Original Article

A study of blood gas analysis and electrolytes in 50 cases of chronic cor pulmonale

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Abstract

Abstract: The term 'cor pulmonale' if broken into its constituents cor (heart) and pulmonale (lungs) means cardiac involvement due to pulmonary disease. The term cor pulmonale was introduced in 1957 by Stuart Harris to denote right heart failure associated with a wide variety of diseases of the lung parenchyma, airways, thoracic cage and respiratory control mechanisms. The basic lesion in cor pulmonale is pulmonary hypertension. The clinical manifestations of cor pulmonale relate to alterations in cardiac output, salt and water homeostasis and in most cases, gas exchange in the lungs. **Aim and objectives**: of this study is to study the clinical features, and chest X-ray, electrocardiographic, echocardiographic features of chronic cor pulmonale. types of acid-base disturbances and serum electrolyte changes – their relation to blood gas abnormalities in chronic cor pulmonale. Fifty patients of chronic cor pulmonale were studied. Majority of patients were in the age group of 50-70 years. Cor pulmonale was seen predominantly in male patients and majority of patients were smokers, chronic bronchitis with emphysema was the major etiological factor. Majority of patients had obstructive defect on spirometry. Majority of patients were had hypoxia, hypercapnia and compensated chronic respiratory acidosis. Super imposed metabolic alkalosis were seen in significant number of patients. Serum sodium and potassium were normal in majority of patients with increasing PaCO2, the serum bicarbonate concentration progressively increased and the serum chloride concentration progressively decreased.
Keywords: Cor pulmonale respiratory acidosis; Metabolic alkalosis; Hypoxia; Hypercapnia

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INTRODUCTION

The term 'Cor pulmonale' if broken into its constituents Cor (heart) and pulmonale (lungs) means cardiac involvement due to pulmonary disease. The term cor pulmonale was introduced in 1957 by Stuart Harris to denote right heart failure associated with a wide variety of diseases of the lung parenchyma, airways, thoracic cage and respiratory control mechanisms. Stuart Harris attempts to emphasize hypertrophy of the right ventricle rather than actual cardiac failure. The basic lesion in cor pulmonale is pulmonary hypertension. The clinical manifestations of cor pulmonale relate to alterations in cardiac output, salt and water homeostasis, and in most cases, gas exchange in the lungs. Even with modem techniques for diagnosis and management, prognosis for the underlying disease has not been greatly improved. The prognosis for cor pulmonale however, is much better because of newer techniques for early recognition of the problem, better understanding of the role of blood gas and electrolyte disturbances, and the early application in many patients of measures to prevent or relieve pulmonary hypertension.

MATERIALS AND METHODS

This study consisted of 50 patients selected at random from the cases admitted to K.R. Hospital, Mysore between March 2013 and August 2013.All patients were evaluated with a detailed clinical history and thorough physical examination as per the proforma. The diagnosis of cor pulmonale was established by clinical, ECG, chest X-ray, and echocardiographic studies. Patients with associated metabolic, renal or endocrine disorders which can complicate the blood gas and electrolyte changes were excluded from the study. The routine blood, urine

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and sputum examinations were done for all patients. Pulmonary function tests were done using the computerized spirometer. Arterial blood gas analysis and serum electrolyte determinations were done simultaneously on the day of admission and repeated after 1 week for all patients. Arterial blood gas analysis was done immediately after collection of the sample by using the blood gas analyzer 'ABL 5' Radiometer, Copenhagen. The serum electrolytes sodium and potassium were estimated by flame photometric method. The chloride content was estimated by mercuric thiocyanate method.The patients were evaluated clinically at the time of blood gas and electrolyte determinations for the presence of the following complicating factors.

1. Rapid deterioration in the general condition of the patient due to acute pulmonary infection.
2. Rapid improvement in the general condition of the patient as a result of treatment.
3. Oxygen inhalation within 2 hrs. preceding the collection of blood.
4. Diuretic therapy without potassium supplementation during the week before sampling of blood.
5. Steroid therapy during the week before sampling of blood.
6. Alkalinization therapy during the week before sampling of blood.
7. Repeated vomiting.
8. Mechanical ventilation.
9. Administration of sedatives.

The results were plotted on the standard acid-base nomogram and the type of acid-base abnormality was determined.

