ARDS: Case Study of a Mystery Killer

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Adult Respiratory Distress Syndrome (ARDS) is a dangerous lung condition brought on by various diseases and injuries. It is an acute failure of the respiratory system distinguished by fluid buildup inside the lungs, causing stiffening and decreased lung capacity. Symptoms include damage to endothelium and lining, low O_2 concentration in blood despite heavy oxygen supplements given to the patient, inflammation of lung cells, and loss of consciousness.[1]

ARDS kills more than 60% of its victims. Over 100,000 people in the United States die from ARDS each year -- a higher death rate than AIDS.[2] In the 25 years since it was first described, few advances have been made to reduce the death rate of ARDS. To devise effective treatments, we need a better understanding of the physiological process responsible for the syndrome.

Ware and Matthay described in their recent paper[3] some of the advancements in the understanding of ARDS, but cited little progress in its specific treatment. Perhaps we lack successful treatment because we have not recognized that the pleural effusion in ARDS involves neuroinflammation and gap formation.

I had the unfortunate opportunity for a case study on this aspect of ARDS when it struck my wife (68 and previously healthy).

She was admitted to ICU on December 28, 1999 with acute respiratory failure secondary to viral pneumonia. Her condition deteriorated rapidly and she required intubation and mechanical ventilation: she was unconscious for 30 days.

Her x-ray reports document her progress.

12/28/99…moderate right upper lobe collapse and consolidation...air space disease involving the left lower lobe…ill-defined and nodular opacities in the left mid lung…heart not enlarged…no evidence of mediastinal or hilar adenopathy.

12/29/99 - Lung volumes have diminished…worsening and more confluent opacification of the left midlung. In addition, new airspace disease in the right middle lobe. The previously described regions of collapse and consolidation in the right upper and left lower lobes have become denser as a result of diminished lung volumes. Overall worsening.

12/29/99 - Patient has been intubated …There is worsening air space disease involving both lungs, which appears worse even though the lung volumes have improved as the result of the intubation. Although distribution would seem in keeping with an infectious etiology, the rapidity of the changes raises the possibility of superimposed non-cardiogenic pulmonary edema.

She required tracheostomy and pure oxygen was administered at maximum pressure. She developed atrial fibrillation which required cardioversion and Type II diabetes. When X-rays on 1/2/00 showed no improvement, the attending physician sadly informed our family that her condition was irreversible; she was kept alive only by ICU support.

I asked permission to examine her and recognized her physical signs to be those of radiculopathy involving several segments.[4] For example: atrophy of the small muscles in her hands (C7-8); severe atrophy of the gluteal muscles and decubitus ulcers in the buttocks and left heel (L5, S1-2); trophedema in the subcutaneous tissues of her dorsal and lumbar backs.

Lungs are innervated by nerves from lower cervical and upper dorsal segments.

The pulmonary edema was likely part of the neuropathic disorder. In desperation, I called upon low level laser therapy, a treatment that is used by physical therapists to soothe inflammation and dissipate edema in soft tissue injury. I directed the laser beam (820nm and 50 mw) between her ribs, into the lungs, and also into spinal muscles overlying affected nerve roots.

X-rays taken 6 hours later showed a change for the better:

1/3/00 - The consolidative changes in the left upper lobe are slightly reduced. The right upper lobe appears slightly better aerated. The right lower lobe also shows some decrease in the amount of consolidation. Impression: The patient with known diffuse bilateral pneumonia shows slight improvement in the left upper lobe and right lower lobe.

1/7/00 - Both lungs are well ventilated. There is widespread interstitial disease in both lungs. In the left base it has taken a honeycomb pattern. There is no lung contracture.

