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Postobstructive Pulmonary Edema:

A Case for Hydrostatic Mechanisms

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Abstract

Background—Postobstructive pulmonary edema is a well-recognized complication of upper airway obstruction. The mechanisms of edema formation are unclear and may be due to increased hydrostatic forces generated by high negative inspiratory pressure or by increased permeability of the alveolar capillary membrane. Measurement of the edema fluid/plasma protein ratio and the rate of net alveolar fluid clearance are two well-validated methods for classifying the underlying mechanism of edema formation. The goal of the current study was to investigate the mechanisms of pulmonary edema formation in patients with postobstructive pulmonary edema by serial sampling of undiluted pulmonary edema fluid.

Methods—A retrospective review of 341 patients who had pulmonary edema fluid collected prospectively after the acute onset of pulmonary edema. All patients had serial samples of edema fluid and plasma collected over the first 8 h after intubation.

Results—Ten of the 341 patients with acute pulmonary edema were identified as having postobstructive pulmonary edema. The mean ($\pm$ SD) edema fluid/plasma protein ratio in these patients was 0.54 ± 0.15 . The mean rate of alveolar fluid clearance over 8 h was $14.0 \pm 17.4\%$ per hour. Nine of the 10 patients survived the hospitalization.

Conclusion—Measurement of the edema fluid/plasma protein ratio and the presence of net alveolar fluid clearance in 10 patients with postobstructive pulmonary edema supports a hydrostatic mechanism for edema fluid formation. The predominantly fast rates of alveolar fluid clearance may explain the rapid resolution of clinical postobstructive pulmonary edema that is typically described.

Keywords

alveolar fluid clearance; edema; fluid/plasma protein; hydrostatic pulmonary edema; postobstructive pulmonary edema

Postobstructive pulmonary edema is an uncommon, but well-described, complication of upper airway obstruction. Two different mechanisms have been proposed for the development of pulmonary edema in the setting of upper airway obstruction. One theory is that postobstructive

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pulmonary edema is caused by significant fluid shifts due to changes in intrathoracic pressure.¹ Negative intrathoracic pressure is generated in the chest when a patient attempts to inspire against a closed glottis or obstructed airway. The short-term drop in intrathoracic pressure increases venous return to the right side of the heart, which in turn increases pulmonary venous pressure. This increase in pressure in the venous circulation creates a hydrostatic transpulmonary gradient with fluid moving from high pressure (pulmonary venous system) to low pressure (pulmonary interstitium and airspaces).¹⁻⁵ The second proposed mechanism involves the disruption of the alveolar epithelial and pulmonary microvascular membranes from severe mechanical stress, leading to increased pulmonary capillary permeability and protein-rich pulmonary edema.¹⁻⁵ The first proposed mechanism of edema formation is similar to hydrostatic pulmonary edema, as seen in patients with congestive heart failure or volume overload states. The latter mechanism is similar to increased permeability pulmonary edema, as seen in patients with acute lung injury or the ARDS.⁶

Measurement of the pulmonary edema fluid/plasma protein ratio is a well-validated method to differentiate between hydrostatic pulmonary edema and increased permeability pulmonary edema.⁷⁻¹⁰ Measurement of the rate of net alveolar fluid clearance, with sequential samples of pulmonary edema fluid, provides an assessment of the capacity of the alveolar epithelial barrier to remove alveolar edema fluid, a property that is usually impaired in patients with acute lung injury yet intact in patients with hydrostatic pulmonary edema.¹⁰⁻¹³

We have previously reported four cases of postobstructive pulmonary edema with a mean pulmonary edema fluid/plasma protein ratio of 0.43, which is consistent with a hydrostatic mechanism.^{5,14} We now report an expanded series of 10 patients with postobstructive pulmonary edema in whom we measured both the edema fluid/plasma protein ratio and the net rate of alveolar fluid clearance. We postulated that postobstructive pulmonary edema would have characteristics similar to hydrostatic pulmonary edema, with both a low edema fluid/plasma protein ratio and rapid rates of net alveolar fluid clearance, based on a similar physiologic mechanism.