CASE SUMMARIES

Case 1:

Mr. A, 70 year old male, agriculturist by occupation was admitted with history of. cough with expectoration since 10 years, progressive exertional breathlessness since 5 years and swelling of feet since 6 months. There was no history of chest pain or fever. Patient was not a known asthmatic and there was no past history of pulmonary tuberculosis. Patient was a chronic smoker of beedies for the past 20 years and had stopped smoking since 2 years.Patient was admitted repeatedly to the hospital for episodes of increasing breathlessness and swelling of limbs and treated. At the time of admission, patient was on oral steroids and loop diuretics. Examination revealed pedal edema. There was no cyanosis or clubbing. Pulse was 92/min, B.P. was 120/70 mmHg and respiratory rate was 28/min. JVP was not raised. Respiratory system examination showed a barrel shaped chest with diminished chest expansion. Breath sounds were normal. Rhonchi and crepitations were heard bilaterally. CVS examination was normal. There was no hepatomegaly. Routine blood and urine examinations were within normal limits. PFT showed an obstructive type of defect. ECG showed Right axis deviation, P pulmonale, Right ventricular hypertrophy, and incomplete RBSS. Chest X-ray showed Emphysematous chest, cardiomegaly enlarged right descending pulmonary artery and prominent pulmonary conus. Echocardiograph showed dilated right atrium and right ventricle and dilated main pulmonary artery.The blood gas and electrolyte estimations were done on admission and repeated after one week. The results were as follows:

Parameters	I	II
PO_2 (mmHg)	76	78
PCO_2 (mmHg)	68	62
pH	7.48	7.39
SO_2%	89	89
Base excess (m.mol/L)	+18	+9
HCO_3^- (m.mol/L)	.48	37
Na^+ (m.mol/L)	140.2	140.0
K^+ (m.mol/L)	3.2	4.2
Cl^- (m.mol/L)	86.4	88.4
Anion gap	5.8	15.2

The patient was diagnosed to have COPD with chronic cor pulmonale with congestive cardiac failure. On admission patient had chronic respiratory acidosis and superimposed metabolic alkalosis indicated by raised pH, bicarbonate and base excess. Serum sodium was normal, chloride was low and patient had hypokalemia. Hypokalemia and metabolic alkalosis was probably induced by diuretic therapy without potassium supplementation and by steroid therapy. Potassium chloride supplementation was given and steroids were tapered and stopped. After 1 week, the serum potassium and pH became normal and metabolic alkalosis disappeared. This shows the beneficial effect of potassium chloride in treating alkalosis in patients with chronic cor pulmonale.

Case 2:

Mr. B, aged about 50 years, agriculturist by occupation was admitted with complaints of cough with expectoration of 6 years· duration and progressive exertional breathlessness of 4 years duration, swelling of feet since 1 month and fever since 1 week. Patient gave H/O dull aching retrosternal chest pain not related to exertion. Since 1 week, cough had increased and sputum had become mucopurulent. There was no haemoptysis. Patient was a chronic smoker of beedies for the last 15 years. There was no H/O Tuberculosis or bronchial asthma. On examination patient was febrile, central cyanosis and pedal oedema were present, and there was no clubbing. Pulse rate was 104/min, BP 110170 mmHg,

respiratory rate was 36/min. JVP was raised. Respiratory system examination showed a barrel shaped chest with decreased movements. Breath sounds were diminished in intensity and rhonchi and crepitations were heard bilaterally. CVS examination showed parasternal heave, epigastric pulsations of right ventricular type, loud P_2 right ventricular S_3 and tricuspid regurgitation. Patient had tender hepatomegaly. CNS examination was normal. The routine blood and urine examinations were normal and sputum for AFB was negative. Sputum culture and sensitivity revealed E. coli sensitive to Gentamicin. Pulmonary function test showed an obstructive type of defect. ECG showed Right axis deviation, 'P' pulmonale, right ventricular hypertrophy and clockwise electrical rotation. Chest X-ray showed Emphysematous chest, borderline cardiomegaly, prominent pulmonary conus and upper lobe diversion of pulmonary veins. Echocardiography showed dilated right atrium and right ventricle, and dilated main pulmonary artery. The patient was diagnosed to have chronic cor pulmonale in congestive cardiac failure secondary to COPD. The arterial blood gas analysis and serum electrolyte estimations were done on the day of admission and repeated after 1 week. The results are as follows.

