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Case Study

Role of 'Aviptadil' in Treatment of Acute Respiratory Distress Syndrome (ARDS): Case Series

Sagar Gupta[1], Sudhir Deshpande[2], Kuldeepsing Raul[3], Vishram Pramanik[4], Kapil Navpute[5], Omprakash Solanke[6]

[1]Assistant Professor, [5]Senior Resident, Shri Ramchandra Institute of Medical Sciences, R K Damani Medical College & Dr Hedgewar Hospital Chh Sambhajinagar, Maharashtra, India
[2]Chief Intensivist, [4]Intensivist, [6]DNB Junior Resident, Dr Hedgewar Hospital Chh Sambhajinagar, Maharashtra, India
[3]Professor & HOD, Department of Internal Medicine, Shri Ramchandra Institute of Medical Sciences, R K Damani Medical College & Dr Hedgewar Hospital Chh Sambhajinagar, Maharashtra, India
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I N F O

Corresponding Author:
Sagar Gupta, Shri Ramchandra Institute of Medical Sciences, R K Damani Medical College & Dr Hedgewar Hospital Chh Sambhajinagar, Maharashtra, India
E-mail Id:
sagargupta2303@gmail.com
Orcid Id:
https://orcid.org/0000-0002-9378-1701
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A B S T R A C T

As we know respiratory failure is a lethal complication of viral illnesses that has remained resistant to conventional treatment and ventilator support. Acute respiratory distress syndrome is life-threatening scenario that requires ICU admissions. Treating this condition requires proper oxygenation strategies with mechanical ventilation and positive pressure (PEEP) to recruit alveoli.

Vasoactive intestinal peptide (Aviptadil) is shown in literature to up-regulate surfactant production, inhibit cytokine synthesis, prevent cytopathic effects and block replication of viral cells in pulmonary cells. Human vasoactive intestinal peptide prevents further lung injury and reduces inflammation. We share our observation and experience via this case series regarding effective and safe use of aviptadil for treating ARDS arising from viral illnesses and others apart from its previous benefits found in COVID19 related ARDS cases[1,2]

Keywords: Acute viral illness, Acute lung injury, ARDS, Acute Respiratory Distress Syndrome, Cytokines, Lung alveolar cells type II, Surfactant replacement, Vasoactive intestinal peptide (Aviptadil)

Introduction

The acute illness known as Acute Respiratory Distress Syndrome (ARDS) is associated with severe respiratory failure and the need for mechanical ventilation. The causes of acute respiratory distress syndrome (ARDS) are diverse and include infections, pneumonia, and trauma, inhalation injuries, and sepsis[3].ARDS mortality rates are still very high, and the outlook is still depressing despite improvements in medical therapies. Even though they offer life support, conventional medicines frequently fail to address the syndrome's underlying complications.

We recognise that aviptadil does not represent a definitive therapy for ARDS but rather serves as an adjunct, and probably helps in buying valuable time until a more comprehensive and definitive treatment takes effect.

In this case series we illustrate the beneficial effects of Aviptadil use in 4 instances of moderate to severe noncovid related ARDS. Aviptadil is a synthetic form of human vasoactive intestinal peptide (VIP) which helps in reducing lung injury by all four possible mechanisms as mentioned below in the chart.

The use of aviptadil has been found in different respiratory conditions such as asthma, chronic obstructive airway disease, cystic fibrosis, pulmonary hypertension, ARDS and sarcoidosis.[1,3,4]

Aviptadil has been identified as a useful tool as an orphan drug by the USFDA in 2001 and the EMA in 2006. It also achieved emergency approval in India from the CDSCO in April 2022 for covid-19 associated ARDS.[3]

Mechanisms causing low oxygenation in ARDS

This is primarily achieved by restoring the function of damaged alveolar epithelial type 2 cells, leading to increased surfactant production and reduced shunting.

Aviptadil exerts anti-inflammatory actions that mitigate interstitial damage and enhance gas diffusion[3] Fig 1.