Materials and Methods

The Committee on Human Research at the University of California San Francisco approved this study. We conducted a retrospective review of 341 intubated patients, who were enrolled in a pulmonary edema fluid and plasma databank at the University of California San Francisco over the 20-year period from 1982 to 2002, to identify all cases of suspected acute postobstructive pulmonary edema. All patients had the acute onset of bilateral infiltrates on chest radiograph. The classification of the etiology of acute pulmonary edema as postobstructive was based on a chart review of clinical characteristics at the time of intubation, as well as the hospital course. Patients were selected if “postobstructive pulmonary edema” was documented as a diagnosis and if the study investigators agreed with that diagnosis after reviewing the medical record. Study investigators were unaware of the edema fluid and plasma protein measurements at the time of the chart review. All patients who had a presentation that was consistent with postobstructive pulmonary edema and met the above criteria were included in the present study. Pulmonary edema fluid was obtained by trained respiratory therapists and physicians, using a previously described method.^{9,11} Briefly, a soft 14F suction catheter was advanced to a wedged position in a distal bronchus through the endotracheal tube.^{9,11} Gentle suction was applied, and samples were collected via a suction trap. Simultaneous plasma samples were collected by venipuncture or aspiration through an already placed IV access site.^{9,11} All samples were collected and processed prospectively. Serial samples were obtained from patients over the next 8 h to evaluate the rate of alveolar fluid clearance. Pulmonary edema fluid and plasma fluid samples were centrifuged, and the supernatant was aspirated and stored at -70°C.

The total protein levels in edema fluid and plasma were measured by the biuret method, which has previously been described.^{9,11} An edema fluid/plasma protein ratio of < 0.65 is consistent with hydrostatic pulmonary edema, while a ratio of > 0.75 is consistent with high-permeability pulmonary edema.^{7-9,11} Patients with a ratio between 0.65 and 0.75 may have a mixed etiology of pulmonary edema or they may have begun to reabsorb some of the edema fluid. This would result in a higher concentration of protein in the edema fluid prior to the initial sampling.¹⁵ The calculation of the rate of alveolar fluid clearance was performed as a percentage of alveolar fluid volume reabsorbed per hour, as has been validated in both clinical and experimental studies.^{9,11-13} Briefly, since the removal of protein from the alveoli is slow compared to the removal of fluid, the percentage of alveolar edema fluid reabsorbed can be estimated with the following equation:

$$\text{percentage of alveolar fluid clearance} = 100 \times [1 - (\text{initial edema protein} / \text{final edema protein})].$$

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Alveolar fluid clearance $\geq 3\%$ per hour is defined as intact alveolar clearance, while clearance of $> 14\%$ per hour is defined as maximal alveolar fluid clearance.^{11,12} All statistical analysis was performed using a statistical software program (SPSS, version 14.0 for Windows; SPSS, Inc; Chicago, IL). The results are reported as either the means $\pm$ SD or median and interquartile range, as appropriate. The Spearman correlation coefficient was employed to determine the strength of correlation between variables.

Results

Ten of the 341 patients (3%) were identified over the 20-year period as having postobstructive pulmonary edema. Eight of the ten patients were men. The causes of postobstructive pulmonary edema included suspected postoperative laryngospasm ($n = 8$), foreign-body aspiration ($n = 1$), and severe patient-ventilator asynchrony with inspiratory attempts during closure of the ventilator inspiratory valve ($n = 1$). The mean age of the patients was 37 ± 24 years. The mean simplified acute physiology II score¹⁶ was 25 ± 13 . All 10 patients required endotracheal intubation and mechanical ventilation due to hypoxemia. The mean duration of mechanical ventilation was 3.7 ± 4.5 days. Nine of 10 patients survived their hospital course.

The initial edema fluid and plasma protein measurements are summarized in Table 1. The mean volume of the initial edema fluid sample was 1.7 ± 1.2 mL. The mean edema fluid/plasma protein ratio was 0.54 ± 0.15 . Of these 10 patients, 7 patients had an initial ratio of ≤ 0.65 , providing unequivocal evidence for a hydrostatic etiology. Two patients had levels that were slightly above this cutoff point at 0.66 and 0.69, still suggesting a predominant hydrostatic mechanism. One patient had an initial ratio of 0.80, suggesting either a nonhydrostatic mechanism or that the sampling of the edema fluid took place after reabsorption had begun.

The net rate of alveolar fluid clearance was also measured in the 10 patients. Alveolar fluid clearance was measurable in all patients, although there was considerable variability in the individual rates of alveolar fluid clearance. The mean rate of net alveolar fluid clearance over the first 8 h after endotracheal intubation was rapid ($14.0 \pm 17.4\%$ per hour). The median time to collection of the first sample of edema fluid after endotracheal intubation was 1.5 h (interquartile range, 0.5 to 5 h). There was no correlation between time to collection of the first sample of edema fluid and the initial edema fluid/plasma protein ratio ($r = 0.13$; $p = 0.71$) or the rate of alveolar fluid clearance ($r = -0.02$; $p = 0.95$).

