ACUTE KIDNEY INJURY DURING COVID-19

UPDATE FROM THE FRONT LINES: PROTESTORS, TEAR GAS & COVID-19

JUNE 17, 2020
WEBINAR SERIES PARTNERS

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<th>Organization</th>
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<tr>
<td>American Academy of Clinical Toxicology (AACT)</td>
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<td>American Academy of Emergency Medicine (AAEM)</td>
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<tr>
<td>American Academy of Emergency Nurse Practitioners (AAENP)</td>
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<tr>
<td>American Association of Poison Control Centers (AAPCC)</td>
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<td>American College of Medical Toxicology (ACMT)</td>
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<td>Asia Pacific Association of Medical Toxicologists (APAMT)</td>
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<td>European Association of Poison Centers and Clinical Toxicologists (EAPCCT)</td>
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<td>Middle East &amp; North Africa Clinical Toxicology Association (MENATOX)</td>
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</table>
ON-DEMAND RESOURCES

All webinars are recorded and posted to the ACMT website

www.acmt.net/covid19web

Questions?
Write to: info@acmt.net
Q&A
will be at end of the Webinar

Please type your questions into the Q&A or Chat function during the webinar and we will get to as many as we can

We monitor all platforms, including YouTube and Facebook, for questions
NONE OF OUR SPEAKERS HAVE ANY CONFLICTS OF INTEREST TO DISCLOSE
Paul M. Wax, MD FACMT
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- Medical Director, Maryland Poison Control
- Associate Program Director, Nephrology Fellowship Program, University of Maryland School of Medicine
ACUTE KIDNEY INJURY DURING COVID-19

MEDICAL AND PUBLIC HEALTH CONSIDERATIONS OF COVID-19

Jonathan M. Barasch, MD, PhD
Professor of Medicine and Pathology and Cell Biology
Columbia University, New York, NY
Imagine a patient presenting with fever, loss of appetite, low blood pressure (105/80) and "pneumonia vs atelectasis" is found. The patient wants antibiotics and discharge. However, the patient’s $Pcr = 1.5$ (baseline $Pcr = 1.0$), defining what is called “Acute Kidney Injury, AKI”. What should the doctor do?

A patient with CHF suffers SOB. Diuretics are initiated, but $Pcr$ rises from $Pcr=1.0$ to $Pcr=2.0$. The cardiologist says there is now “AKI”. What should the cardiologist do?

Imagine a patient presenting after a motorcycle accident with reddish-brown urine, and an elevated CPK level. You want to perform a CT Scan with contrast. $Pcr = 1.0$. What should the doctor do?
AKI and Pcr

• The ratio of Pcr and Ucr can tell us about kidney function.

  o GFR = \( \frac{\text{Ucr mg/ml} \times \text{Vml/min}}{\text{Pcr mg/ml}} \)
  
  o GFR = Organ function i.e. the excretory capacity of the kidney
  
  o *At Steady State*

  o Rate of appearance of Pcr in the glomerular filtrate (mg/min) =
  
  o Rate of appearance of Pcr in the urine (mg/min)
  
  o *At Steady State*

Creatinine
AKI and Pcr

- PCr is used to approximate acute injury (which is a non steady state event).
- In fact the AKI Diagnosis is scaled to the height of Pcr.
- AKI means an injury to kidney cells

The RIFLE Consensus

- **Risk**
  - \( \Delta S_{Cr} \times 1.5 \) or \( \downarrow \text{eGFR} \times 25\% \)
  - UO < 0.5 mL/kg/h \( \times 6 \) hr

- **Injury**
  - \( \Delta S_{Cr} \times 2 \) or \( \downarrow \text{eGFR} \times 50\% \)
  - UO < 0.5 mL/kg/h \( \times 12 \) hr

- **Failure**
  - \( \Delta S_{Cr} \times 3 \) or \( \downarrow \text{eGFR} \times 75\% \)
  - UO < 0.5 mL/kg/h \( \times 24 \) hr
  - ARF \times 4 \) wks