Parameters	I	II
PO_2 (mmHg)	60	74
PCO_2 (mmHg)	66	54
pH	7.28	7.40
SO_2%	83	89
Base excess (m.mol/L)	+1	+8
HCO_3^- (m.mol/L)	28	34
Na^+ (m.mol/L)	140.2	144.0
K^+ (m.mol/L)	4.8	4.8
Cl^- (m.mol/L)	99.2	94.8
Anion gap	**13.0**	**15.2**

Thus on admission, patient had hypoxia, hypercapnia and acute respiratory acidosis superimposed on chronic respiratory acidosis. The serum electrolytes and anion gap were within normal limits. Patient was treated with antibiotics, bronchodilators, diuretics with potassium supplementation, and oxygen. Patient improved with the above treatment and after 1 week, showed chronic compensated respiratory acidosis with increased PO_2, decreased PCO_2. pH returned to normal range. A rise in the bicarbonate levels and a positive base excess indicates compensatory metabolic alkalosis. The serum sodium and potassium concentration remained normal but there was a fall in the serum chloride concentration. Patient was discharged with the advice to stop smoking and to continue bronchodilators and diuretics with potassium supplementation.

Case 3: Mr. C, aged about 61 years, manual labourer by occupation was admitted with history of cough with expectoration since 10 years, progressive exertional breathlessness since 6 years, swelling of feet since 15 days, fever and chest pain since 8 days. Sputum was mucopurulent, copious. There was no haemoptysis. Patient was a chronic smoker of beedies for the past 25 years. There was no history of tuberculosis or bronchial asthma. On examination patient was febrile, central cyanosis and pedal oedema were present and there was no clubbing. The pulse rate was 118/min, blood pressure was 116/80 mmHg and the respiratory rate was 28/min. JVP was raised. Respiratory system examination revealed a barrel shaped chest with diminished movements. Breath sounds were diminished in intensity and rhonchi and crepitations were heard bilaterally. CVS examination showed a parasternal heave, epigastric pulsation of right ventricular type, a loud P_2, right ventricular S_3 and tricuspid regurgitation. Patient had tender hepatomegaly. Patient was drowsy and flapping tremors were present. There was no papilloedema. The routine blood and urine examinations were normal, sputum culture revealed klebsiella sensitive to ciprofloxacin. PFT showed an obstructive type of defect. ECG showed Right axis deviation, P pulmonale and RVH with strain. Chest X-ray showed Emphysematous chest, right lower zone pneumonitis, RV type of cardiomegaly prominent pulmonary conus and upper lobe diversion of pulmonary veins. Echo cardiography showed right ventricular enlargement, right atrial enlargement, dilated main pulmonary artery and tricuspid regurgitation. The patient was diagnosed to have COPD with chronic cor pulmonale with congestive cardiac failure and acute respiratory failure. The blood gas and electrolyte estimations were as follows.

Parameters	I	II
PO_2 (mmHg)	62	66
PCO_2 (mmHg)	92	76
pH	7.10	7.42
SO_2%	79	84
Base excess (m.mol/L)	-2	+18
HCO_3^- (m.mol/L)	26	45
Na^+ (m.mol/L)	135.2	141.4
K^+ (m.mol/L)	5.8	4.3
Cl^- (m.mol/L)	94.2	90.2
Anion gap	**15**	**6.2**

Thus on admission, patient had hypoxia, hypercapnia, and acute respiratory acidosis superimposed on chronic respiratory acidosis. Serum sodium and bicarbonate concentrations were normal, chloride was low and patient had hyperkalemia. Patient was treated with antibiotics, bronchodilators, diuretics without potassium supplementation, oral sodium bicarbonate and oxygen. Patient improved significantly and the sensorium improved and flapping tremors disappeared. The serum potassium concentration became normal and there was a rise in serum bicarbonate and a fall in serum chloride

concentration. A plot on the acid-base nomogram showed that the patient had developed a superimposed metabolic alkalosis probably induced by diuretic and alkalinization therapy, and posthypercapnic alkalosis. Alkalinization therapy was stopped and potassium chloride supplementation was given. After an initial improvement the patient's general condition rapidly deteriorated with increasing cough and sputum production and the patient expired 13 day after admission.

