الجمعية التونسية لطب الانعاش
ASSOCIATION TUNISIENNE DE REANIMATION

INTOXICATION AU METHANOL.

ETAT DES LIEUX EN TUNISIE ET PRINCIPES DE PRISE EN CHARGE.

METHANOL POISONING

FROM REVIEW OF LITERATURE TO PROTOCOLS

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03 December 2021

CONFLICT OF INTEREST DISCLOSURE.

I have No conflict of Interest to report in relation to this Presentation.

American Academy of Clinical Toxicology Practice Guidelines on the Treatment of Methanol Poisoning

The American Academy of Clinical Toxicology Ad Hoc Committee on the Treatment Guidelines for Methanol Poisoning:* Donald G. Barceloux, G. Randall Bond, Edward P. Krenzelok, Hannah Cooper, and J. Allister Vale

American Academy of Clinical Toxicology, Harrisburg, Pennsylvania

C TOX 2002

Intensive Care Med (2005) 31:189–195
DOI 10.1007/s00134-004-2521-0

REVIEW

ICM 2005

Bruno Mégarbane
Stephen W. Borron
Frédéric J. Baud

Current recommendations for treatment of severe toxic alcohol poisonings

Recommendations for the Role of Extracorporeal Treatments in the Management of Acute Methanol Poisoning: A Systematic Review and Consensus Statement

CCM 2015

Darren M. Roberts, PhD, FRACP[1,2]; Christopher Yates, MD[3]; Bruno Megar
James F. Winchester, MD[5]; Robert Maclaren, PharmD[6]; Sophie Gosselin, MD[7];
Thomas D. Nolin, PharmD, PhD[8,9]; Valéry Lavergne, MD[10]; Robert S. Hoffman, MD[11];
Marc Ghannoum, MD[12]; on behalf of the Extracorporeal Treatments in Poisoning Workgroup

Réanimation 2001 ; 10 : 426-34

S1164675601001438/SSU

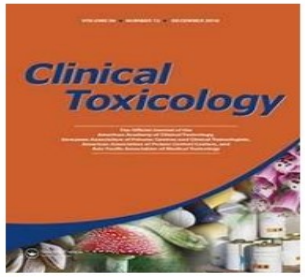
MISE AU POINT

Intoxication aiguë par les glycols et alcools toxiques : diagnostic et traitement

B. Mégarbane*, N. Brahmi, F. Baud

Réanimation médicale et toxicologique, hôpital Lariboisière, 2, rue Ambroise-Paré, 75010 Paris, France

(Reçu le 31 janvier 2001 ; accepté le 30 mars 2001)



Clinical Toxicology

ISSN: 1556-3650 (Print) 1556-9519 (Online) Journal homepage: https://www.tandfonline.com/loi/ictx20

Consensus statements on the approach to patients in a methanol poisoning outbreak

Hossein Hassanian-Moghaddam, Nasim Zamani, Darren M. Roberts, Jeffrey Brent, Kenneth McMartin, Cynthia Aaron, Michael Eddleston, Paul I. Dargan, Kent Olson, Lewis Nelson, Ashish Bhalla, Philippe Hantson, Dag Jacobsen, Bruno Megarbane, Mahdi Balali-Mood, Nicholas A. Buckley, Sergey Zakharov, Raido Paasma, Bhavesh Jarwani, Amirhossein Mirafzal, Tomas Salek & Knut Erik Hovda

INTRODUCTION

- A public health problem in many countries..
- Known as “Wood Alcohol”.
- Component of washing fluids, antifreeze formulations, photocopying fluids, perfumes and paint removers.
- Dermal application, inhalation or ingestion…
- Despite legislations, it exists illegally: spirits, cologne..

Brahmi N et al Clin Tox 2007

WHEN DO WE SUSPECT IT?

CONTEXT

- Young age…
- Poor / Rich conditions, Week end party…
- Report of deaths…
- Clinical signs…

Am Academy CLIN TOX 2002

CLINICAL PRESENTATION (1)

- Latent period : 12-24 Hours.
- First symptoms:

➢ Headache, vertigo.

➢ Abdominal pain, vomiting.

Surgical emergency?

➢ Hyperventilation related to metabolic acidosis.

Brahmi N et al Clin Tox 2007

CLINICAL PRESENTATION (2)

Then:

- *Ocular signs (12-24 h).*

➢ Blurred vision.

➢ Visual loss.

➢ Impaired color Vision.