Clinical Case 1

A 40-year-old female resident of a small village presented to the emergency department at our hospital with presentation of experiencing a dry cough, cold and fever for a week associated with shortness of breath worsening over a period of the last 3 to 4 days. On admission day1 she had mild tachypnea, was afebrile, and had a mild cough with scanty sputum. Her respiratory distress was significant necessitating high levels of oxygen support as she required oxygen by nasal cannula at 4 liter per minute with admission in ICU.

As her O2 requirement increased and condition worsened, later she was put on HFNC support with prone position and frequent change of posture. Simultaneously she was advised to do spirometry & deep breathing exercise.

On Day 1 – her lab investigations were – Hb 11.8, WBC – 2000, and Platelet – 164000. Total Bilirubin – 0.3, SGOT – 87, SGPT – 28, ALP – 54, Urea 8.5, Creatinine – 0.5, Na – 137 – K – 3.3. Procalcitonin – 0.18, CRP -73.8, Sputum microscopy – gram + cocci, sputum for AFB – negative. HIV/HBsAg/HCV – negative. On ABGA – pH 7.5, PCO2 28, pO2 51, Lactate 0.9, SPO2 91.6, HCO3 25.

On Day 4 – Hb 11.2, WBC – 3080, Platelet – 234000. Creatinine –0.5, pH –7.5, po2–56, pCO2–29.9, SO2–95, lactate 0.6.

Her chest x-ray (Table 1) revealed bilateral scattered infiltrates in both lungs suggestive of atypical pneumonia ARDS. Her HRCT (Table 1) chest imaging showed bilateral extensive ground glass opacities (GGO) on the 3rd day of presentation. Her 2D echo showed normal ejection fraction of 60% and a normal IVC having a typical respiratory pattern.

She was treated with antibiotics covering both typical and atypical bacteria considering community acquired pneumonia.

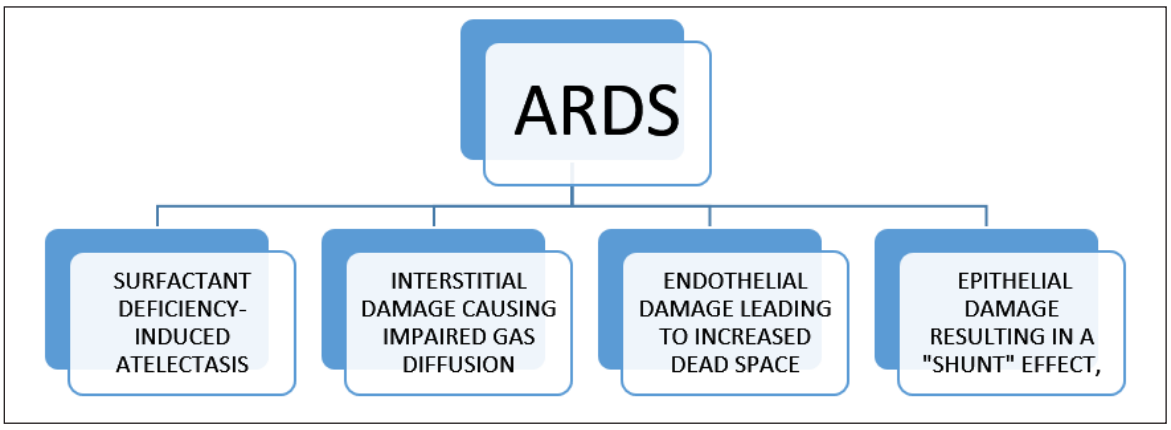

Figure 1.Pathophysiological Mechanisms of Acute Respiratory Distress Syndrome (ARDS)

