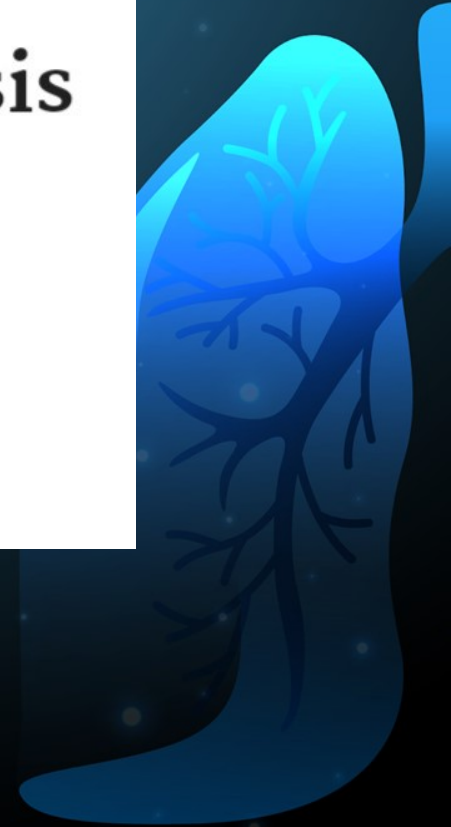


# Unexplained pulmonary hypertension in peritoneal dialysis and hemodialysis patients

[Article in English, Portuguese]

J Etemadi et al. Rev Port Pneumol. 2012 Jan-Feb.

**Conclusion:** Unexplained PAH seems to be more frequent in patients undergoing HD than patients in PD group. Moreover, hemoglobin and serum iron levels are lower in patients with PAH compared to those in normal PAP group.



[Indian J Nephrol.](#) 2019 Mar-Apr; 29(2): 95–101.  
doi: [10.4103/ijn.IJN\\_407\\_17](#)

PMCID: PMC6440332 | PMID: [30983749](#)

## Pulmonary Hypertension in Various Stages of Chronic Kidney Disease in Indian Patients

[Kalpana S. Mehta](#), [Abhishek K. Shirkande](#),  
[Sandip P. Bhurke](#), [Atim E. Pajai](#), [Rudramani S. Swami](#),  
and [Swapnil N. Jadhav](#)




### Conclusions

[Go to: ▶](#)

The prevalence of PH in CKD patients is 60.5%. Prevalence of PH had positive correlation with stage of CKD, duration of CKD, those on hemodialysis, and those with AVF. The severity of PH was also directly proportional to the duration of CKD and duration of hemodialysis. Calcium phosphorus product were significantly higher in CKD patients with PH than without it.

# Risk Factors of Pulmonary Hypertension in Patients on Hemodialysis: A Single Center Study



**Authors** [Nagaraju SP](#), [Bhojaraja MV](#) , [Paramasivam G, Prabhu RA, Rangaswamy D, Rao IR](#) , [Shenoy SV](#) 

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**Accepted for publication** 9 December 2021

**Published** 24 December 2021 Volume 2021:14 Pages 487–494

## Conclusion

HD patients have a higher prevalence of PH. Presence of traditional risk factors, dialysis-related factors, anemia, components of CKD-MBD, and inflammation/oxidative stress did not influence the prevalence of PH in our HD population. HD patients with PH have both left and right systolic dysfunction and it is significantly associated with the presence of mitral regurgitation.



# Pulmonary hypertension in hemodialysis patients: frequency and risk factors

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**Journal of Current Medical Research and Practice**

2022, 7:39–44

## Objective

The authors aimed to determine the prevalence of pulmonary hypertension in patients with end-stage kidney disease on regular hemodialysis (HD). The authors also aimed to study the possible correlation between the occurrence of pulmonary hypertension and fluid overload, arteriovenous fistula (AVF) blood flow, valvular calcification, and other laboratory variables.

## Patients and methods

This cross-sectional descriptive study was conducted on 80 patients with end-stage kidney disease on regular HD for more than one year in Al Agoza Hospital in Cairo, Egypt. General data were collected. A transthoracic 2D echocardiography was done for all studied patients within one hour after HD. Pulmonary hypertension's definition was an estimation of pulmonary artery systolic pressure greater than 35 mmHg by echocardiography.

## Results

Pulmonary hypertension (pulmonary artery systolic pressure >35 mmHg) presented in 21 (26.2%) patients, whereas mild (35–50 mmHg), moderate (51–70 mmHg), and severe pulmonary hypertension (>70 mmHg) presented in seven (8.8%), 10 (12.5%), and four (5%) patients, respectively. Pulmonary hypertension presented in 13 (62%) female patients vs eight (38%) male patients, which is of statistical significance ( $P<0.001$ ). Mean duration of dialysis was significantly higher in patients with pulmonary hypertension (PHTN) than those without ( $8.54\pm 3.53$  years vs  $3.88\pm 1.01$  years;  $P<0.001$ ). Valvular calcification, fluid overload, and blood flow through AVF were significantly correlated to the development of PHTN.