➢ Areactive mydriasis (papillary edema)

Case series suggest that visual dysfunction occurs when formate concentrations exceed 20–30mg/dL (200–300mg/L)

Am Academy CLIN TOX 2002

CLINICAL PRESENTATION (3)

- *Shock* (hypovolemia, myocardial depression) usually leading to death.
- *Respiratory failure.*
- *Convulsions-Seizures.* (Parkinson Like extra pyramidal syndrome).
- *Renal failure* (Rhabdomyolyses, Myoglobinuria).
- *Pancreatitis.*
- *Hypomagnesemia, hypokalemia…*

Am Academy CLIN TOX 2002

DIAGNOSIS (WHO 2014)

- Combination of the history, the signs and symptoms and the following laboratory findings:

➢ *Metabolic acidosis*

➢ *Elevated anion gap*

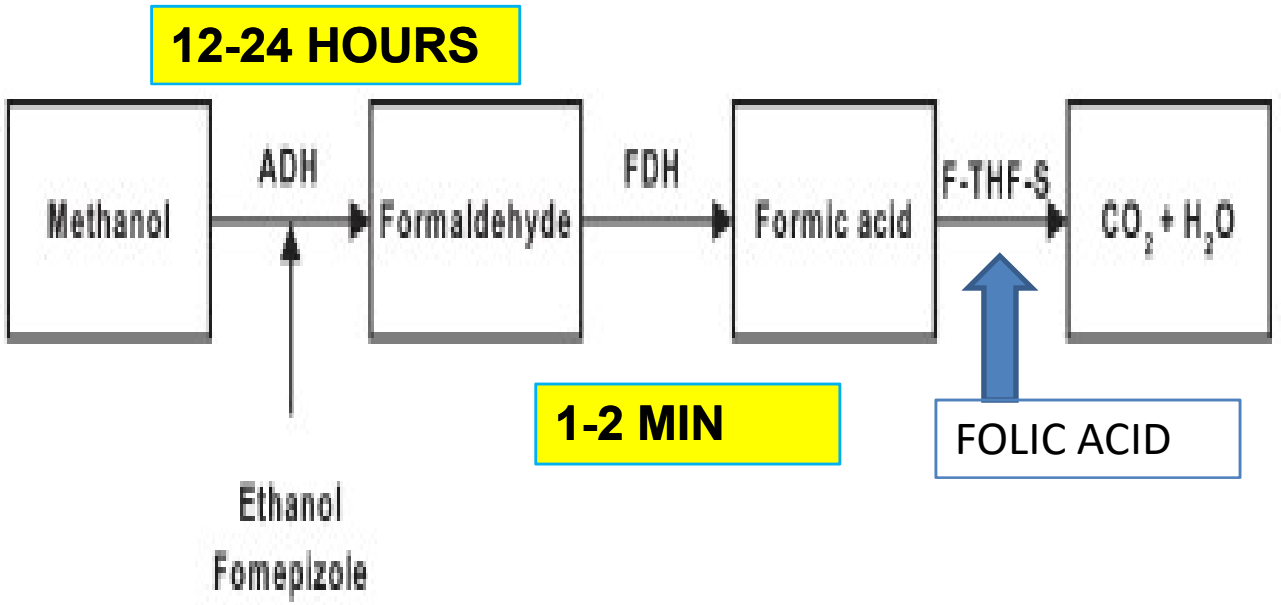
➢ *Elevated osmolal gap (the difference between measured and calculated osmolality)*

➢ *Positive serum methanol and/or serum formate assay.*

➢ *Serum methanol level can be estimated by multiplying the osmolar gap by 3.2.* ***(Hovda and al.Intensive Care Med (2004))***

MECHANISMS OF TOXICITY?

MECHANISMS OF TOXICITY (1)



ADH: alcohol dehydrogenase; FDH: formaldehyde dehydrogenase
F-THF-S: 10-formyl tetrahydrofolate synthetase