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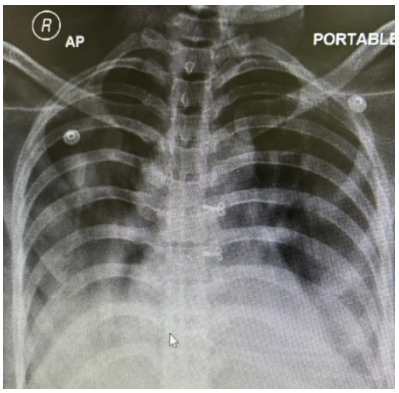
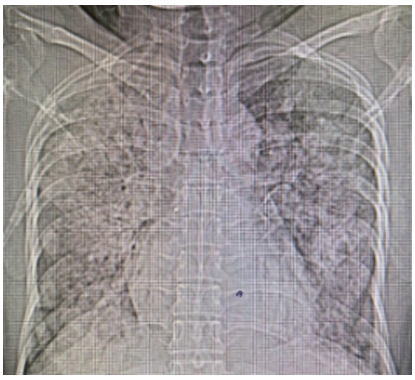
Table 1.CHEST –X ray and HRCT CASE - I

DAY 1	DAY 4
	

Over three successive consecutive days Inj. AVIPTADIL infusion was given at escalating doses of 0.166, 0.332, and 0.498 mcg/kg/hour for 10 hours each. Later there was a reduction in oxygen requirement over the next three days. Gradually she became better on 5th day and was weaned from HFNC on day 6th and put on nasal prongs wit 1 to 2 L per min O2 on the day 7 shifted to the ward from ICU.

The patient improved clinically within a period of next 5 days with reduced oxygen requirement and ventilator support.

PA View Suggestive of Bilateral Scattered Infiltrates Suggestive of Atypical Pneumonia

Clinical Case 2

A-33-year-old male patient presented to the emergency department with shortness of breath of insidious onset over 3 days with a recent history of surgery for fracture tibia fibula and on the left side secondary to road traffic accident which was managed at outside hospital. On examination: HR: 130/min, Temp: 102 F, BP: 150/90 mm hg, right arm supine position. His SPO2 was 60% on NRBM mask at the presentation hence he was admitted at the ICU. He was suspected of having postoperative pulmonary thromboembolism due to limb immobilization or viral ARDS as he had cough, cold, fever and breathlessness for 03 days after discharge.

Investigations: on Day 1 - Hb – 11.1, WBC – 7400, Platelet 122000, N- 80/ L – 12, E – 2, M – 2. Bili – 1.9, SGOT 48 , SGPT 42 , ALP – 77 , Urea – 22 , Creatinine – 0.8 , Na – 140, K – 3.8, d-dimer – 2.44. RBS – 109, HIV /HBSAG / HCV - Negative. On Day 4 – Hb 11.0, WBC – 8530, Platelet – 172000, SGOT– 40, SGPT – 38, Creatinine – 0.9. TRUE-NAT PCR for H1N1, H3N2 were negative.

On X-ray chest (Table 2): Bilateral infiltrates seen. Findings in CT pulmonary angiography with HRCT chest did not reveal e/o pulmonary thromboembolism but bilateral infiltrates suggested ARDS.

Course of treatment: He was kept on non-invasive ventilator support initially and gradually shifted to HFNC as breathlessness worsened over time. He was treated symptomatically for bilateral pneumonitis along with IV antibiotics and HFNC support.

Inj. Aviptadil infusion at the dose of 0.166 mcg/kg/hr. for 10 hrs. was given simultaneously with prone positioning. Increasing dose of Aviptadil (0.332 and 0.498 mcg/kg/hr.) were given for next 3 days with prone positioning.

Additionally he was given chest physiotherapy with frequencer and spirometry with due course of hospitalisation. He gradually weaned from HFNC to O2 mask and then to nasal prongs over the next 4 days and shifted to the ward. He came off O2 on 6th day, on day 7 he was maintaining SPO2 of 97% without O2 support.

The patient improved clinically within a period of next 5 days with reduced oxygen requirements and ventilator support.

PA View Suggestive of Bilateral Scattered Infiltrates Suggestive of Atypical Pneumonia, and fracture long bone

Clinical Case 3

A 40 year old female patient with no previous comorbid condition or chronic illness came to the emergency department with c/o cough, cold and fever of one week duration. Also had breathlessness and restlessness with occasional blood-stained cough with sputum for 3 days

At presentation her HR was 102 bpm, SPO2 78 % on room air, BP was 100/60 mmhg, RR was 26 breaths per minute with mildly tachypnea present, was febrile RS -bilateral crepitation present CVS showing no murmur, HS were normal. CNS: Patient conscious oriented and Abdomen was soft, nontender. Patient denies h/o travel. Her bladder bowel habits were normal Table 3.