- **Loss**
  - ESRD \times 3 \) mos

- **ESRD**
  - ESRD \times 3 \) mos

- **RIFLE, AKIN, KDIGO.**
(1) All Kidney Diagnoses are Made in Retrospect

(2) Insensitive Test

(3) Creatinine Rises for Many Reasons

<table>
<thead>
<tr>
<th>Pre Renal Azotemia (Hemodynamic)</th>
<th>Intra-Renal Vascular Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive Heart Failure</td>
<td>MAHA</td>
</tr>
<tr>
<td>Hepatorenal Failure</td>
<td>Thrombosis</td>
</tr>
<tr>
<td>Volume Depletion</td>
<td>Associated with Glm disease</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Intra-Renal Tubular Damage</th>
<th>Post Renal Obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension</td>
<td>Stones</td>
</tr>
<tr>
<td>Enzymes</td>
<td>Tumors</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Neurogenic Bladder</td>
</tr>
<tr>
<td>Glomerulonephritis</td>
<td>Prostatic Hypertophy</td>
</tr>
<tr>
<td>Nephrotoxins</td>
<td>CAKUT</td>
</tr>
<tr>
<td>Metals</td>
<td></td>
</tr>
</tbody>
</table>

And Resolves by Day 3 (75% of Cases)

Raising the Question of Injury
Pcr: Difficult Decisions @ Patient Contact

Chirag Parikh, Johns Hopkins
NGAL Neutrophil Gelatinase Associated Lipocalin

Iron, Gallium, Plutonium

Kd=10^{-49}M

Triserine Lactone

Catechol

Bacterial Siderophores

Pocket #1

Pocket #2

Pocket #3

Molecular Cell, 2002
NGAL is Made in Mouse Kidney

Control          Volume Depletion          Ischemia

CD             Loops of Henle

Xu and Shen, JASN
NGAL is Made @Ischemia
NGAL@Ischemia not Volume Depletion

Paragas & Qiu, Barasch Nature Medicine 2011
(1) NGAL Temporal Relationship = Stimulus

Paravicini Barasch Pediatric Research, 2010

Devarajan, Lancet 2005

Krawczeski, Goldstein, Devarajan et al, JACC 2011
AKI and NGAL

Rapid Rise ~3 hrs

Sensitive Dose Dependent

Specific for Sustained Azotemia
NGAL in ED Predicts AKI

Nickolas et al Annals of Internal Medicine; JACC
### Table 2b. Summary Statistics for Urine Biomarkers by Diagnosis

<table>
<thead>
<tr>
<th></th>
<th>PRA (N = 55)</th>
<th>HRS (N = 15)</th>
<th>ATN (N = 39)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tubular injury markers</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NGAL (ng/mL)</td>
<td>54 (17-180)</td>
<td>115 (51-373)</td>
<td>565 (76-1000)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IL-18 (pg/mL)</td>
<td>15 (15-49)</td>
<td>37 (15-90)</td>
<td>124 (15-325)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>KIM-1 (ng/mL)</td>
<td>4.4 (1.8-11.7)</td>
<td>7.6 (4.5-10.1)</td>
<td>8.4 (4.1-18.3)</td>
<td>0.03</td>
</tr>
<tr>
<td>L-RBP (ng/mL)</td>
<td>9 (4-18)</td>
<td>14 (6-20)</td>
<td>27 (8-103)</td>
<td>0.02</td>
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<tr>
<td>Tubular function marker</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>FENa (%)</td>
<td>0.27 (0.13-0.58)</td>
<td>0.10 (0.02-0.23)*</td>
<td>0.31 (0.12-0.65)***</td>
<td>0.01</td>
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<tr>
<td>Glomerular injury marker</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Albumin (mg/dL)</td>
<td>21 (4-70)</td>
<td>24 (13-129)</td>
<td>82 (44-253)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

