



## ARDS and Pregnancy

**Dr. Ravindra Gundeli (M.D.)**

Shri Markendeya Solapur Sahakari Rughalaya

**Abstract:**

*Patients of pregnancy and ARDS are less 2% of admission to hospital it is related or may not be related to pregnancy. Our case is lung related and pneumonitis with respiratory distress. Other are like aspiration, embolism, eclampsia and HELLP syndrome.*

**Introduction**

A 22-year-old pregnant woman admitted with 7 Months pregnant and 2nd gravida. She had a mild dry cough, denied chest pain, fever, hemoptysis or night sweats since from 5 days. She had no joint pains, rashes, anorexia, abdominal pain, nausea or vomiting. Less Than 2% Of Women Require Admission To Icu During Pregnancy Or Postpartum Period. Patients With Acute Respiratory Failure Generally Present With Distress ,Dysnoea, Unable To Speak Complete Sentences.

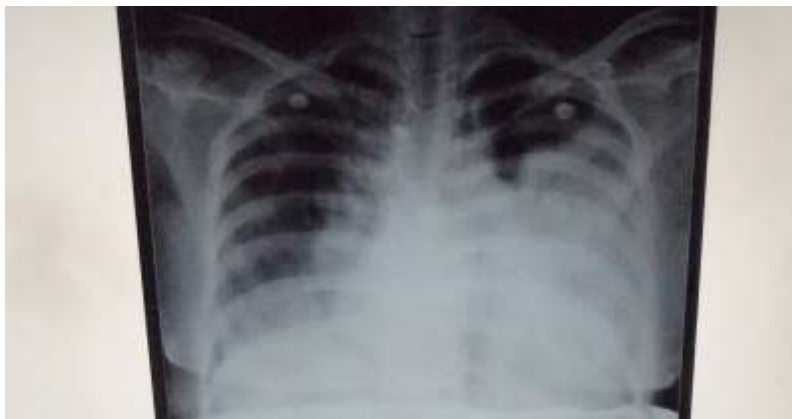
**Case Report:**

The patient was complaint with regular antenatal care and she was treated for Staphylococcus aureus UTI two-weeks prior to admission. Gestational history revealed multiple spontaneous abortions due to cervical incompetence and she had prophylactic cerclage this pregnancy. She denied toxic habits or travel history; On initial evaluation she was tachycardic, normotensive and afebrile. Her respiratory rate was 28 breaths per minute and her oxygen saturation was 95% in ambient air. There was no jugular venous distension. Rales were heard at lung bases. The abdomen was soft, gravid, and not tender. No skin lesions were noted. Initial laboratory results are shown Chest-x-ray on admission showed bilateral diffuse nodular infiltrates. She was transferred to ICU for acute hypoxic respiratory failure and treated with antibiotics for community acquired pneumonia and suspected infective endocarditis. Her respiratory status worsened requiring high FiO<sub>2</sub>. Intravenous steroids were initiated for fetal maturity. On day three of hospitalization she developed worsening infiltrates and was electively intubated followed by hemoptysis. Fiberoptic Bronchoscopy (FOB) revealed diffuse alveolar hemorrhage (DAH). (Table 2) Elective caesarian section with delivery of viable fetus was performed. Her hospital course was complicated by ARDS, septic shock and multiple organ failure. Work up for vasculitis was negative. Beta HCG declined post delivery. Cytology and microbiology of bronchoalveolar lavage specimens was negative. Echocardiogram showed no vegetations and normal heart function. Open lung biopsy could not be done due to high oxygen and PEEP requirements. Discharged on day 17 with good condition