ON Investigations – Day 1 – hb 12.8, WBC 3160, Platelet 109000, n–77, L-18, M–3, E–1, T bilirubin–0.7, SGOT–1100,

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SGPT–374, ALP–254, Urea–42, Creatinine–0.9, Na–141, K–4.74, Cl-109, H1N1 / H3N2 not detected, Scrub PCR–not detected, Dengue NS1/IgM/IgG–Note detected. ABGA – pH 7.4, pCO2 – 34, pO2 45, lac 1.3, SO2b 84, HCO3 25. HIV/ HBsAg/HCV–negative.

DAY 4 – Hb 12.2, WBC – 10350, Platelet – 293000, N- 88 L 7.7 M 5.3 E 0.1, Urea 55, Creat – 0.8, Na 136, K 3.9, Cl 104. ABGA – ph 7.5, pCO2 35, pO2 60, Lac 0.7, SO2 92, HCO3 30.

She was admitted in the ICU and put on HFNC support on day 1 and shifted to NIV support on day 2 as breathlessness increased. 2d ECHO done showed a normal ejection fraction of 60%.

Over three successive consecutive days Inj. AVIPTADIL infusion was given at escalating doses of 0.166, 0.332, and 0.498 mcg/kg/hours for 10 hours each.

Patients respiratory parameters improved after receiving aviptadil treatment FIO2 requirement was reduced gradually, the patient was weaning from O2 therapy over the next 3 days.

PA View Suggestive of Bilateral Scattered Infiltrates Suggestive of Atypical Pneumonia

Clinical Case 4

A 29-year-old female patient admitted to casualty with c/o cough with expectoration, fever and generalised weakness since 5 days and she was a known case of Diabetes Type 1 (IDDM).

On admission her HR: 102 bpm and her Temp was 102 degree F. BP 122/82 mmHg, BSL 378, SPO2 was 92% on room air, ECG was showing sinus tachycardia, RR was 22 breaths per min, she was put on NASAL O2 supplement on admission. Her throat examination showed congestion and granular pharyngitis, her TRUE NAT test of the throat swab revealed H3N2 influenza detected positive H1NI detected negative and RS AEEBS bilateral crepitation. Her BAL did not reveal any organism.

On Investigation – Day 1 – Hb 11.8, WBC 3.6, Platelet 129000, N- 66 L 20 M 13 , E 00, Creat 0.5, Urea 20, Total Bili 1.2, SGOT – 22, SGPT 15, Na 140, K 4.3, PCT – 0.30, H1N1 – Negative H3N2 - Detected. ABGA – pH 7.5, pCO2 – 37, pO2 45, Lac 0.8, SO2 – 90, HCO3 28. CRP – 149 HIV/ HBsAg/ HCV – negative

On Day 4 – Hb – 13.2, WBC – 6000, Platelet 162000, N – 83, L – 18, E – 0, M – 4, Procalcitonin – 0.15.

CXR (Table 4) showed early ARDS, HRCT bilateral infiltrates + s/o ARDS. BSL was managed with Inj Insulin. Her work of breathing was increased on day 3 she was shifted to ICU and put on HFNC support (30/60). Gradually the patient was weaned off to on O2 mask on day 5 and shifted to ward on day 6 on O2 support by nasal prongs.

Over three successive consecutive days Inj. AVIPTADIL infusion was given at escalating doses of 0.166, 0.332, and 0.498 mcg/kg/hours for 10 hours each. Patient showed significant clinical improvement in her respiratory status after receiving this therapy.