## Conclusion

Pulmonary hypertension in patients with end stage kidney disease (ESKD) on regular HD is frequent. Blood flow through AVF and chronic fluid overload may be involved in the development of PHTN in patients with ESKD who are on regular hemodialysis.



# The problem of pulmonary arterial hypertension in end-stage renal disease: can peritoneal dialysis be the solution



Abdullah K. Alhwiesh<sup>1</sup>, Ibrahim Saeed Abdul-Rahman<sup>1\*</sup>, Abdullah Alshehri<sup>2</sup>, Amani Alhwiesh<sup>1</sup>, Mahmoud Elnokeety<sup>1</sup>, Syed Essam<sup>1</sup>, Mohamad Sakr<sup>1</sup>, Nadia Al-Oudah<sup>1</sup>, Abdulla Abdulrahman<sup>3</sup>, Abdelgalil Moaz Mohammed<sup>1</sup>, Hany Mansour<sup>1</sup>, Tamer El-Salamoni<sup>1</sup>, Nehad Al-Oudah<sup>1</sup>, Lamees Alayooobi<sup>1</sup>, Hend Aljenaidi<sup>1</sup>, Ali Al-Harbi<sup>4</sup>, Dujanah Mousa<sup>4</sup>, Abdulghani Abdulnasir<sup>4</sup> and Sami Skhiri<sup>4</sup>

**Conclusions:** Long term APD ( $\geq 1$  years) seemed to decrease pulmonary arterial pressure, right atrial pressure and improve left ventricular ejection fraction (LVEF). Risk factors for PAH in ESRD were hypervolemia, abnormal ECHO findings and low hemoglobin levels. Clinical and echocardiographic abnormalities and complications are not uncommon among ESRD patients with PAH. Identification of those patients on transthoracic echocardiography may warrant further attention to treatment with APD.

[Multidiscip Respir Med.](#) 2011; 6(3):155–160.

Published online 2011 Jun 30.

doi: [10.1186/2049-6958-6-3-155](https://doi.org/10.1186/2049-6958-6-3-155)

PMCID: PMC3463070 | PMID: [22959121](https://pubmed.ncbi.nlm.nih.gov/22959121/)

## Renal transplant improves pulmonary hypertension in patients with end stage renal disease

[Serife Savas Bozbas](#),<sup>✉1</sup> [Suleyman Kanyilmaz](#),<sup>2</sup>  
[Sule Akcay](#),<sup>1</sup> [Huseyin Bozbas](#),<sup>2</sup> [Cihan Altin](#),<sup>2</sup>  
[Emir Karacaglar](#),<sup>2</sup> [Haldun Muderrisoglu](#),<sup>2</sup> and  
[Mehmet Haberal](#)<sup>3</sup>

### Conclusion

These findings indicate that patients with ESRD accompanied by PH may benefit from renal transplant. Further research is required for more concrete conclusions to be drawn on this subject.





# Incidence, Clinical Correlates, and Outcomes of Pulmonary Hypertension After Kidney Transplantation: Analysis of Linked US Registry and Medicare Billing Claims

Krista L. Lentine, MD, PhD,<sup>1</sup> Ngan N. Lam, MD,<sup>2</sup> Yasar Caliskan, MD,<sup>1</sup> Huiling Xiao, MD,<sup>1</sup> David A. Axelrod, MD,<sup>3</sup> Salvatore P. Costa, MD,<sup>4</sup> Deborah J. Levine, MD,<sup>5</sup> James R. Runo, MD,<sup>6</sup> Helen S. Te, MD,<sup>7</sup> Janani Rangaswami, MD,<sup>8</sup> Darshana M. Dadhania, MD,<sup>9</sup> Mark A. Schnitzler, PhD,<sup>1</sup> Bertram L. Kasiske, MD,<sup>10</sup> and Todd C. Villines, MD<sup>11</sup>

diagnosed posttransplant P-HTN were similar. **Conclusions.** Posttransplant P-HTN is diagnosed in 1 in 10 KTx recipients and is associated with an increased risk of death and graft failure. Future research is needed to refine diagnostic, classification, and management strategies to improve outcomes in KTx recipients who develop P-HTN.

(*Transplantation* 2022;106: 666–675).

## INTRODUCTION

Kidney transplant (KTx) is the optimal therapy for patients with end-stage kidney disease (ESKD), as it is associated

with longer patient survival, better quality of life, and lower costs to the healthcare system.<sup>1,2</sup> To realize these benefits, KTx recipients must avoid or overcome various

# Diagnosis

## • Sign and symptoms :

Generalized fatigability ,dizziness, exertional dyspnea, exertional angina, exertional syncope .