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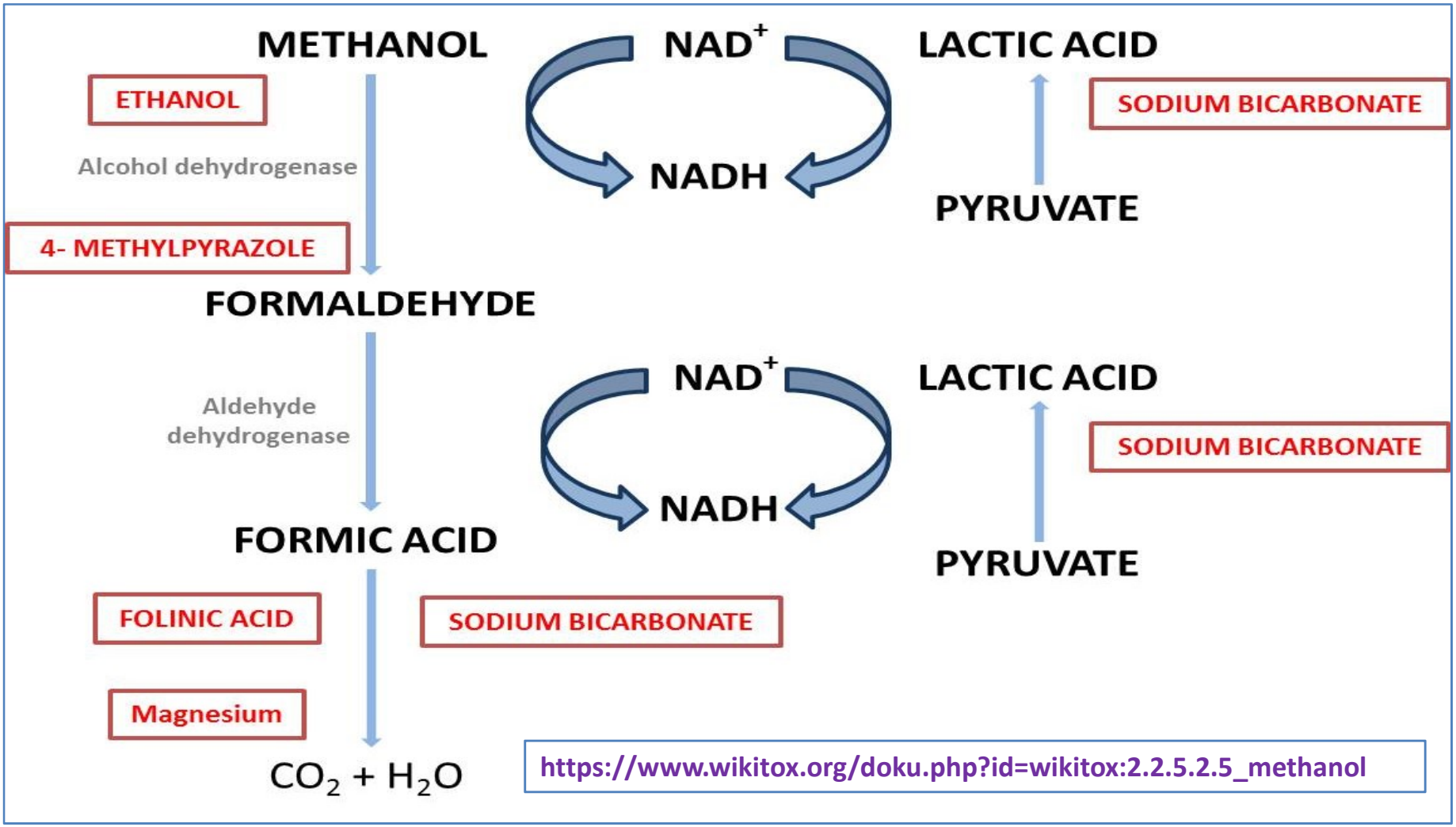
MECHANISMS OF TOXICITY (2)



MECHANISMS OF TOXICITY (3)

- Bioavailability ingestion: 100%.
- Lethal dose: 1ml/Kg.
- Factors controlling the rate of formic acid metabolism in humans:

➢ **Presence of adequate dietary folic acid (tetrahydrofolate is derived from folic acid).**

➢ **Efficiency with which tetrahydrofolate is regenerated during formate oxidation.**

the potential usefulness of folate in the treatment of methanol poisoning

MECHANISMS (4)
THE ROLE OF FORMIC ACID
"THE SILENT KILLER"

- **Inhibition of Cytochrome Oxidase** Complex in the mitochondria "histotoxic hypoxia."
- **Metabolic acidosis:** Lactic acid, Formate.
- **Ocular Toxicity:** optic disc edema, breakdown of the myelin sheaths and optic nerve lesions.
- **Neurotoxicity:** edema and necrotic damage to the basal ganglia of the brain, ***the putamen***, and hemorrhages in the subcortical white matter (Mechanism?).