### References

- Verna and Nickolas, 2012
- Blecher and Parikh
- uNgal Associates with Sustained sCr
<table>
<thead>
<tr>
<th>RISK</th>
<th>DIPSTICK (ng/mL)</th>
<th>ELISA (ng/mL)</th>
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<tbody>
<tr>
<td>Low</td>
<td>7.54</td>
<td>3.99</td>
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<tr>
<td>Inconclusive</td>
<td>134.23</td>
<td>83.85</td>
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<tr>
<td>High</td>
<td>1076.99</td>
<td>525.59</td>
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</table>
Is NGAL Unique? No!
Volume Depleted vs Ischemia

<table>
<thead>
<tr>
<th>GENE</th>
<th>ISCHEMIA REPERFUSION</th>
<th>VOLUME DEPLETION</th>
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</thead>
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<tr>
<td></td>
<td>Region with Most Significant FC</td>
<td>Fold Change</td>
</tr>
<tr>
<td>Spp1</td>
<td>OSOM</td>
<td>49.50</td>
</tr>
<tr>
<td>Cxcl1</td>
<td>OSOM</td>
<td>219.96</td>
</tr>
<tr>
<td>Lcn2</td>
<td>OSOM</td>
<td>214.29</td>
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<tr>
<td>Clu</td>
<td>OSOM</td>
<td>32.67</td>
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<tr>
<td>Havcr1</td>
<td>OSOM</td>
<td>536.83</td>
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<tr>
<td>Timp1</td>
<td>OSOM</td>
<td>123.42</td>
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<td>Naglu</td>
<td>OSOM</td>
<td>0.50</td>
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<td>Ilf1m</td>
<td>OSOM</td>
<td>145.76</td>
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<td>LIF</td>
<td>OSOM</td>
<td>89.14</td>
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<tr>
<td>B2m</td>
<td>ISOM</td>
<td>0.45</td>
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<td>Timp2</td>
<td>ISOM</td>
<td>0.43</td>
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<tr>
<td>Gsta1</td>
<td>Cortex</td>
<td>42.19</td>
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<tr>
<td>Il6</td>
<td>Cortex</td>
<td>468.98</td>
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<td>Gstp1</td>
<td>Cortex</td>
<td>2.16</td>
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<tr>
<td>Ccl2</td>
<td>OSOM</td>
<td>11.42</td>
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<td>Cyr61</td>
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<td>5.41</td>
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<td>S10a3</td>
<td>OSOM</td>
<td>2.77</td>
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<tr>
<td>Fabp1</td>
<td>Cortex</td>
<td>7.93</td>
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<tr>
<td>Vegfb</td>
<td>OSOM</td>
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<tr>
<td>Serpina1a</td>
<td>Glom</td>
<td>19.72</td>
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<td>Ntn1</td>
<td>ISOM</td>
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<tr>
<td>Igfbp7</td>
<td>OSOM</td>
<td>0.59</td>
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<tr>
<td>Tnfsf10</td>
<td>OSOM</td>
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<tr>
<td>Hgf</td>
<td>ISOM</td>
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<tr>
<td>Col4a1</td>
<td>Cortex</td>
<td>2.05</td>
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<tr>
<td>Vegfa</td>
<td>OSOM</td>
<td>0.64</td>
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<tr>
<td>Ccl11</td>
<td>OSOM</td>
<td>3.28</td>
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<tr>
<td>Vnn1</td>
<td>OSOM</td>
<td>2.94</td>
</tr>
</tbody>
</table>

Xu, JASN
Is NGAL Unique? No!
Volume Depleted vs Ischemia

Ischemic AKI
- Fibrin clotting cascade
- Cytokine & chemokine signaling
- TLR signaling
- JAK/STAT signaling
- TGF-β signaling
- S1P, S1P1 pathways
- MAP Kinase pathways

Pre renal AKI
- TCA Cycle
- Gluconeogenesis
- Transport of Ions & Amino Acids
Biomarkers Change the Definition of AKI