### RIGHT SIDED ♥ FAILURE

(Cor Pulmonale)

- Fatigue
- ↑ Peripheral Venous Pressure
- Ascites
- Enlarged Liver & Spleen
- Dependent Edema
- May be secondary to chronic pulmonary problems
- Distended Jugular Veins
- Anorexia & Complaints of GI Distress
- Weight Gain



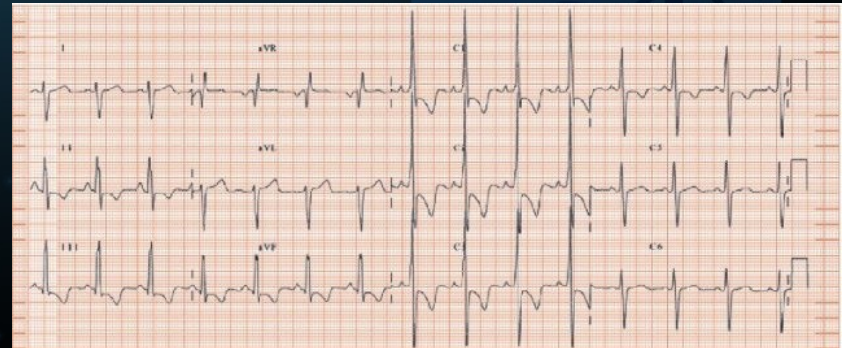
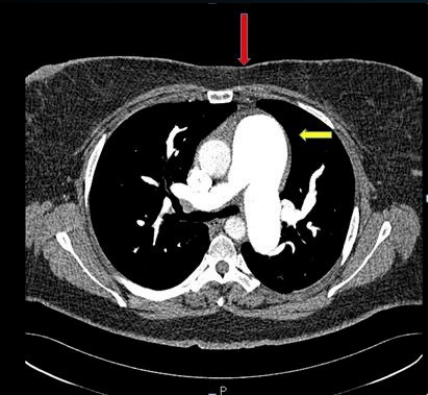
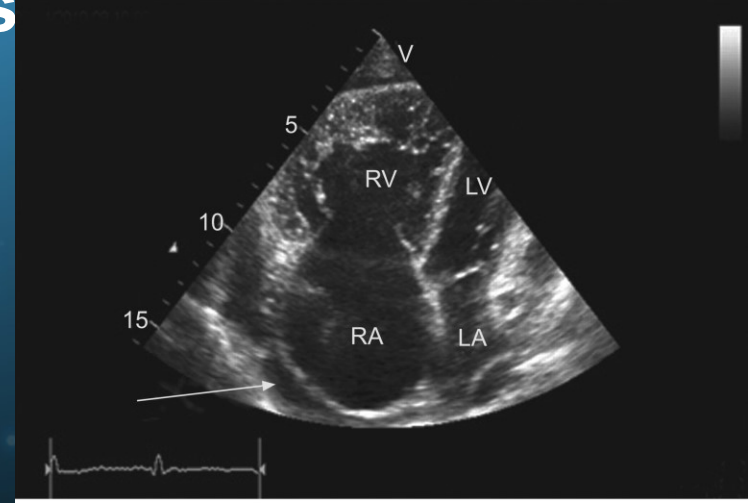
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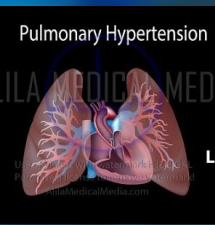
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# Investigations

- 1 – Echocardiography
- 2 – High resolution C.T scan
- 3 – sleep study (polysomnography)
- 4 – V/Q scan
- 5 – ECG
- 6 – Lab :
  - ANA, antiscle AB
- 7- Rt heart catheterization





# Treatment

## General measurement

- 1-Treatment of underlying cause
- 2- Diuretics to reduce edema
- 2- oxygen supply if needed
- 3- anticoagulant

## Specific measurement

- 1- calcium channel blockers  
nifedipin (vasoactive test +)
- 2- pulmonary vasodilator (vasoreactive test -)
  - A- endothelin I inhibitor (bosentan)
  - B- increase nitrose oxide phosphodiesterase inhibitor ( sildenafil)
  - C- prostacyclin analoge (iloprost)
- 3- B/L lung and heart transplantation

### VASOREACTIVITY

- Testing is done with right heart catheterization
- Defined as mean pulmonary artery pressure decrease by at least 10 mm Hg to a level below 40 mm Hg, with no decrease in cardiac output.
- Agents approved
  - Epoprostenol IV
  - Adenosine IV
  - Nitric oxide inhaled

## *Specific points in the mangment of pulmonary hypertension in CKD patients*

- no specific intervention trial aimed at reducing pulmonary hypertension in patients with CKD has been performed.

Correcting of volume overload and treating anaemia and left ventricular disorders may be important factors for relieving pulmonary hypertension in patients with CKD



## Pulmonary Hypertension



## —Take home message

- PAH is prevalent in patients with C.K.D. specially in H.D. patients
- P.A.H. in C.K.D. is multifactorial
  - Duration of dialysis and arteriovenous fistula has been strongly related to the development of PAH
  - PAH has been associated with high risk of mortality in CKD patients
  - The other secondary causes of pulmonary hypertension should also be recognize and treated accordingly.
- kidney transplantation and closure of A-V fistula and transferee to peritoneal dialysis can be treatment options of PAH in CKD patients. All are need further study and evaluation.



Thank you for attention