Brahmi N et al Clin Tox 2007

RadioGraphics

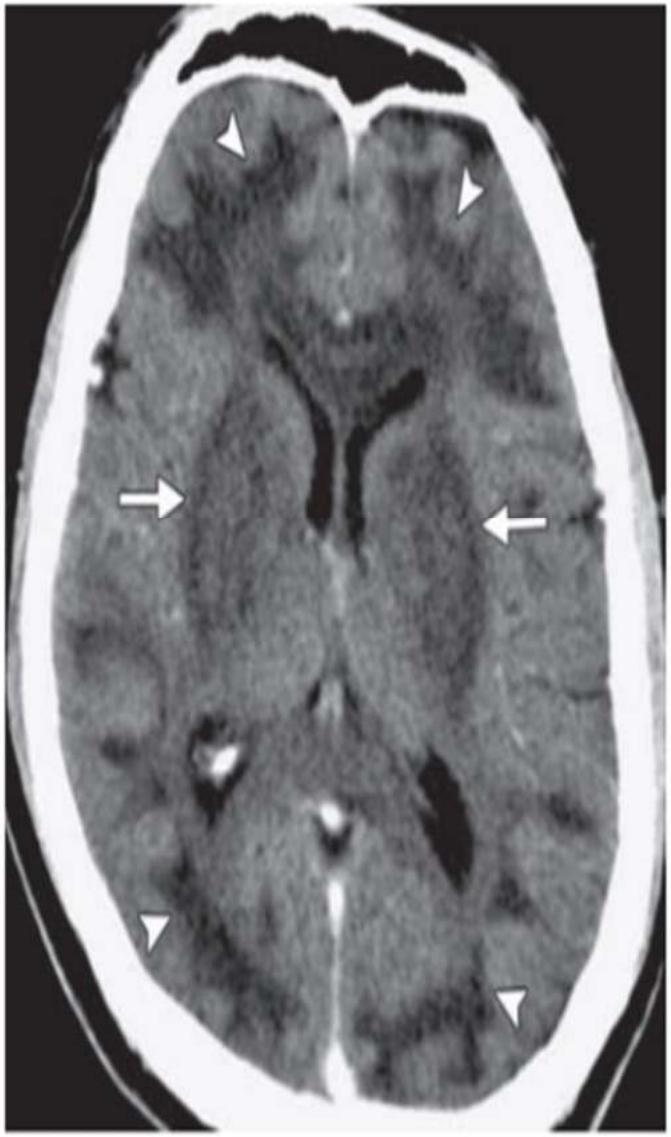
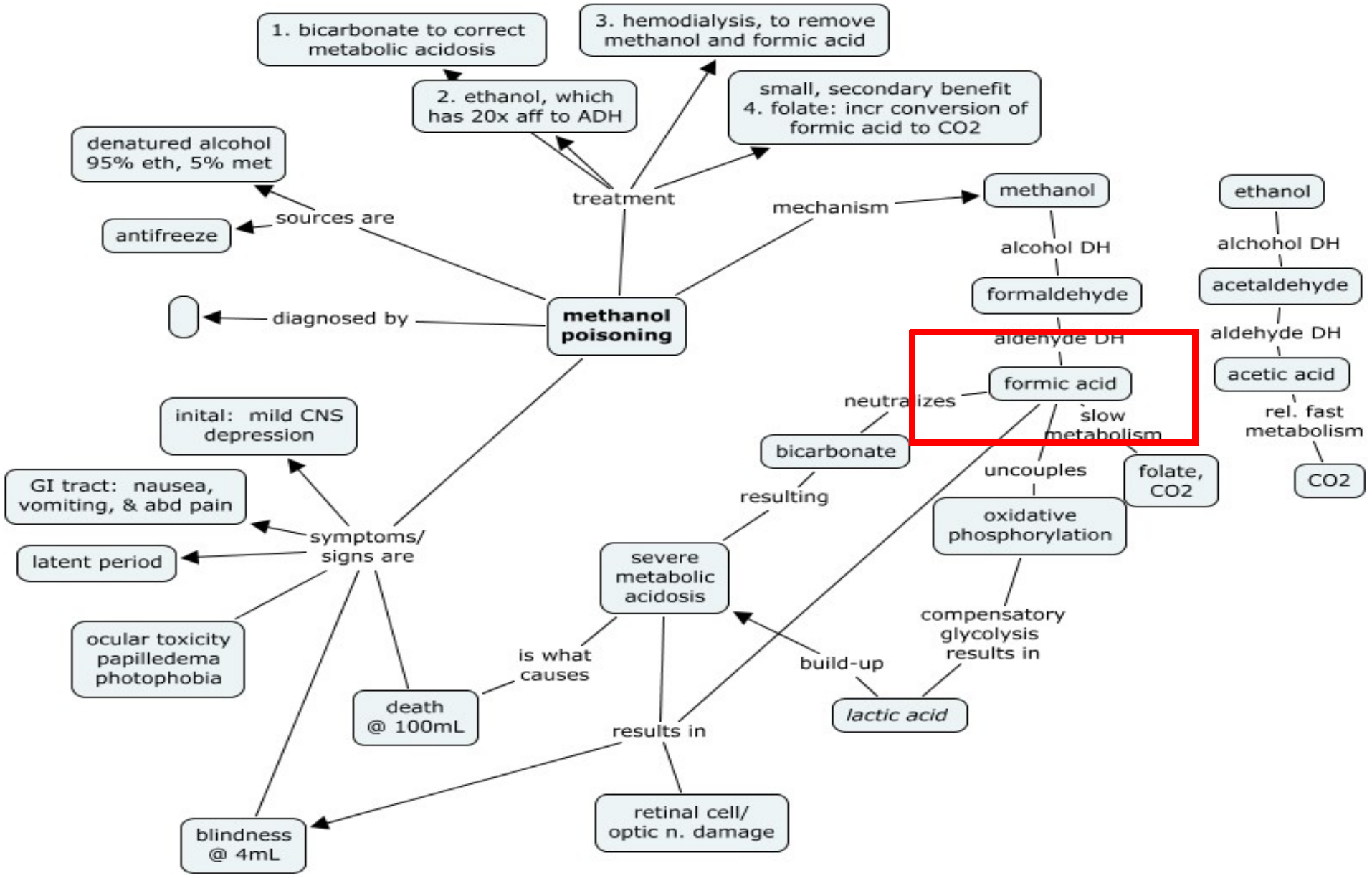


Figure 4. Methanol poisoning in a 41-year-old man who presented with altered mental status and retrobulbar pain. Contrast material–enhanced CT scan demonstrates hypoattenuating areas in the lentiform nuclei (arrows), corpus callosum, and subcortical deep white matter in the frontal and parieto-occipital regions (arrowheads). (Courtesy of Anirudh Kohli, MD, Breach Candy Hospital Trust, Mumbai, India.)