OBSERVATIONS AND RESULTS

Table 1: Age distribution

Age group (years)	Male	Female	Total	Percentage
30 – 39	-	1	1	2
40 – 49	7	1	8	16
50 – 59	19	-	19	38
60 – 69	14	2	16	32
70 – 79	5	-	5	10
80 – 89	1	-	1	2

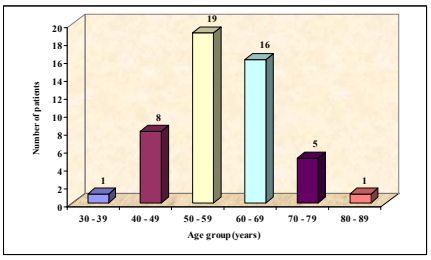

Figure 1: Age distribution

Age of patients ranged from 35 years to 86 years with a mean age of 56.5±10.4 years.

Table 2: Sex distribution

Sex	Number of cases	Percentage
Male	46	92
Female	4	8

Majority of patients were males. The male: female sex ratio was 11.5:1.

Table 3: Smoking Habits

Habits	Number of patients	Percentage
Smokers	44	88
Non-smokers	6	12

Majority of patients were smokers. All females were non-smokers.

Table 4: Presenting symptoms

Symptom	Number of patients	Percentage
Cough	50	100
Breathlessness	50	100
Fever	7	14
Swelling of limbs	28	56
Chest pain	4	8

Cough and breathlessness were present in all patients. Swelling of limbs was present in 56% of patients 3 patients had bronchial asthma and 3 patients gave past history of pulmonary tuberculosis.

Table 5: Physical signs

Signs	Number of patients	Percentage
Cyanosis	18	36
Clubbing	6	12
Pedal Oedema	31	62

Cyanosis and pedal oedema were present in majority of patients.

Table 6: Respiratory Signs

Signs	Number of patients	Percentage
Tachypnoea	28	56
Barrel shaped chest	34	68
Tracheal deviation	3	6
Diminished chest expansion	28	56
Diminished breath sounds	26	52
Bronchial breathing	3	6
Rhonchi	42	84
Crepitations	50	100

Majority of patients had barrel shaped chest, diminished chest expansion, rhonchi and crepitations.

Table 7: Cardiovascular findings

Signs	Number of patients	Percentage
Tachycardia	9	18
Raised JVP	21	42
Left parasternal heave	23	46
Epigastric pulsations	36	72
Loud P_2	31	62
Right ventricular S_3	7	14
Tricuspid regurgitation	11	22

Majority of patients had raised JVP, left parasternal heave, epigastric pulsation and loud P_2 17 patients (34%) had hepatomegaly, 2 patients (4%) had ascites and 4 patients (8%) had flapping tremors.

Table 8: Types of diseases

Pulmonary disease	Number of patients	Percentage
Chronic bronchitis with emphysema	36	72
COPD with Tuberculosis	3	6
COPD with Bronchiectasis	4	8
Post-tubercular fibrosis	2	4
Bronchial asthma	3	6
Bronchiectasis	2	4

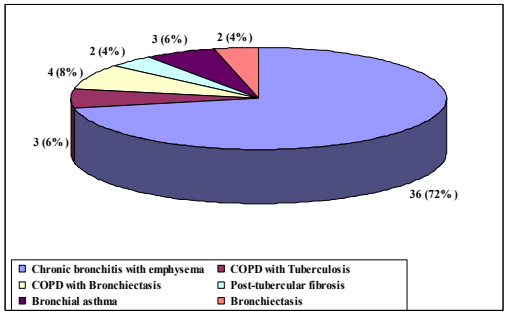

Figure 2: Types of diseases

In majority of patients, chronic cor pulmonale was due to chronic bronchitis with emphysema.

Investigations

All patients had normal haemoglobin levels. Sputum for AFB was positive in one patient. FBS, blood urea and serum creatinine were normal in all patients.

Table 9: Chest X-ray findings

X-ray findings	Number of patients	Percentage
Chronic bronchitis with emphysema	43	86
Fibrothorax	2	4
Bronchiectasis	6	12
Right ventricular hypertrophy	18	36
Prominent pulmonary conus	28	56

Majority of patients had chronic bronchitis with emphysema on chest X-ray. Significant number of patients showed prominent pulmonary conus.

Table 10: ECG findings

ECG signs	Number of patients	Percentage
'P' pulmonale	28	56
Right axis deviation	21	42
Right ventricular hypertrophy	16	32
Clockwise rotation	17	34
Right bundle branch block	2	4

P pulmonale and right axis deviation were the commonest ECG findings.