PA View Suggestive of Bilateral Scattered Infiltrates Suggestive of Atypical Pneumonia

Table 2.X-ray chest and HRCT CASE -2

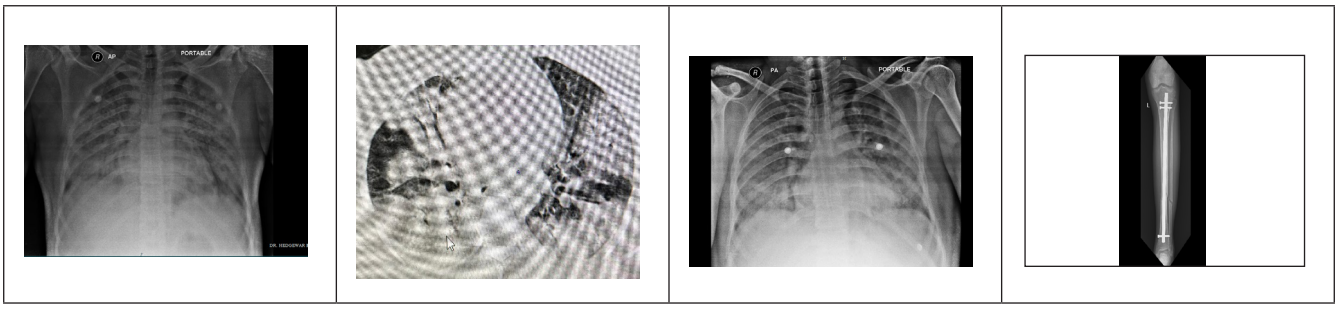

Table 3.CHEST –X ray and HRCT CASE -3

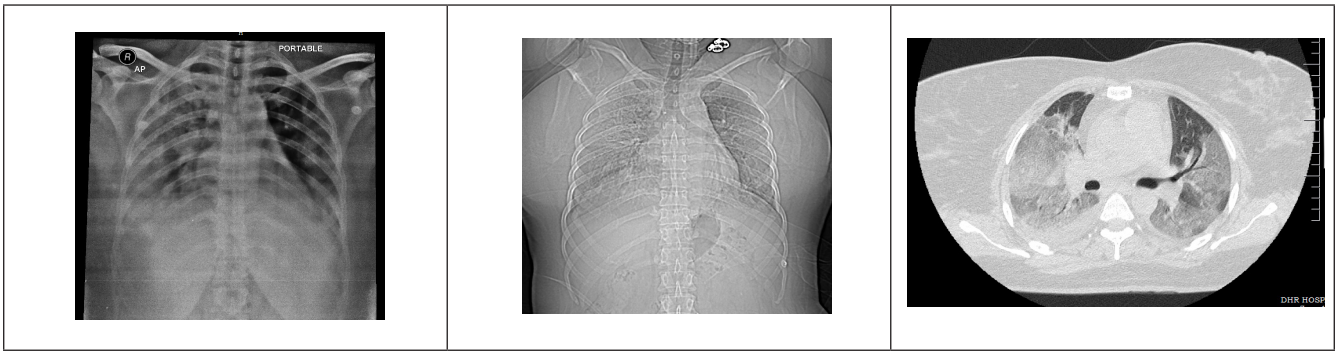

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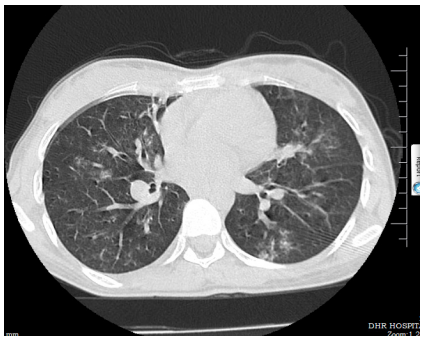
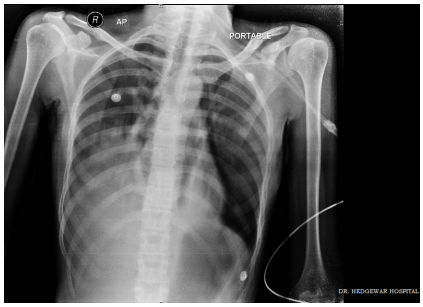
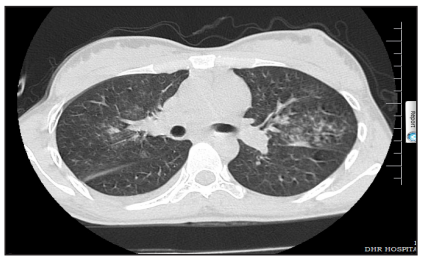
Table 4.CHEST –X ray and HRCT CASE – 4