Blindness: Retinal toxicity of Methanol.



antidotes for toxic agents - methanol poisoning (ihmc.us)

Visual and neurologic sequelae of methanol poisoning in Saudi Arabia

Alberto Galvez-Ruiz, MD, Sahar M. Elkhamary, MD, Nasira Asghar, PhD, Thomas M. Bosley, MD.

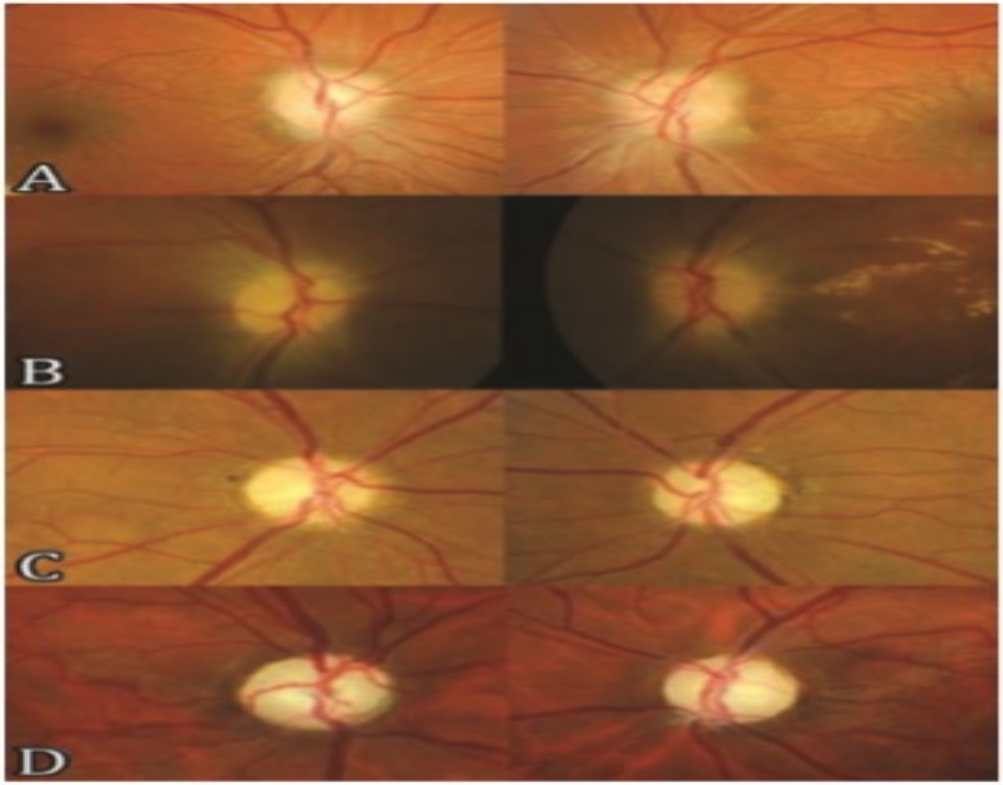


Figure 1 - Acute and chronic fundoscopic changes. Fundus images of 4 patients with the right optic disk displayed on the left side and the left optic disk displayed on the right side. Images showing the optic disks of 2 patients taken within one week of methanol exposure showing modest pallid edema of the optic disks extending onto the peripapillary retina (**Figures 1A & 1B**). Images depicting the optic disks of 2 different patients taken approximately 6 weeks after methanol exposure, and showing flat, moderately pale optic disks bilaterally with no residual optic disk or retinal edema (**Figures 1C & 1D**).

Formate-Induced Inhibition of Photoreceptor Function in Methanol Intoxication[1]

MARINA T. SEME, PHYLLIS SUMMERFELT, MICHELE M. HENRY, JAY NEITZ, and JANIS T. EELLS

Departments of Pharmacology and Toxicology (M.T.S., M.M.H., J.T.E.) and Cellular Biology and Anatomy (P.S., J.N.), Medical College of Wisconsin, Milwaukee, Wisconsin

Accepted for publication December 11, 1998 This paper is available online at http://www.jpet.org