- **Stimulus** → **Cell Response** → **Troponin** → **EKG Changes** → **Decreased Ejection Fraction**
  - Vasodilator; Thrombolytic

- **Stimulus** → **Cell Response** → **Cell Damage** → **Organ Damage** → **Rise in Creatinine**
  - Antibiotic

- **Stimulus** → **Cell Response** → **?Cell Damage** → **?Organ Damage** → **Rise in Creatinine**
  - Saline

**Time x Severity**

Organ Failure
Biomarkers Change the Definition of AKI

- **SCr normal**
  - Tubular damage without excretory dysfunction e.g. subclinical AKI
  - Normal urine output → ↑Urine biomarker

- **SCr elevated**
  - Tubular damage with excretory dysfunction e.g. sepsis, ischaemia nephrotoxin-induced tubular damage
  - Low urine output → ↑Urine biomarker

- **SCr normal**
  - No tubular damage and no excretory dysfunction e.g. ‘normal kidneys’
  - Normal urine output → ↓Urine biomarker

- **SCr elevated**
  - No tubular damage but with excretory dysfunction e.g. volume depletion, mild CHF, diuretics
  - Low urine output → ↓Urine biomarker
Worsening Renal Function in Acute Heart Failure Patients Undergoing Aggressive Diuresis Is Not Associated with Tubular Injury
Ahmad, Bonventre, Wilson, Coca, Testani Circulation
PCr lacks intrinsic characteristics to act as a surrogate marker of injury. It marks functional loss, but in the absence of steady state, PCr cannot quantify this loss.

- All diagnoses of Acute Kidney Injury (tubular injury) based on PCr are
  - Retrospective in nature,
  - Must fail volume challenge to be considered injury,
  - Ischemic $\Delta sCr \neq$ Volume Related $\Delta sCr$ are unrelated in terms of gene expression and of course treatment.

$\Delta sCr$ may (ischemia) or may not (rapid reversible, diuretics) correspond to NGAL & Biomarkers

- NGAL is not unique; there are 100’s of ischemic biomarkers that do not respond to volume perturbation.
- Conversely, there are volume related markers.

In Summary

Most changes in PCr are due to volume related events and are short lived.

We suggest a “second hit” convert a volume depleted state into the ischemic state.
ARDS and AKI: 22-44% of patients with all forms of pre Covid ARDS develop elevated Pcr and 10-14% of patients with all forms of pre Covid ARDS required dialysis.

SARS-CoV-2 AKI: is associated with acute loss of renal function (SCr elevates rapidly) that persisted through hospitalization (Sustained AKI = ATN) usually requiring dialysis.

Many patients seen by Nephrology in ICU had fever and dyspnea, had evidence of ARDS and were on pressors and had elevated Scr with anuria ~24hrs (10 new patients per day). Lab tests showed inflammatory markers, including interleukin-6. In contrast ~30% of the patients demonstrated elevated Scr and inflammatory markers without ARDS.

One quarter required hemodialysis (2-3 new patients/day) for approximately 2-4 weeks, and of these one fifth were able to stop dialysis and two fifths died (lower than other centers).

In total, one third of ICU patients required hemodialysis. Most of the patients ~50% of patients had DM or CKD; ~50% were on ACEI or ARB’s.

Covid Pathology: Notable for a wide range of pathologies including various types of immune mediated Glomerulopathy with evidence of cytokine storm such as interferon responses. Acute Tubular Injury was common characterized by tubular ectasia and loss of brush boarder and vacuolization of the proximal tubular cells. There was interstitial inflammation and edema.
COVID AKI-Not Volume Depletion?
COVID AKI-Not Volume Depletion?

Kidney360 Publish Ahead of Print, published on June 2, 2020 as doi:10.34067/KID.0003352020

Urinary Sediment Microscopy in Acute Kidney Injury Associated with COVID-19

Cesar F. Hernandez-Arroyo¹, Vipin Varghese², Muner M.B. Mohamed¹, Juan Carlos Q. Velez¹,²
COVID AKI-Not Virus?