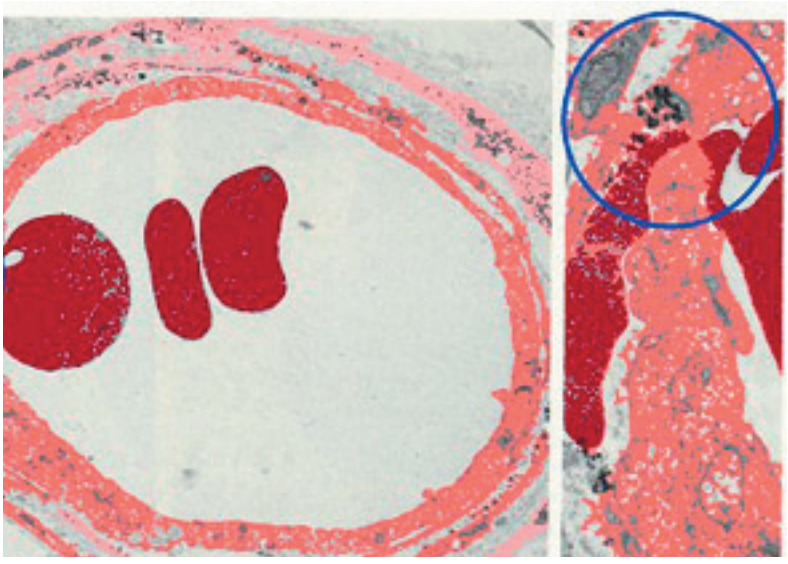
1/15/00 - Both lungs are well ventilated. The interstitial thickening is now localized to the periphery of the right upper zone, right lower zone and left lower zone.

1/20/00 - Patchy interstitial thickening in the periphery of both lungs. No consolidation or pulmonary edema.

From commencement of daily laser therapy, it had taken 17 days to clear the lungs.

John Hunter in 1794 had recognized that inflammation is a more or less stereotyped response to injury. Therefore similarities can be expected between inflammation in soft tissue and in lung and review of one may provide clues to the nature of the other.

Inflammation in soft tissue causes the permeability of small blood vessels in and around the injured area to increase. This allows large amounts of fluid and plasma protein to escape into the extravascular space to form inflammatory exudate. Inflammatory exudate is not a static puddle but a high-turnover pool with a massive amount of protein passing from blood to lymph.[5] While small molecules rapidly diffuse across the vascular wall, plasma protein escapes through transient gaps that appear in the junctions between adjacent endothelial cells. Gap formation, which is initiated by histamine and like substances, is believed to be caused by activation of a contractile protein, similar to actomyosin, within the cytoplasm of endothelial cells. Gap formation is a reversible process: when leakage ceases, the cells come together again without any evidence of damage to endohelial cells.[6] Since inflammation in the lung is likewise associated with loss of epithelial integrity and influx of protein-rich edema fluid, gap formation likely also occurs in the lung.



Soft tissue inflammation generally follows injury, but it can (like soft tissue pain) also result from peripheral neuropathy. Radiculopathy is not uncommon in the very young and old, the two age groups that are at risk. Is lung inflammation, generally associated with injury, a neuropathic manifestation? These are questions that may be answered by searching for signs of peripheral neuropathy in patients with respiratory distress. But, whether or not the condition is related to neuropathy, low level laser therapy, a simple modality with few undesirable side-effects should be tried. The decubitus ulcers, well known for their reluctance to heal, were cleared in days. Myotomal muscle wasting was electrically stimulated with a transcutaneous electrical stimulator [TENS]. Treatment should preferably be early in the course of the disease.

References

Greene A et al. ARDS (adult respiratory distress syndrome). Health Illustrated Encyclopedia. A.D.A.M. 2000.

Repine J. Adult Respiratory Distress Syndrome (ARDS): The mystery killer. Webb-Waring Institute. 2000 http://www.uchsc.edu/sm/waring/webpages/secondlevel/science/ards.html

Ware LB, Matthay MA. The acute respiratory distress syndrome. N Engl J Med 2000; 342 18 1334-49.

Gunn CC. Early and subtle signs in low-back sprain. Spine 1978, 3 3 100- 114.

Pontinen PJ. Low Laser Therapy As A Medical Treatment Modality. Art Upo, Tampere 1992.

Hurley JV. In EDEMA, Editors, Staub and Taylor, Raven Press, New York, 1984; 463-488.