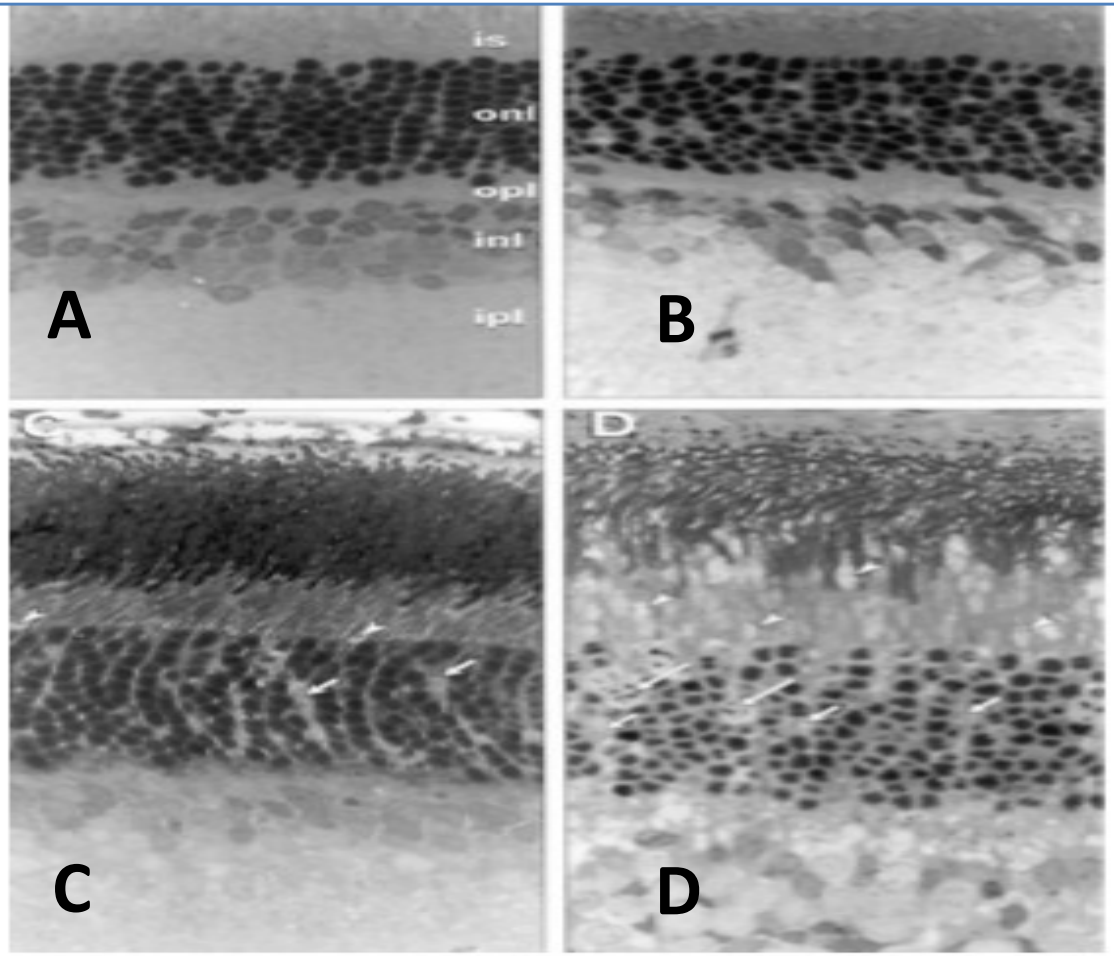


Fig. 6. Effect of methanol intoxication on retinal histology. Light micrographs of retinal tissue prepared from untreated-control rat (A) and methanol-intoxicated rats at 24 h (B), 48 h (C), and 72 h (D) after the initial dose of methanol. Sections were taken from the posterior pole of the retina within two disc diameters of the optic nerve in any direction. A: rpe, retinal pigment epithelium; os, photoreceptor outer segments; is, photoreceptor inner segments; onl, outer nuclear layer; opl, outer plexiform layer; inl, inner nuclear layer; ipl, inner plexiform layer. The short arrows indicate areas of retinal edema, as evidenced by increased spacing between the nuclei in the outer nuclear layer. The arrowheads indicate enlargement and swelling of the photoreceptor inner segments, and the long arrows indicate the fragmented appearance of photoreceptor nuclei. (Toluidine blue, 450×)

Journal of The Association of Physicians of India ▪ Vol. 66 ▪ April 2018

REVIEW ARTICLE

Methanol Poisoning 2018

DB Kadam[1], Sonali Salvi[2], Ajay Chandanwale[3]