Discussion

In this study, we report the pulmonary edema fluid/plasma protein ratio in 10 patients in whom acute postobstructive pulmonary edema developed. The mean edema fluid/plasma protein ratio was 0.54, which is consistent with hydrostatic causes of pulmonary edema. To our knowledge, only three prior studies^{5,14,17} have reported the edema fluid/plasma protein ratio in postobstructive pulmonary edema. Two of the reports,^{5,14} also from our group, reported measurements in four patients in whom postobstructive pulmonary edema developed. These patients had edema fluid/plasma protein ratios within the hydrostatic range (mean ratio, 0.43) and were included in the current report.^{5,14} The other report¹⁷ had only one patient, who had a complicated hospital course, including prolonged resuscitation, cerebral infarction, and the development of ARDS. In that study, the edema fluid/plasma protein ratio was 0.83, suggesting the possibility of increased permeability pulmonary edema.¹⁷ However, the measurement may have been made some time after intubation; in the presence of intact alveolar fluid clearance mechanisms, protein concentrations in the pulmonary edema fluid will rise over time and may reach levels higher than those in plasma.⁹ Thus, for the classification of the etiology of pulmonary edema, it is critical to collect pulmonary edema fluid as soon as possible after endotracheal intubation. In our current study, the median time to collection of fluid after intubation was 1.5 h. The one patient in the current study (patient 10) who had an edema fluid/plasma protein ratio of > 0.75 had an unremarkable hospital course. He was extubated the same day that pulmonary edema developed and was subsequently discharged from the hospital without any prolonged pulmonary sequelae. His unremarkable hospital course makes a diagnosis of significant acute lung injury unlikely; therefore, his high edema fluid/plasma protein ratio was probably due to the absorption of lung water and the subsequent concentration of edema fluid protein prior to sampling.

To our knowledge, this is the first study to report the rate of net alveolar fluid clearance in patients with postobstructive pulmonary edema. Although the individual rates of alveolar fluid clearance were quite variable, all patients had evidence of some net alveolar fluid clearance. The mean rate of net alveolar fluid clearance was within the range of maximal clearance ($> 14\%$ per hour), as previously defined in animal and clinical studies.^{11,12} Alveolar fluid clearance is usually maintained in patients with hydrostatic pulmonary edema since the alveolar/capillary barrier is uninjured and intact. In the largest study¹¹ to report rates of alveolar fluid clearance in patients with severe hydrostatic pulmonary edema, 75% of patients had intact net alveolar fluid clearance with a mean rate of 13% per hour. By contrast, in a study¹⁸ of 79 patients with acute lung injury and ARDS, net alveolar fluid clearance was intact in only 44% of patients, with a mean rate of 6% per hour. Thus, the current findings in postobstructive pulmonary edema (intact net alveolar fluid clearance, 70% of patients; mean clearance rate, 14.0% per hour) are consistent with intact alveolar epithelial fluid transport function and an absence of significant alveolar epithelial injury. This finding provides further evidence that postobstructive pulmonary edema is a form of hydrostatic pulmonary edema since the alveolar fluid clearance mechanisms remained functional and intact.

Hydrostatic mechanisms of postobstructive pulmonary edema are thought to arise from the large negative intrathoracic pressures generated by inspiring against a closed or obstructed airway. Loyd et al¹⁹ reported that the greater the negative pressure applied to the intrathoracic cavity of awake sheep, the greater the increase in extravascular lung water, suggesting that the amount of negative intrathoracic pressure was the primary determinant of the degree of resultant pulmonary edema. Healthy human subjects can generate very high levels of negative inspiratory pressure with a reported maximum of -140 cm H₂O.²⁰ This negative intrathoracic pressure increases the return of blood to the right side of the heart and concomitantly increases pulmonary venous pressures. The negative intrathoracic pressure, along with the resultant hypoxia, also has a depressing effect on cardiac output by increasing myocardial wall stress at

end-systole as well as increasing systemic vascular resistance.⁵ The fall in cardiac output elevates in succession end-diastolic pressure, left atrial pressure, pulmonary venous pressure, and pulmonary microvascular pressure, further increasing the hydrostatic forces that favor the formation of pulmonary edema.^{5,21} Thus, the combination of increased preload and increased afterload creates a marked increase in hydrostatic pressure in the pulmonary microvasculature, and, as dictated by the Starling equation, fluid filters out of the microcirculation into the lung interstitium. When a critical quantity of edema fluid collects in the interstitial compartment, alveolar flooding occurs.^{22,23}