**X-Ray Report-1**



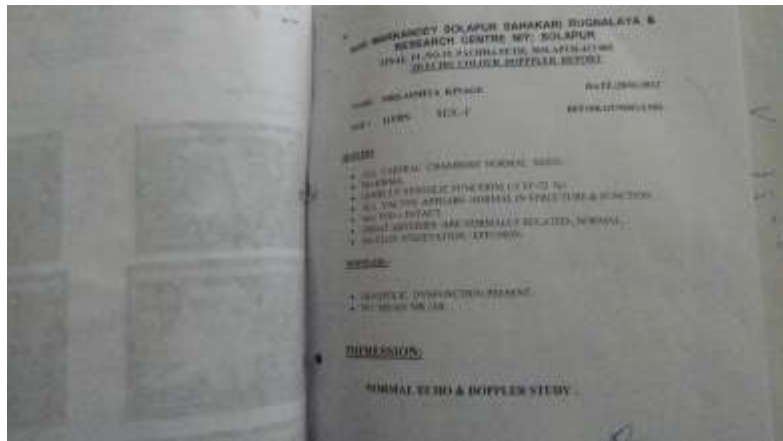
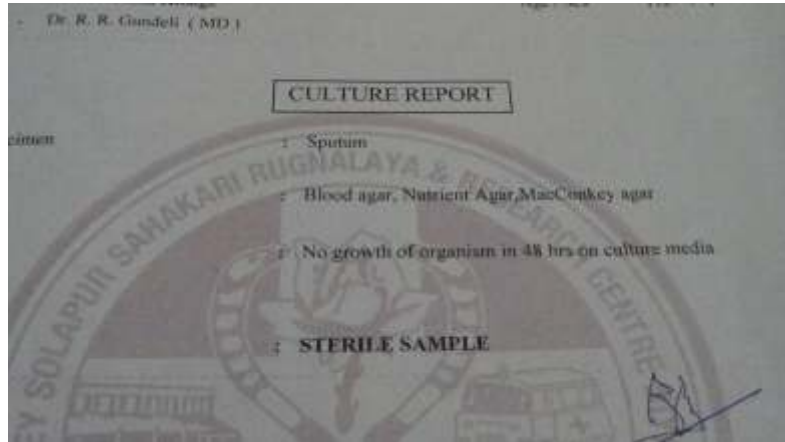
**X-Ray Report Report-2**



**X-Ray Report Report-3**



## Culture Report



Final P.L.No.19 Pachha Peth Solapur-413005 2D Echo Colour Doppler Report	
Age- 22 Years SEX- F	Ref: Dr. Gundeli MD.
<p><b>FINDINGS</b></p> <ul style="list-style-type: none"> <li>ALL CHAMBERS CHAMBERS NORMAL SIZED</li> <li>NO IMPACT</li> <li>NO MS/AS/MR/AR (72%)</li> <li>NO MS/AS/MR/AR</li> <li>NO MS/AS/MR/AR</li> <li>NO MS/AS/MR/AR</li> </ul> <p><b>IMPRESSION</b></p> <p>Normal ECHO and DOPPLER Study</p>	

### On Exam--

Conscious Febrile Tachypnoenic and in Ditrass.

Pulse--110/Min Blood Pressure--110/70 Spo2--88%On Room Air .Immediately Patient Was Taken on Bipap.

INIAL Reports Were Done. CXR Was Suggestive of Massive Pneumonitis On LT-side and Some Part of RT LZ Also. Patient Started With Higher Antibiotics And Other Supportive Measures. Next 12 Days She Was In ICU. Discharged on Day 17 With Full Recovery.

### **Discussion--**

ARDS in Pregnancy or Peripartum Period May Be Due To Routine Respiratory Infection or Pregnancy Related Disorder.

#### **Common Disorders are--**

- PULMONARY EDEMA
- COMMUNITY ACQUIRED PNEUMONIA
- ASPIRATION PNEUMONIA
- PULMONARY EMBOLISM
- ASTHMA EXACERBATION
- VENOUS EMBOLISM

### **Treatment---**

Usually Directed To Specific Cause. Supportive Care Involves Oxygenation And Ventilation, Hemodynamic Support, Fluid Management, Nutrition Support, Venous Thromboembolism Prophylaxis.

### **Conclusion:**

Little evidence exists regarding the management of ARDS specifically in pregnancy, and thus, treatment approaches must be drawn from studies performed in a general patient population. A multidisciplinary approach involving maternal-fetal medicine, neonatology, anesthesiology, and intensives clinicians is essential to optimizing maternal and fetal outcomes.