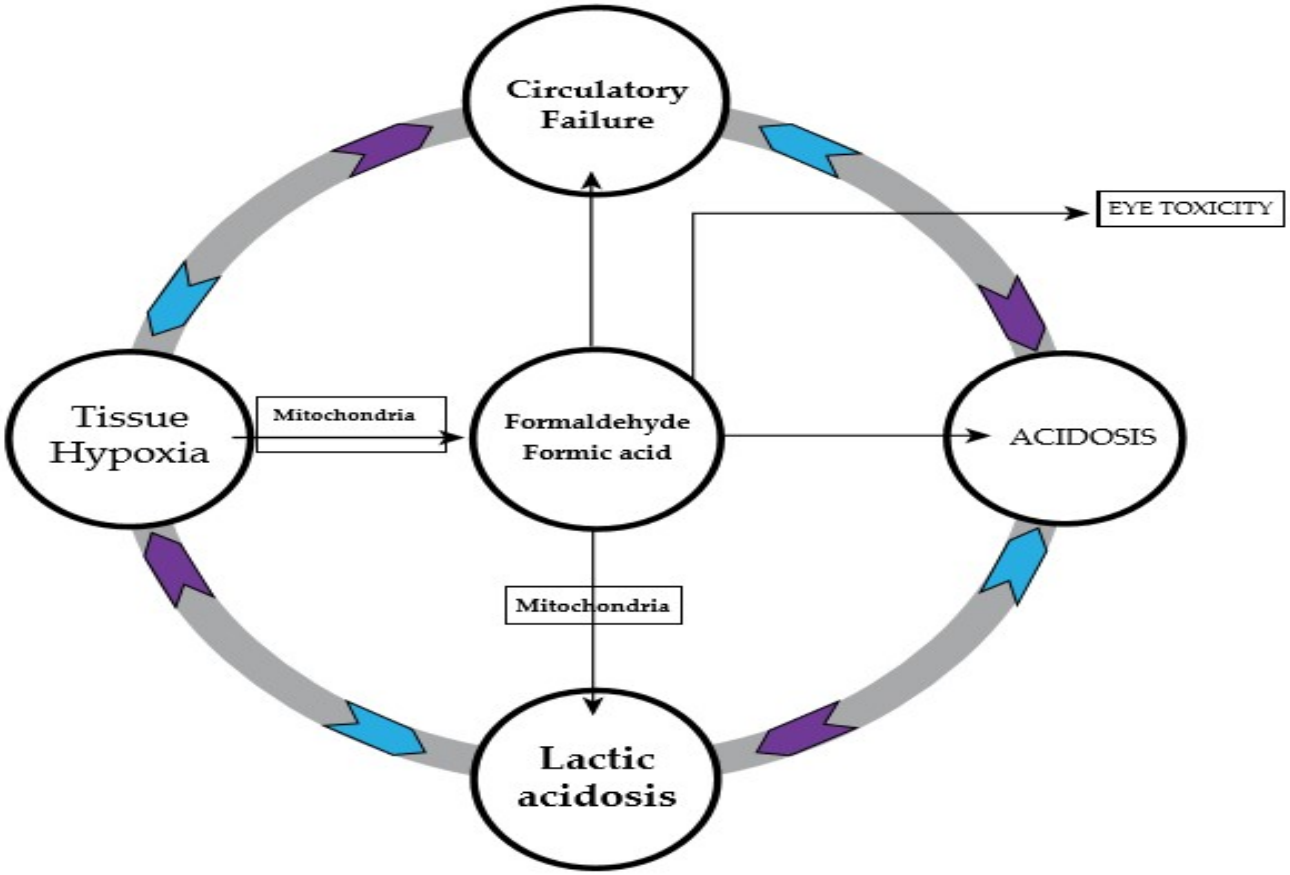


Fig. 6: Diagrammatic representation of circulus hypoxicus

CURRENT SITUATION IN TUNISIA

CENTER MAHMOUD YAACOUB TUNIS, ICU. RETROSPECTIVE STUDY (N=49).

Period	**2003-2020.** **Two Outbreaks (2003, 2019).**
Age **Sex**	33 +/- 16 years. Male (98%).
Methanol Level	1,34+/-1.48g/l [0 , 6,60].
Treatment	Alkalization (80%) Folic Acid (90%). Hemodialysis (n=33 (67%)). Median 6H. Ethanol (n=30 (61%)).
Outcome	Blindness (12%). Mortality: n=12 (24%).