Table 11: Pulmonary function tests

Type of abnormality	Number of patients	Percentage
Obstructive	48	96
Restrictive	2	4

Majority of patients had obstructive type of defect.

Table 12: Echocardiography findings

RV chamber size (cms)	Number of patients	Percentage
2.6 - 3.0	9	18
3.1 - 3.5	16	32
3.6 - 4.0	10	20
4.1 - 4.5	11	22
4.6 - 5.0	4	8

All patients showed right ventricular enlargement. Main pulmonary artery dilatation was seen in 34 (68%) of patients.

Acid-base disturbances

One hundred blood gas determinations were done on 50 patients. The types of acid-base disturbances seen in patients with chronic cor pulmonale were as follows.

Table 13: Acid-base disturbances

Type of acid-base disturbance	Number of determinations	Percentage
Normal	14	14
Chronic respiratory acidosis	64	64
Chronic respiratory acidosis with superimposed metabolic alkalosis	13	13
Chronic respiratory acidosis with superimposed acute respiratory acidosis	9	9

Metabolic alkalosis was the commonest superimposed acid base abnormality. The incidence of complicating factors in patients with metabolic alkalosis were as follows

Table 14: Complicating factors

Complicating factor	Number	Percentage
Recent recovery from acute exacerbation	5	26.3
Diuretic therapy without potassium supplementation	10	52.6
Steroid therapy	2	10.5
Alkalinization therapy	1	5.26
Repeated vomiting	1	5.26

The commonest cause of superimposed metabolic alkalosis was diuretic therapy without potassium supplementation. Out of 5 patients who had superimposed metabolic alkalosis on admission, 4 patients who took potassium chloride therapy showed disappearance of metabolic alkalosis whereas one patient who failed to take potassium chloride therapy persisted to have metabolic alkalosis.

Arterial blood gases

Out of 100 blood gas determinations, 78 determinations were done on patients who were in stable state without superimposed acid-base abnormality. The blood gas values in this group of patients were as follows:

Table 15: Blood gas estimation before treatment

Parameters	Minimum	Maximum	Mean
PaO_2 mmHg	52	98	75.69±11.75
$PaCO_2$ mmHg	35	80	54.88±10.62
pH	7.29	7.45	7.39±0.03
SO_2%	76	98	87.71±5.03
Base excess m.mol/L	-3	+16	7.24±3.24

Thus patients with chronic cor pulmonale in stable state will have hypoxia, hypercapnia, pH within normal range and a positive base excess indicating the presence of compensatory metabolic alkalosis. Nine blood gas determinations were done on patients who were in acute exacerbation on admission. Blood gas estimation was repeated after treatment when the patients reached a stable state. The results are tabulated below.

Table 16: Blood gas estimation after treatment

Parameters	Acute exacerbation	Stable state	P value
PaO_2 mmHg	63.44±5.77	74.89 ± 7.15	< 0.01
$PaCO_2$ mmHg	70.44±12.16	59.11±11.40	< 0.05
pH	7.24±0.08	7.42±0.05	< 0.001
SO_2%	82.55±3.32	88.45±2.29	< 0.001
Base excess m.mol/L	2.44±1.58	7.24±3.24	< 0.001

Thus with treatment, there was a significant increase in PaO_2, oxygen saturation of haemoglobin and a significant decrease in $PaCO_2$. The pH increased to normal range with a positive base excess indicating the development of compensatory metabolic alkalosis.

Serum electrolytes

Sodium: The serum sodium concentration was normal in majority of patients with a mean concentration of 139.53±2.94 m.mol/L. Hyponatremia was seen only in 3 determinations and hypematremia was not encountered in this study.

Potassium: Majority of patients had normal serum potassium levels with a mean concentration of 4.23±0.68 m.mol/L. Hyperkalemia was seen in only 3 patients who were in acute exacerbation. Hypokalemia was seen in 4 patients and all had superimposed metabolic alkalosis.

Chloride: The serum chloride concentration ranged from 86 m.mol/L to 104.6 m.mol/L with a mean concentration of 95.98±6.55. Majority of patients showed hypochloremia. Hyperchloremia was not seen in any determinations.

Bicarbonate: The serum bicarbonate levels ranged from 21-48 m.mol/L with a mean concentration of 33.27±5.64. Majority of patients showed increased bicarbonate levels. Anion gap was normal in majority of patients. The serum electrolyte changes in 9 patients who were in acute exacerbation on admission before and after treatment were as follows.