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## CARDIAC ARRHYTHMIAS

**Dr. Ravindra Gundeli (M.D.)**

Shri Markendeya Solapur Sahakari Rignalaya

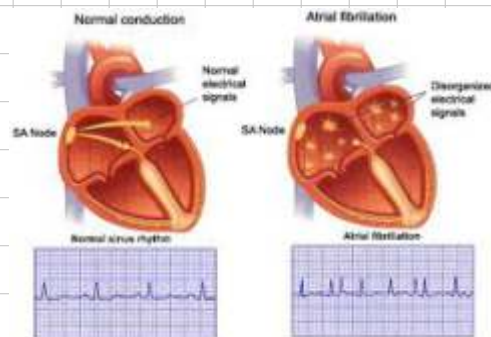
### Definition

Arrhythmias are disorders of impulse formation or disorders of impulse conduction or combination of both.

Mechanisms for arrhythmias in acute myocardial infarction:

### They Include -

- Autonomic nervous system imbalances
- Electrolyte disturbances
- Ischaemia or necrosis
- Slowed conduction



Activation of receptors within atrial or ventricular myocardium by ischaemic or necrotic tissue may cause enhanced efferent sympathetic activity, increased concentrations of circulating catecholamines and local release of catecholamines from nerve endings within the heart. The last phenomenon may also result from direct ischaemic damage of adrenergic neurons.

Experimental and clinical studies have suggested that electrolyte disturbance; elevated free fatty acid levels and oxygen-derived free radicals also contribute to development of arrhythmias. The severity of these abnormalities with size of infarction and perfusion status of infarct related artery, appear to determine a patient's risk for developing most serious rhythm disturbance - primary ventricular fibrillation.

Definite relationship between episodic and constant hypoxaemia, after acute myocardial infarction also proved in study done by Dr. Galatius Jensen S, Hansen J, et al.



On the contrary ventricular tachycardia was rarely initiated by an 'R' on 'T' types of Ventricular premature beats and its peak occurrence was at time when 'R' on T ventricular ectopic complexes were rare.

Thus it was concluded by end of study done by Dr. Ronald F. Cawbell, Allan Murray et al at university department of Cardiology, Freeman Hospital, New Castle - upon - Type - that within first 12 hours from onset of symptoms of acute myocardial infarction, the initiation of primary ventricular fibrillation and ventricular tachycardia is different. Primary ventricular fibrillation is typically 'R' on T initiated whereas almost all episodes of ventricular tachycardia are not.

## 2) Accelerated Idioventricular Rhythm

Commonly defined as a ventricular rhythm with a rate of 60 to 125 beats/min and frequently called 'slow ventricular tachycardia'.

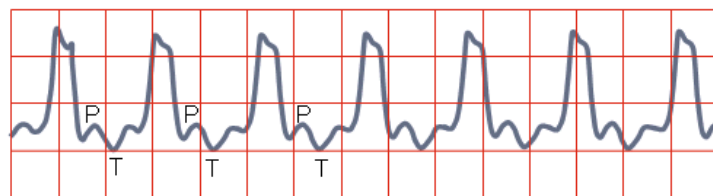
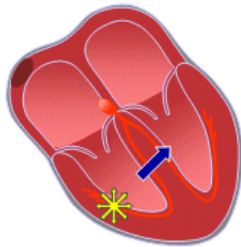
Occurs up to 20% of patients with acute myocardial infarction and occurs frequently during first 2 days with about equal frequency in anterior and inferior wall infarction and probably results from enhanced automaticity of perkinje fibres. Most episodes are of short duration and may terminate abruptly, slow gradually before termination or be overdriven by acceleration of basic cardiac rhythm. Variation in rate is common.

Accelerated idioventricular rhythms are thought not to affect prognosis in acute myocardial infarction and so most are left untreated except in rare patients with clear-cut haemodynamic compromise.

## 3) Ventricular Tachycardia

### VENTRICULAR TACHYCARDIA

Impulses originate at ventricular pacemaker



Wide ventricular complexes. Rate > 120/min

It is most frequently encountered life threatening arrhythmia. Its prompt recognition and acute treatment is critical.

It is defined as series of three or more ventricular complexes occurring at a rate of 100 - 250 beats/min, where the origin of activation is within ventricle.

Typically QRS complexes are wide (usually > 120 milliseconds).

Also other types classified depending upon duration of ventricular tachycardia.

Sustained Ventricular tachycardia - refers to similar above described rhythm but lasting longer than 30 seconds or cause haemodynamic compromise that requires intervention.