Rare Detection of CoV-2 in Kidney

- SARS-CoV-2 (Control probe)
- SARS-CoV-2 (C1 probe)
- SARS-CoV-2 (C1 probe)
Renin-angiotensin system inhibition in COVID-19 patients

A. A. F. de Vries

![Diagram of the renin-angiotensin system inhibition in COVID-19 patients.](image-url)
Renin-angiotensin system inhibition in COVID-19 patients

A. A. F. de Vries

**Healthy lung tissue**
- ADAM17
- ACE secretase
- ACE2
- ACE
- sACE
- sACE2
- AT1R
- MasR

**SARS-CoV-2-infected lung tissue**
- viral particle
- viral genome
- sACE
- sACE2
- AT1R
- MasR

- Detrimental
  - ACE2 (AngI)/AT1R
  - ACE2 (AngI-8)/AT1R

- Beneficial
  - ACE2 (AngI)/MasR
  - ACE2 (AngI-8)/MasR

- Balanced ACE/ACE2 activity
- ACE2 ↓: ACE/ACE2 balance disturbed
- SARS-CoV-2-induced inflammation & SARS-CoV-2-mediated cell death

Neth Heart J
https://doi.org/10.1007/s12471-020-01439-5
Renin-angiotensin system inhibition in COVID-19 patients

A. A. F. de Vries

https://doi.org/10.1007/s12471-020-01439-5
Summary

• RIFLE KDIGO and AKIN are insufficient as diagnostic criteria.
• Biomarkers change the definition of AKI because they demonstrate a set of molecular responses to damage stimuli.
• Stimulus=Cell Type?
• Focusing on collecting duct
  – Acute Tubular Injury=>a rich inflammatory response (innate, complement, coagulation), response to iAKI = response to UTI.
  – Volume Depletion=>a non inflammatory metabolic response
• The Evolutionary Significance the iAKI/UTI response is to defeat UTI
• The Evolutionary Significance vAKI is to prolong life in the setting of volume depletion.
• Covid-19 represents an inflammatory form of acute kidney damage (Upper Airway => Lower Airway => Systemic Inflammatory disorder). Kidney Damage is not due to volume depletion nor viral invasion.
Imagine a patient presenting with fever, loss of appetite, low blood pressure (105/80) and "pneumonia vs atelectasis" is found. The patient wants antibiotics and discharge. However, the patient’s Pcr = 1.5 (baseline Pcr = 1.0), defining what is called “Acute Kidney Injury, AKI”. What should the doctor do?

A patient with CHF suffers SOB. Diuretics are initiated, but Pcr rises from Pcr=1.0 to Pcr=2.0. The cardiologist says there is now “AKI”. What should the cardiologist do?

Imagine a patient presenting after a motorcycle accident with reddish-brown urine, and an elevated CPK level. You want to perform a CT Scan with contrast. Pcr = 1.0. What should the doctor do?
Thank you.

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  – Kiyoshi Mori, Kyoto Graduate University     --- Iqbal Hamza, U of Maryland
  – Guanhu Bao, Anhui University                 --- Peter Sims, Columbia
  – Wenqiang Yu, Fudan University, Shanghai      --- Nick Tatonetti, Columbia
  – Meghan Sise, Columbia=>Harvard              --- Qais Al-Awqati, Columbia
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Alex Tornato Justin Koenig Rebecca Wax Efrat Bruck
THANK YOU

PLEASE REACH OUT IF YOU HAVE ANY QUESTIONS

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UPDATE FROM THE FRONT LINES: PROTESTORS, TEAR GAS & COVID-19
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Rittirak Othong, MD, FACMT, FTCEP
• Deputy Director, Department of Emergency Medicine
• Vajira Hospital, Navamindradhiraj University
• Bangkok, Thailand
During COVID-19 Pandemic

Here Are the 98 U.S. Cities Where Protesters Were Tear-Gassed

By K.K. Rebecca Lai, Bill Marsh and Anjali Singhvi  June 16, 2020

Tear Gas Use During COVID-19 Pandemic Irresponsible; Moratorium Needed, Says American Thoracic Society

11-Jun-2020 4:50 PM EDT, by American Thoracic Society (ATS)

Lebanese protesters are sprayed with water during a protest against corruption and against the government’s failure to resolve a crisis over rubbish disposal, near the government palace in Beirut on August 23, 2015.
PROTESTERS REACT TO TEAR GAS AT GEORGE FLOYD PROTESTS IN WASHINGTON, D.C.
WHAT ARE THEY?