Table 17: Serum electrolyte

Electrolyte	Acute exacerbation	Stable State	P value
Sodium (m.mol/L)	139.06±3.21	139.96±2.69	> 0.05
Potassium (m.mol/L)	4.89±0.82	4.29±0.53	> 0.05
Chloride (m.mol/L)	98.16±2.54	93.58±4.71	< 0.05
Bicarbonate (m.mol/L)	28.44±1.88	35.45±5.66	< 0.01

Thus the serum sodium and potassium concentrations did not change significantly. The serum chloride concentration decreased significantly and the serum bicarbonate concentration increased significantly after treatment.

Relation between blood gases and electrolytes

Out of 100 blood gas and electrolyte estimations, 78 determinations were done on patients who were in stable state without superimposed acid-base disturbances. The relation between blood gases and electrolytes was seen in this group of patients.

Table 18: Effect of hypercapnia

$PaCO_2$ (mmHg)	No.	pH	Cl^- (m.mol/L)	HCO_3^- (m.mol/L)
35-44	14	7.41±0.03	102.02±1.97	24.35±2.13
45-54	29	7.41±0.05	97.75±5	32.97±2.93
55-64	22	7.40±0.05	93.06±2.74	35.88±3.47
65-74	9	7.38±0.04	91.01±2.93	39.45±3.64
75-84	4	7.35±0.04	90.48±6	41.20±4.96

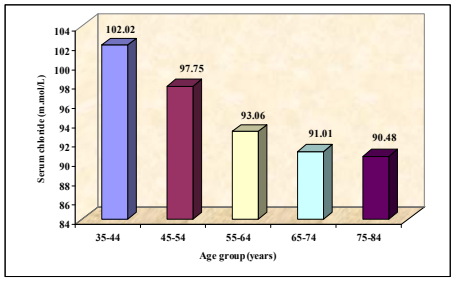

Figure 3: Relation between $PaCO_2$ and Serum chloride concentration

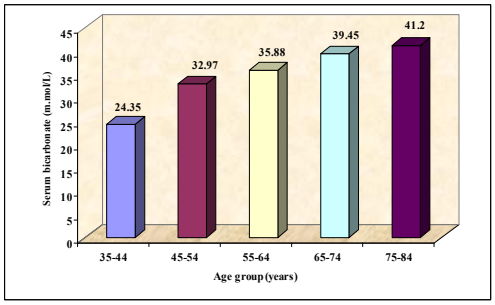

Figure 4: Relation between $PaCO_2$ and Serum bicarbonate concentration

With increasing degrees of hypercapnia, serum chloride concentration gradually decreased and the serum bicarbonate concentration gradually increased. Although the pH gradually decreased with increasing $PaCO_2$, if was maintained within the normal range. Thus the renal compensation is adequate throughout the range of hypercapnia.

Table 19: Effect of hypoxia

PaO_2 (mmHg)	No.	pH	HCO_3^- (m.mol/L)	Cl^- m.mol/L)
50-59	8	7.38±0.04	34.87±2.99	94.07±4.81
60-69	14	7.37±0.03	37.94±4.94	93.20±3.10
70-79	24	7.40±0.02	33.96±3.15	95.39±4.23
80-89	19	7.41±0.01	34.64±5.95	93.92±13.42
90-99	13	7.40±0.02	27.28±5.46	100.71±3.27

Thus, hypoxia had no relation to pH, bicarbonate and chloride levels in chronic cor pulmonale.

DISCUSSION

Fifty cases of chronic cor pulmonale were studied.

The mean age group in this study was 56.5 ± 10.4 years.

Table 20: Comparison of age distribution

Studies	20-29	30-39	40-49	50-59	60-69	70-79	80-89
Misra and Padmavathi (1969)	15.4	31.3	46.9	3.1	3.1	-	-
Present Study	-	2	16	38	32	10	2

The present study shows a maximum incidence between age groups 50-59 years whereas Misra and Padmavathi (1969) noticed a maximum incidence between 40-49 years. Gupta *et al*. (1989) reported a mean age of 50.2±12 years. **Sex:** In the present study, males accounted for 92% of cases with a male: female sex ratio of 11.5:1.