Or

Non-sustained Ventricular tachycardia - lasting < 30 seconds without

haemodynamic compromise.

Electrocardiogram appearance may be monomorphic or polymorphic. This may be important because former is more likely to be due to a myocardial scar and requires aggressive strategies to prevent its recurrence and later may be more responsive to measures directed against ischaemia.

Occurs up to 67% of patients in acute myocardial infarction in first 12 hours and hospital mortality with sustained ventricular tachycardia during first 48 hours following acute myocardial infarction is about 20%.

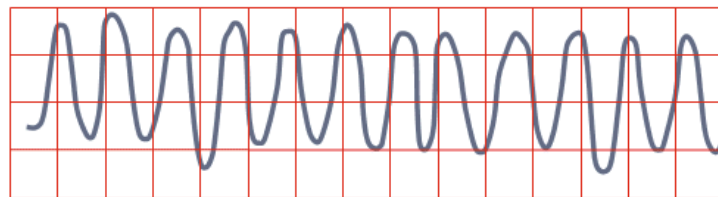
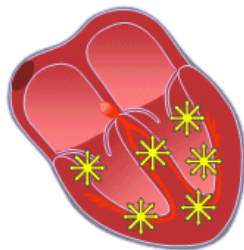
More common in patients, with transmural infarction and left ventricular dysfunction, in settings of acute anterior wall myocardial infarction. It again emphasizes the early monitoring of patients is beneficial to lower mortality in CCU.

One of important differentiating point already discussed was rarity of 'R' on T' types of Ventricular premature beats - its peak occurrence was significantly reduced in ventricular tachycardia.

#### 4) Ventricular Fibrillation

##### VENTRICULAR FIBRILLATION

Chaotic ventricular depolarization



Rapid, wide irregular ventricular complexes

Ventricular fibrillation is the result of rapid, repetitive activation of ventricles from multiple coalescing and fractionating wave fronts of depolarization. It is associated with disorganized mechanical contraction, haemodynamic collapse and sudden death.

Electrocardiogram reveals irregular and rapid oscillations (250-400 beats/min) of highly variable amplitude without uniquely identifiable QRS complexes or T waves. The combination of metabolic and electrolyte abnormalities, as well as neurophysiological and neurohumoral changes on the susceptible ischaemic myocardium is evolving as a fundamental electrophysiological concept for the mechanism of initiation of potentially lethal arrhythmias like ventricular fibrillation and ventricular tachycardia.

This arrhythmia is the commonest remediable cause of death in acute myocardial infarction and therefore emphasizes prompt treatment.

Occurs in three settings in hospitalized patients as below: a) Primary Ventricular Fibrillation Occurs suddenly and unexpectedly in patients with no or few signs or symptoms of left ventricular failure.

Primary ventricular fibrillation occurred in up to 10% of patients' hospitalized patients with acute myocardial infarction. Approximately 60% of episodes occur within 4 hours and 80% within 12 hours of onset of



### **Symptoms**

b)Secondary Ventricular Fibrillation On the other hand, it is often the final event of a progressive downhill course with left ventricular failure and cardiogenic shock.

c)Late Ventricular Fibrillation So called late as it develops more than 48 hours following acute myocardial infarction and frequently but not exclusively occurs in patients with large infarcts and ventricular dysfunction.

Other than above mentioned the group of patients who can still develop ventricular fibrillation are with intraventricular conduction defects and anterior wall infarcts with associated other supraventricular arrhythmias (which are discussed further). Also patients with right ventricular infarction who require ventricular pacing are at higher risk for developing late in hospital ventricular fibrillation.

### **Outcome Prognosis**

Effect of ventricular fibrillation on prognosis continues to be debated.

The MILIS study suggested that it does not have an adverse effect on hospital mortality where as first GISSI trial suggested excess in hospital mortality due to primary ventricular fibrillation.

On the other hand secondary ventricular fibrillation entails a very poor prognosis in hospital mortality rate of 40 to 60%

Other studies reported incidences of in hospital mortality are 50% and 54% respectively for primary and secondary ventricular fibrillation.

Approximately 20% of patients with acute myocardial infarction with primary ventricular fibrillation suffer a recurrence and mean time of recurrence is 4 days. Recurrence rate is nearly 50%.

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