Discussion

Acute Respiratory Distress Syndrome (ARDS) is a severe inflammatory condition that can rapidly progress to acute respiratory failure. According to the Berlin criteria, ARDS is characterised by the following:[4]

1. A known insult associated with hypoxia.
2. Bilateral radiographic pulmonary infiltrates.
3. A PaO2/FiO2 ratio of less than 300 mmHg on positive end-expiratory pressure (PEEP) > 5 cm H2O.[4]

In all four of our patients, ARDS was managed in accordance with established standard-of-care protocols, including lung protective ventilation with NIV ventilation, high-flow nasal cannula (HFNC) support and prone positioning. Despite the varied etiologies associated with ARDS, such as infectious pneumonia, sepsis and trauma, the use of AVIPTADIL (Vasoactive Intestinal Peptide, VIP) demonstrated significant clinical benefits.

Dose protocol as below, over three consecutive days Inj. AVIPTADIL infusion was given at escalating doses of 0.166, 0.332, and 0.498 mcg/kg/hour for 10 hours each. No adverse effect was observed with Aviptadil in any of the patients.[1,3,4]

Typically, during the first week of aviptadil treatment, both radiological and clinical improvements were observed, facilitating timely weaning and preventing complications associated with prolonged ventilator support too.[3]

Aviptadil plays a protective role in the lungs affected by ARDS. VIP is naturally present in the lungs and has critical effects on the respiratory and immune systems[1]. It relaxes pulmonary vasculature and bronchial smooth muscles, inhibits cellular proliferation, induces bronco-dilatation, and prevents apoptosis.

VIP consists of 28 amino acids, which were first discovered in 1970[1]. Although initially identified in the intestinal tract, human VIP is now known to be produced throughout the body. It is highly localized in the lungs (70%) and binds with ATII cells via VIP receptor type-1 (VPAC1)[1]

Furthermore, VIP reduces the severity of the cytokine storm, a major contributor to ARDS, by binding to VPAC1 receptors on alveolar type II cells[1]. These cells are responsible for producing surfactant, which maintains alveolar stability and prevents collapse, thereby promoting efficient oxygen transfer.

The pathogenesis of ARDS involves significant damage to alveolar epithelial cells, often triggered by microorganisms. This leads to a loss of epithelial integrity and impaired surfactant production, ultimately resulting in compromised gas exchange and alveolar collapse (atelectasis). The destruction of the epithelial layer exposes the endothelium to cytokines and various antigens, further amplifying the inflammatory response. The subsequent release of pro-inflammatory cytokines exacerbates lung injury, contributing to the progression of ARDS.

Aviptadil treatment is associated with a reduction in interleukin-6 (IL-6) levels, a key marker of the cytokine storm that drives ARDS progression[2]. By mitigating inflammation, improving oxygenation, and preserving alveolar function, aviptadil has shown promising results in ARDS management. This cascading protective effect throughout the lung parenchyma helps to prevent diffuse alveolar damage and enhances overall pulmonary recovery.

Aviptadil serves as a valuable adjunct therapy in ARDS treatment. Its ability to modulate inflammation, support alveolar function, and improve oxygenation makes it a promising option for mitigating lung injury and facilitating patient recovery.

Following flow (Fig2 & Fig 3) charts explains the mechanisms of aviptadil use and its benefits in simplified way[3].