Table 21: Comparison of sex distribution

Studies	Male (%)	Female (%)
Flint (1954)	84	16
Padmavathi (1959)	54	46
Basavaraj Urs and Shankar (1965)	83	17
Bhargava (1971)	77	23
Chatterjee *et al* (1971)	83	17
Gupta *et al* (1989)	96	4
Present study	92	8

Thus all studies indicate a high incidence of chronic cor pulmonale in males. **Smoking:** In the present study, 88% of patients were smokers. All females were non-smokers. Gupta *et al* (1989) studied 30 patients with cor pulmonale and all were smokers. Thus in all studies smoking was a major risk factor.

Table 22: Comparison of etiological factors

Etiological factors	Padmavathi (1959)	Padmavathi and Misra (1969)	Present study
Chronic bronchitis with emphysema	59.8	43.4	86
Pulmonary tuberculosis	5.5	7.0	4
Bronchial asthma	13.4	5.1	6
Bronchiectasis	17.3	43.2	4

The incidence of chronic bronchitis with emphysema in chronic cor pulmonale has been reported as 79.4% by Vakil (1948), 74.4% by Vishwanathan *et al* and 92% by Basavaraj Urs and Shankar (1965). Thus in all studies chronic bronchitis with emphysema forms the major etiological factor in causing chronic cor pulmonale.

Clinical features

In the present study, cough and breathlessness were present in all patients. Cyanosis was present in 36%, clubbing in 12% and pedal edema in 62% cases. According to Bhargava *et al* (1971) and Vishwanathan, breathlessness, cough with expectoration and edema were present in 100% of cases. Clubbing was reported as 31% by Vishwanathan and 70% by Bhargava (1971), Padmavathi (1959) reported dyspnea in 100% cases, edema in 90.4% and cyanosis in 83.2% cases. According to Gupta *et al* (1989) cyanosis was seen in 50% of cases. Ghosh *et al* (1988) reported cough and breathlesness in all the 25 cases studied, Chest pain in 20% and haemoptysis in 16% cases.

Table 23: Comparison of incidence of respiratory findings

Finding	Gupta *et al* (1989)	Ghosh *et al* (1988)	Present study
Tachypnoea	100%	*	56%
Barrel shaped chest	66%	40%	68%
Diminished chest expansion	80%	80%	56%
Rhonchi	80%	50%	84%
Crepitations	*	40%	100%
Bronchial breathing	*	36%	6%

* Not mentioned

Thus in all studies majority of patients had tachypnoea, barrel shaped chest, diminished chest expansion, rhonchi and crepitations.

Table 24: Comparison of incidence of cardiovascular findings

Cardiovascular findings	Gupta *et al* (1989)	Present study
Left parasternal heave	50%	46%
Epigastric pulsation	100%	72%
Loud P_2	57%	62%
Tricuspid regurgitation	7%	22%

Padmavathi (1959) reported loud P_2 in 65% cases. Thus in all studies majority of patients had left parasternal heave, epigastric pulsation and loud P_2. Raised JVP was seen in 42% of patients in the present study. Padmavathi (1959) reported raised JVP in 88% cases.

Table 25: Chest X-ray

Findings	Gupta *et al* (1989)	Ghosh *et al* (1988)	Present
Chronic Bronchitis with emphysema	53.3%	56%	86%
Fibrosis	13.3%	32%	4%
RV enlargement	20%	*	36%
Prominent pulmonary conus	36.7%	*	56%
Bronchiectasis	13.3%	4%	12%

* Not mentioned

Padmavathi (1959) reported prominent pulmonary conus in 75% cases. Thus the major X-ray findings in all studies were chronic bronchitis with emphysema and prominent pulmonary conus.

Table 26: Comparison of ECG

Findings	Padmavathi (1969)	Gupta *et al* (1989)	Present study
Right axis deviation	78%	33.3%	42%
RV hypertrophy	87.5%	50%	32%
P-pulmonale	85%	43.3%	56%
Clockwise rotation	*	27.3%	34%

Right bundle branch block	*	6.7%	4%

* Not mentioned

Thus in all studies the important ECG findings were right axis deviation and 'P' pulmonale. The incidence of right ventricular hypertrophy was less in the present study

Pulmonary function tests

96% of patients showed obstructive defect and 4% patient showed restrictive defect on spirometry. Gupta *et al* (1989) reported obstructive defect in 96.6% cases and restrictive defect in 3.4% of cases. Chandra Mohan (1991) reported obstructive defect in 85% cases and restrictive defect in 15% cases. Echocardiography: All patients had right ventricular hypertrophy and dilated main pulmonary artery was seen in 68% of cases. Ghosh *et al* (1988) reported an increase in the internal diameter of the right ventricle in all patients and 60% had pulmonary artery dilatation. Thus echocardiography was more sensitive in detecting RVH than X-ray and ECG in cases of cor pulmonale. Acid-base disturbances: One hundred blood gas determinations were done on 50 patients. Acid-base abnormalities were seen in 86% determinations. Majority of patients (64%) had chronic respiratory acidosis. A superimposed metabolic alkalosis was seen in 13% of determinations.