The most common cause of postobstructive pulmonary edema is laryngospasm during intubation or after anesthesia in the postoperative period.²⁴ Laryngospasm has been reported to be the cause in > 50% of cases of postobstructive pulmonary edema.²⁴ This finding is consistent with our study in which acute postobstructive pulmonary edema developed in 8 of 10 patients in the postoperative period.²⁴ Other reported causes of postobstructive pulmonary edema include the following: strangulation²; epiglottitis²⁵; foreign-body aspiration²⁶; hypothyroidism²⁷; inspissated tracheal secretions²⁴; hiccups²⁸; croup²⁹; thyroid goiter³⁰; temporomandibular joint arthroscopy³¹; difficult intubation³²; hematoma²⁴; upper airway tumor¹; oropharyngeal surgery³³; Ludwig angina³⁴; obesity²⁴; acromegaly³⁰; obstructive sleep apnea²⁶; mediastinal tumor²⁴; and biting the endotracheal tube or laryngeal mask.^{2,35} Patients in whom postobstructive pulmonary edema develops generally have an uncomplicated hospital course followed by the rapid resolution of the pulmonary edema and short hospital stays.^{33, 36} The patients in our study had similarly uncomplicated courses with an average ICU length of stay of < 4 days, a relatively low severity of illness as measured by the simplified acute physiology II score, and only a 10% mortality rate. The one patient who died experienced anoxic brain injury during airway obstruction, prompting the family to withdraw care.

The primary limitation of our report is that this is a retrospective analysis of data and we relied on the medical record to appropriately classify patients as having a postobstructive etiology of acute pulmonary edema. However, the data in support of this conclusion were convincing in each case. An additional concern is that protein production by airway epithelial cells and possible sampling admixture of bronchial secretions could confound the protein measurements. However, when quantified, airway secretions have a low protein concentration (approximately 0.5 g/dL) and thus tend to dilute rather than increase the total protein concentration.^{18,37-39} Furthermore, the methods used to sample pulmonary edema fluid, and to measure the edema fluid/plasma protein ratio and the rate of alveolar fluid clearance in this study have been used in multiple prior clinical studies from our group.^{10,11,18,40} In these prior clinical studies, there have been correlations between the rate of alveolar fluid clearance and meaningful clinical indexes of improvement in patients with pulmonary edema such as improvement in chest radiograph findings and oxygenation. Thus, we believe that our method correctly estimates the rate of net alveolar fluid clearance in the majority of patients. The technique of edema fluid sampling has also been verified in dogs using alveolar micropuncture.⁴¹

Conclusions

In summary, both the edema fluid/plasma protein ratio and the rate of net alveolar fluid clearance in patients with acute postobstructive pulmonary edema were in the range previously reported for patients with acute hydrostatic pulmonary edema. These findings provide further support for the hypothesis that hydrostatic forces are the primary mechanism behind postobstructive pulmonary edema and that the alveolar epithelium remains functionally intact in acute postobstructive pulmonary edema.

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Table 1
Laboratory Measurements in 10 Patients With Postobstructive Pulmonary Edema

Patient	Time From Intubation to Sample Collection, h	Edema Fluid Protein, g/dL	Plasma Protein, g/dL	Edema Fluid/Plasma Protein Ratio	Alveolar Fluid Clearance, % clearance/h
1 [*]	0	1.17	3.87	0.30	7.3
2	0.25	2.38	5.53	0.43	57.1
3 [*]	10.25	3.0	6.88	0.44	2.7
4	3.25	1.53	3.43	0.45	21.0
5 [†]	23.5	2.06	4.41	0.47	27.3
6 [*]	2.25	3.18	6.14	0.52	7.0
7	0.75	3.62	5.71	0.63	1.6
8	0.67	4.62	6.06	0.66	9.3
9	2.25	2.72	3.94	0.69	4.3
10	1.0	4.72	5.89	0.80	2.4

^{*} Previously reported patients.⁵

[†] Previously reported patient.¹⁴