Powder

Not Gas!

Chemical Irritant

Commonly referred to as “Tear Gas”

Carron PN, Yersin B. BMJ. 2009;19;338:b2283.
## Commonly Used Chemicals

<table>
<thead>
<tr>
<th>Chemical Code</th>
<th>Description</th>
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<tbody>
<tr>
<td>CN</td>
<td>Chloroacetophenone</td>
</tr>
<tr>
<td>CS</td>
<td>Chlorobenzylidene-malononitrile (Corson and Stoughton)</td>
</tr>
<tr>
<td>CR</td>
<td>Dibenzoxazepine</td>
</tr>
<tr>
<td>DM</td>
<td>Adamsite or Diphenylaminochloroarsine</td>
</tr>
<tr>
<td>OC</td>
<td>Pepper Spray Oleoresin capsicum</td>
</tr>
<tr>
<td>PAVA</td>
<td>Pelargonic acid vanillylamide</td>
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Carron PN, Yersin B. BMJ. 2009;19;338:b2283.
AGENT – HOST - ENVIRONMENT

Victim Characteristics

Agent: Chemical, Device

Environment
# Physical and Chemical Characteristics of Tear Gases

*Pharmaceuticals and Medical Care.* Carron PN, Yersin B. BMJ. 2009;19;338:b2283.

<table>
<thead>
<tr>
<th>Name</th>
<th>Characteristics</th>
<th>Time to activation</th>
<th>Duration of action (minutes)</th>
<th>Relative potency*</th>
<th>Ict 50 (mg/min per m^3^)†</th>
<th>Lct 50 ‡ (mg/min per m^3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloroacetophenone</td>
<td>Apple odour; powder or emulsion; aerosol</td>
<td>3-10 seconds</td>
<td>10-20</td>
<td>1</td>
<td>20-50</td>
<td>8500-25 000</td>
</tr>
<tr>
<td>Chlorobenzylidene malononitrile</td>
<td>Pepper odour; microparticles; dispersing effect (grenades)</td>
<td>10-60 seconds</td>
<td>10-30</td>
<td>5</td>
<td>4-20</td>
<td>25 000-100 000</td>
</tr>
<tr>
<td>Dibenzoazepine</td>
<td>Odourless; aerosol; persists for prolonged periods in the environment or on clothes</td>
<td>Instantaneous</td>
<td>15-60</td>
<td>20-50</td>
<td>0.2-1</td>
<td>&gt;100 000</td>
</tr>
<tr>
<td>Diphenylaminochloroarsine</td>
<td>Odourless or slightly bitter almond odour; emetic</td>
<td>Rapid</td>
<td>&gt;60</td>
<td>0.5-2</td>
<td>50-100</td>
<td>10 000-35 000</td>
</tr>
<tr>
<td>Oleoresin capsicum</td>
<td>Pepper odour; persists for prolonged periods in the environment or on clothes; short distance spray</td>
<td>Rapid</td>
<td>30-60</td>
<td>Not applicable</td>
<td>Not applicable</td>
<td>&gt;100 000</td>
</tr>
</tbody>
</table>

*Refers to the irritant effect.
†Ict 50=the concentration that causes incapacitation in 50% of individuals after one minute.
‡Lct 50=the concentration that causes death in 50% of individuals after one minute.
VICTIM CHARACTERISTICS