Table 27: Acid-base disturbances

Study	Superimposed metabolic alkalosis (%)
Eugene D. Robin (1963)	12
L. Brasseur *et al* (1966)	4
Guleria *et al* (1970)	15.4
Present study	13

Potassium chloride therapy had a significant role in the treatment of patients with superimposed metabolic alkalosis. The role of potassium chloride has been documented in the case reports of Frank Q. Nuttall (1965), Eugene D. Robin (1963), Robert T. Cochran (1963) and R.A. Ghose (1964).

Arterial blood gases: Seventy-eight determinations were done on patients who were in stable state without superimposed acid-base abnormality. The PaO_2 ranged between 52-98 mmHg (mean 75.69 ± 11.75), $PaCO_2$ between 35-80 mmHg (mean 54.88 ± 10.62) and pH between 7.29-7.45 (mean 7.39 ± 0.03). According to Gupta *et al*, the PaO_2 ranged between 41-94 mmHg (mean 57.5 ± 17.1 mmHg). $PaCo_2$ between 36-68 mmHg (mean 51.9 ± 9.8 mmHg) and pH between 7.34 to 7.51 (mean 7.41 ± 0.05).Thus majority of patients with cor pulmonale had hypoxia, hypercapnia and normal pH. In 9 patients who were in acute exacerbation, there was a significant increase in PaO_2, oxygen saturation of haemoglobin and a significant decrease in $PaCO_2$ with treatment. The pH increased to normal range with a positive base excess indicating the development of compensatory metabolic alkalosis.

Serum electrolytes

Sodium and potassium concentrations were normal in majority of patients. Hyperkalemia was seen only in patients with severe respiratory acidosis. All patients with hypokalemia had a superimposed metabolic alkalosis. The serum sodium and potassium were reported to be normal in patients with chronic respiratory acidosis by Eugene D. Robin (1963)8 and Newton C. Brackett (1969). In 1958, Schwartz noticed hyperkalemia in 5 of 15 patients with cor pulmonale. J.S. Guleria *et al* (1970) noticed hypokalemia in 6.6% cases. With increasing $PaCO_2$, the serum chloride concentration progressively decreased and the serum bicarbonate concentration progressively increased. A similar relation has been described in the studies conducted by Eugene D. Robin (1963), L. Brasseur *et al* (1966), Newton C. Brackett *et al* (1969) and J.S. Guleria *et al* (1970). Hypoxia had no relation to pH, bicarbonate and chloride levels in chronic cor pulmonale. In 1967, Daniel G. Sapir *et al* showed that hypoxia had no significant influence on electrolyte and acid-base balance in normocapnia and chronic hypercapnic states. With increasing $PaCO_2$, the pH decreased progressively but was within the physiological range indicating the presence of effective renal compensation throughout the range of increasing $PaCO_2$.This inverse relationship between $PaCO_2$ and pH has been observed by Eugene D. Robin (1963), Mauracio J. Dulfano *et al* (1966), Newton C. Brackett *et al* (1969) and Guleria *et al* (1971) but above the PCO_2 of 6570 mmHg, renal compensation was inadequate and pH was in the acidotic range. A study of serum electrolyte profile before and after treatment in 9 patients who were in acute exacerbation on admission showed that there was no significant change in the sodium and potassium concentration after treatment. Serum chloride concentration decreased and bicarbonate concentration increased which were statistically significant

CONCLUSIONS

1. Majority of patients with chronic cor pulmonale will have chronic respiratory acidosis. Superimposed acid-base disturbances are common of which the commonest is metabolic alkalosis.
2. The serum sodium and potassium were normal in majority of patients. With increasing $PaCO_2$, the serum bicarbonate concentration progressively increased and the serum chloride concentration progressively decreased.
3. The renal compensation was adequate to

maintain a normal pH throughout the range of $PaCO_2$ in the chronic stable state.

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