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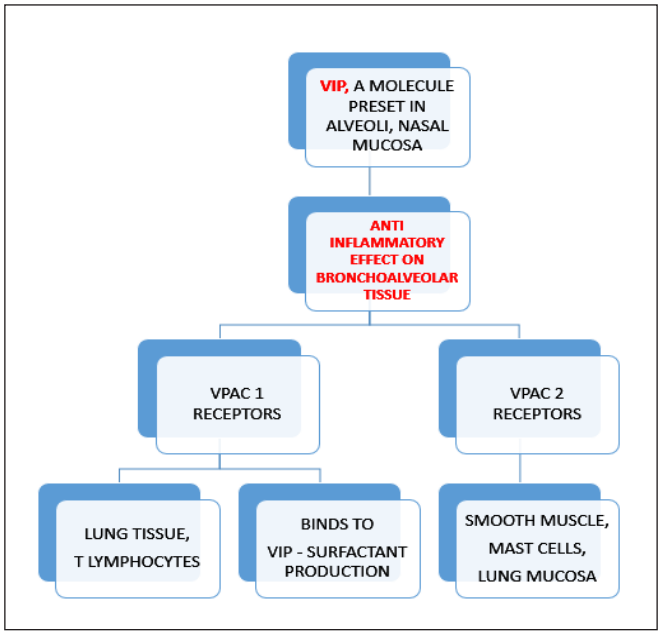

Figure 2.Role of Vasoactive Intestinal Peptide (VIP) in Bronchoalveolar Inflammation Modulation

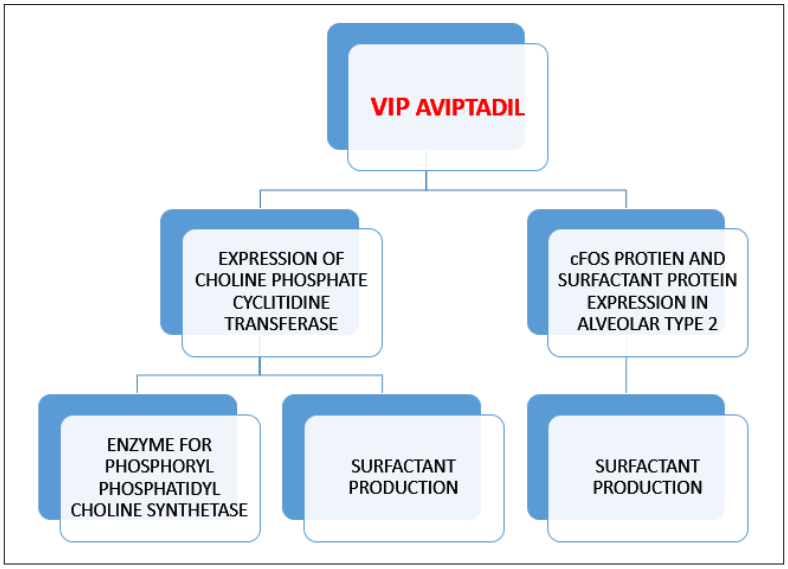

Figure 3.Mechanism of Action of VIP Aviptadil in Enhancing Surfactant Production

Conclusion

Inj. AVIPTIDIL treatment is associated with clinically meaningful improved survival from respiratory failure in ARDS associated with viral illnesses and other etiologies.

The evidence of improved lung oxygenation and reduced inflammation in first week of therapy suggest that biological effect of AVIPTADIL was seen significant in respiratory failure.

Continued research and well-designed randomized controlled trials will help to define its specific role and contribution in enhancing overall treatment strategies for ARDS patients.

Conflict of Interest: None

Source of Finding: None

Author Contribution: SG: Designed the study, and was involved in the design and supervision of the case series, collected clinical data, and contributed to manuscript drafting and final approval. SD: Managed patient care, contributed to data collection and analysis, KR: Participated in treatment planning, critically reviewed and revised the manuscript for intellectual content. VP: Helped in preparing clinical records, performed follow-up of cases, and supported the manuscript editing process.

All authors: Have read and approved the final manuscript and agree to be accountable for all aspects of the work.

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Declaration of Generative AI and AI-Assisted Technologies in the Writing Process: None

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