- Asthma
- Chronic obstructive pulmonary disease
- Cardiovascular disease
- Severe hypertension
- Young children
- Patients over 60 years
- Ocular diseases
- Contact lenses
RESPIRATORY EFFECTS

- Irritation of the nose, throat, and chest
- Coughing and difficulty breathing
- Exacerbation of existing asthma or chronic lung disease
- Rare severe complications
- Potential long-term complications: Reactive Airway Dysfunction Syndrome
CUTANEOUS EFFECTS

- Burning sensation increases with presence of moisture, and higher temperature
- Rashes, blisters, and burns
- Heavy exposures produce vesicles and reddening that resemble a second-degree burn 14-16 hours postexposure, if exposed skin left without decontamination
- Delayed dermal manifestations (12-24 h); allergic contact dermatitis and acute generalized pustulosis

Photograph: Courtesy of CG Hurst, US Army Medical Research Institute of Chemical Defense.
OCULAR EFFECTS

- Lacrimation, eye burning
- Blepharospasm, transient conjunctivitis, lid swelling
- More severe eye injuries are possible but not frequent
- Remove contact lenses
- Blow air or irrigate with saline or water
- Slit lamp exam with possible topical antibiotics and mydriatics
- Oral analgesics but not topical analgesics
- Remove victims from the scene
- Prevent secondary exposures
  - Decontamination
  - Personal protective equipment
- Decontamination
  - Dry and Wet
- Supportive care
TEAR GAS USE - BANGKOK – THAILAND, AUGUST 2008 – DR. RITTIRAK OTHONG

- No previous knowledge about the tear gas used
- Little knowledge of the emergency department preparedness and management of victims

http://www.prachathon.org/forum/index.php?topic=8442.0
- 5-8 minutes from Scene to Vajira Hospital
- No Emergency Department notification, no scene decontamination

http://www.osknetwork.com/modules.php?name=News&file=article&sid=3073&mode=read&order=0&thold=0
- Porters and EMTs brought 19 victims into the triage area
- Male patients → dry decontamination
- Female patients → wet and dry decontamination

Health care providers wore plastic aprons, gloves, surgical masks and protective glasses (some with goggles)
- Porters and EMTs brought victims into triage area
- Male patients → dry decontamination
- Female patients → wet & dry decontamination

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Health care providers wore plastic aprons, gloves, surgical masks and protective glasses (some with goggles)
LESSONS IDENTIFIED

- Tear gas is not a gas, it’s a solid particle. Dry decontamination is not adequate.
- Set up a decontamination facility outside the ED.
- Took several days or weeks for turmoil to escalate to the climax and tear gas would be employed. Pre-set up the decontamination facility while situation escalating.
- Use appropriate PPE for triage-decontamination team.
- Sorting and separating patients according to their roles.
First Aid Instructions for Tear Gas Exposure

For Medical Personnel

1. Leave scene immediately, be above wind, and at higher place
2. Take off contaminated clothing, put them in plastic bag and seal it
3. Take off your ornament
4. Take off contact lens
5. Rinse water to wash out residue from eyes, mouth, body at least for 15 min. (Don’t use lime juice or milk)

For General Public

1. Wear long sleeve shirt and pants; bring another set of clothing for change
2, 3, 4, 5: Bring swimming goggles, a face mask, water and a plastic bag
6: If you have asthma, bring your inhaler

Tear gas causes irritation such as tearing, red eyes, sore mouth and throat. If all do not go way in 30 min, seek medical care.


- Decon facility should be outside ED with good ventilation
- Triage area after decon can be in front of or in ED
- Decon victims with copious water
- Use PPE level C with canister type facemask
- Take off all victim clothing and ornaments; and put them in plastic bags and seal them
- Don’t open sealed bag, tear gas will be released
- Warning for flaming
Thank You for Your Attention!
Q&A
ON-DEMAND RESOURCES

All webinars are recorded and posted to the ACMT website

www.acmt.net/covid19web

Questions?
Write to: info@acmt.net
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Wednesday, June 24, 2020
3:00 PM EDT

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