Lung Impedance-guided preemptive treatment of evolving pulmonary congestion or edema in the course of acute myocardial infarction reduces the use of furosemide.

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Conflict of interest: Member Board of Directors
RSMM, manufacturer of the impedance device
The Problem

During admission for Acute Myocardial Infarction (AMI) nearly 35-40% of patients develop Killip II-IV class pulmonary congestion or pulmonary edema.\(^1,2\)

Today we cannot detect or predict evolving pulmonary edema at the asymptomatic stage because clinical signs of edema are absent. Therapy for pulmonary edema is initiated only after the appearance of clinical signs of pulmonary edema.\(^2\)

There are two main approaches to monitoring patients possess risk of pulmonary edema development:

1. Monitoring of changes in pressure (Intracardiac Pressure, PCWP or Pulmonary Artery Pressure).
2. Monitoring changes in Lung Fluid Content.

Pressure monitoring in course of AMI is invasive, difficult and disappointing. The use of Pulmonary Artery Catheterization has decreased in US by 81%.\(^3\)

Monitoring a Lung Fluid Content. Evolution of Pulmonary Edema (PED)

Dry lungs - Insignificant amount of fluid in lungs

- High impedance

Congested lungs – Increased amount of fluid accumulation in lungs

- Low impedance

LI

LI

1. Redistribution
2. Bronchial Cuffing
3. Kerley A, B, C lines
4. Increased Heart Size

Normal Lung
Interstitial Edema
Alveolar Edema (PED)

Asymptomatic stage
Symptomatic stage

High impedance
Low impedance

Evolution of Pulmonary Edema
Monitoring changes in Lung Fluid Content - State of the art

1. Invasive Impedance devices (as Opti Vol pacemaker based devices). Are not applicable in course of AMI. For CHF have low (38%) positive predictive value for pulmonary Edema hospitalizations.

2. Non-invasive (based on traditional “Kubichek's” theorem as “BioZ”) device. The problem is that device measures a “full thoracic impedance” (picture 1). A Lung Impedance is a small part of “full thoracic impedance”. The result is a low sensitivity measurements of small changes in Lung Impedance at early preclinical stage of evolving pulmonary edema. As the result of that devices based on “Kubichek's” principle didn’t find a wide use in monitoring pulmonary edema patients in course of AMI.

We used a non-invasive 6 electrodes impedance monitor based on transverse distribution of electromagnetic field through the chest (picture 2). Multiple paired measurements between electrodes Enables calculation “net” Lung Impedance. This increases a sensitivity of Lung Impedance measurement by 25 times and makes possible determination a small changes in lung fluid content at preclinical stage of pulmonary edema.

580 STEMI patients without clinical and radiological signs of pulmonary congestion or edema at admission were included.

We have shown that a Lung Impedance (LI) decrease of 12-14% from baseline (admission) level, when patients are still asymptomatic, reflects the beginning of transition from interstitial to alveolar edema.

358 patients decreased their LI ≤ 12% with no clinical and radiological signs of pulmonary edema throughout admission (control group).

222 patients decreased their LI by 12-14% and were randomized (2:1) to:

- **Group 1:** 148 patients treated according to common practice.
- **Group 2:** 74 patients treated preemptively according to LI.

Protocol of Furosemide treatment: Push IV Furosemide 40 mg with continuous IV Furosemide drip (125 mg/day) until recovery of LI to the baseline.
## Demographic, laboratory and instrumental data of two groups patients

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>P values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>61.3± 14.1</td>
<td>60.0±12.5</td>
<td>NS</td>
</tr>
<tr>
<td>Men</td>
<td>79%</td>
<td>83%</td>
<td>NS</td>
</tr>
<tr>
<td>Follow-up (months)</td>
<td>84.9±25</td>
<td>84.5±22</td>
<td>NS</td>
</tr>
<tr>
<td>Lung Impedance at onset (Ohms)</td>
<td>57.5±15.4</td>
<td>57.7±15.3</td>
<td>NS</td>
</tr>
<tr>
<td>Echocardiograms during admission</td>
<td>99.3%</td>
<td>100%</td>
<td>NS</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>45.9±12.4</td>
<td>46.9±11.8</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>34%</td>
<td>30%</td>
<td>NS</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>66%</td>
<td>66%</td>
<td>NS</td>
</tr>
<tr>
<td>Smoking</td>
<td>49%</td>
<td>58%</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>48%</td>
<td>49%</td>
<td>NS</td>
</tr>
<tr>
<td>Thrombolytic therapy</td>
<td>70(49%)</td>
<td>36(51%)</td>
<td>NS</td>
</tr>
<tr>
<td>Primary PCI</td>
<td>64(45%)</td>
<td>32(45%)</td>
<td>NS</td>
</tr>
<tr>
<td>Transient STEMI</td>
<td>8(6%)</td>
<td>3(4%)</td>
<td>NS</td>
</tr>
<tr>
<td>PCI during hospitalization (Primary PCI included)</td>
<td>128(90.1%)</td>
<td>63(88.7%)</td>
<td>NS</td>
</tr>
<tr>
<td>CABG</td>
<td>13(9.2%)</td>
<td>8(11.3%)</td>
<td>NS</td>
</tr>
<tr>
<td>Anterior STEMI</td>
<td>53%</td>
<td>49%</td>
<td>NS</td>
</tr>
<tr>
<td>Peak CK (mg/dl)</td>
<td>2078±1330</td>
<td>1920±1612</td>
<td>NS</td>
</tr>
<tr>
<td>Admission/discharge creatinine (mg/dl)</td>
<td>1.06±0.28</td>
<td>1.02±0.35</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>1.10±0.38</td>
<td>1.07±0.39</td>
<td>NS</td>
</tr>
<tr>
<td>Admission hemoglobin (gr/dl)</td>
<td>14.1</td>
<td>14.3</td>
<td>NS</td>
</tr>
</tbody>
</table>
**Time course of Pulmonary Edema evolution and therapy**

**Group 2.** LI-guided preemptive treatment was started at randomization (point B).

**Group 1.** Complains beginning on PED (point F). Treatment for PED was started.

- Time AB = 9±7.2 hours
- Time BF = 9±5.6 hours

No patients died

6 patients died

End of follow up

Mild-Moderate Pulmonary Edema (PED)

Moderate PED

Severe PED
Overall dosage of Furosemide

Results

Group 1. Treatment of evolving pulmonary edema according common practice

Group 2. Lung Impedance-guided preemptive treatment of evolving pulmonary edema.
Results

Group 1: Treatment of evolving pulmonary edema according to common practice

Group 2: Lung Impedance-guided preemptive treatment of evolving pulmonary edema.

Duration of Furosemide treatment by groups

P < 0.01)
Results

Group 1. Treatment of evolving pulmonary edema according common practice

Group 2. Lung Impedance-guided preemptive treatment of evolving pulmonary edema.

Length of hospital stay by groups

P < 0.0001
Conclusions

Lung Impedance guided preemptive treatment of evolving pulmonary congestion-edema in course of acute myocardial infarction:

1. Reduces the use of furosemide

2. Reduce the duration of furosemide treatment

3. Reduce the length of hospital stay