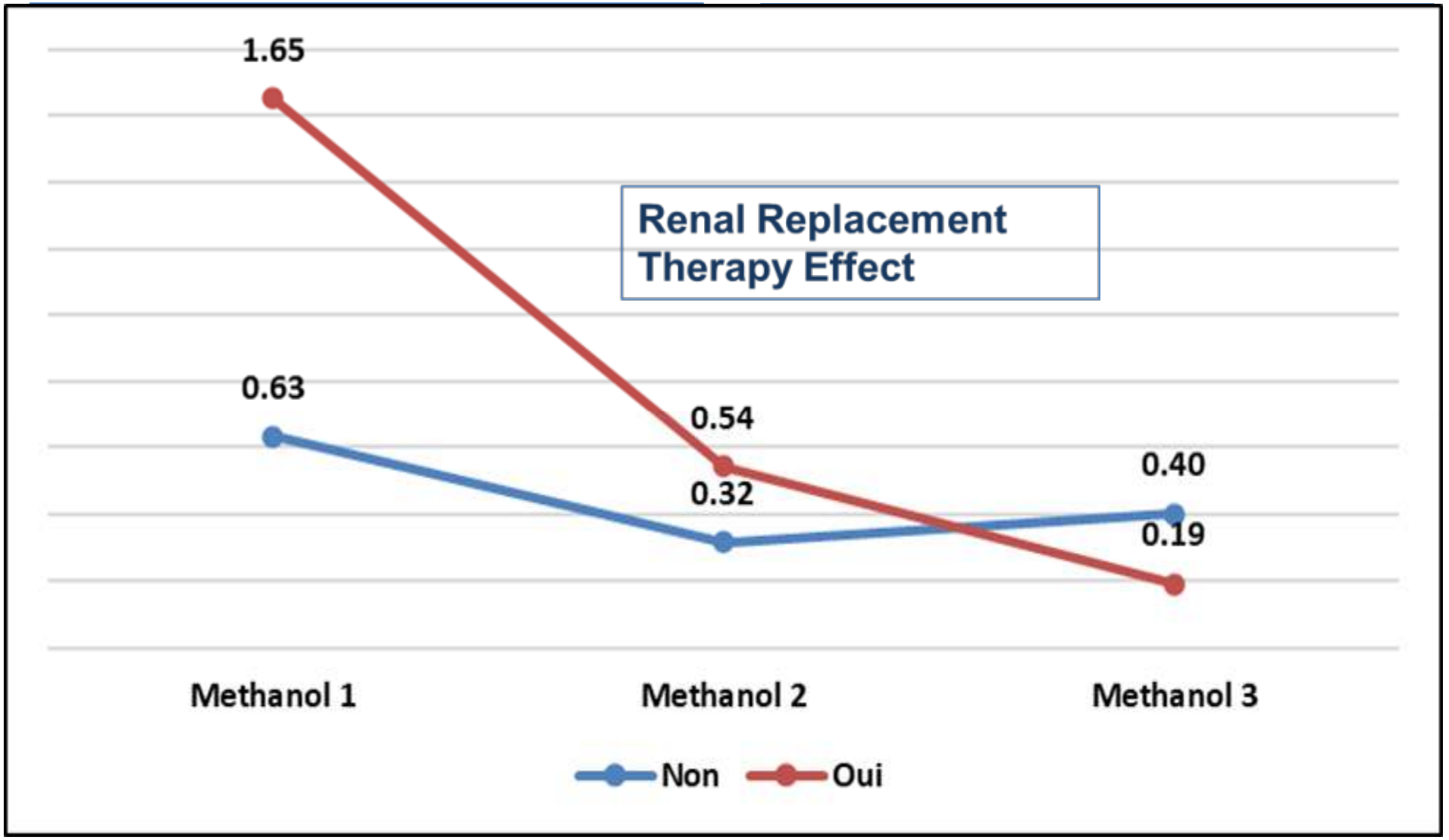
Doctoral Thesis 2003-2020 Submitted

CENTER MAHMOUD YAACOUB TUNIS, ICU. RETROSPECTIVE STUDY (N=49).

Period	**2003-2020.**
Neurological signs **Brain Imaging**	Coma (n=13, 26%). necrosis putamen/subcortical white matter (n=2).
Ocular Signs	Blurred Vision (n=15, 30%). Blindness (n=6, 12%).
pH	<7 (n=10, 20%). 7-7.20 (n=15, 30%). 7.20-7.38 (n=18, 37%).
Correlations	Correlation Glasgow- pH($p<0.001$).

Doctoral Thesis 2003-2020 Submitted

Outcome
Patients included: N=49



Doctoral Thesis 2003-2020 Submitted

OUTCOME (UNIVARIATE ANALYSIS). PATIENTS INCLUDED: N=49

Age	**0.006**
Glasgow Coma Scale (GCS)	0.016
Mydriasis	<0.05
Methanol Level	<0.001
Acute Kidney Injury	<0.05
Metabolic acidosis	<0.001
Anion Gap> 16	0.08

Doctoral Thesis 2003-2020 Submitted

MANAGEMENT PRINCIPLES OF METHANOL POISONING?

BASICS

- Alkalization: 400 à 600 mmol/24 h
- Intubation/ mechanical ventilation.
- Anticonvulsants.
- Fluids/ Inotropes.

• Antidotes:

- Ethanol PO or IV: Loading dose 0.6g/kg
  Maintenance dose: 66 -154 mg/kg/h
  (Objective : Ethanolemia 1 g/l)
- 4 Methyl-pyrazole (Fomepizole): Loading dose 10 to 15mg/kg (30min) then 10mg/kg every 12 hours till complete methanol clearance.

WHAT ELSE?

- Folic Acid: (Oxydation cofactor of formic acid 50 mg IV, every 6 hours.
- RRT: Hemodialysis+++(Visual Disturbance, Renal failure).
- No indication for Gastric Lavage or activated charcoal.

AMERICAN GUIDELINES 2002

Proposed Indications for the Treatment of Methanol Poisoning with Ethanol or Fomepizole

Criteria
Documented plasma methanol concentration >20 mg/dL (>200 mg/L)[53]
Or
Documented recent history of ingesting toxic amounts of methanol and osmolal gap >10 mOsm/kg H_2O[a]
Or
History or strong clinical suspicion of methanol poisoning *and* at least two of the following criteria: (A) Arterial pH <7.3 (B) Serum bicarbonate <20 meq/L(mmol/L) (C) Osmolal gap >10 mOsm/kg H_2O[a]

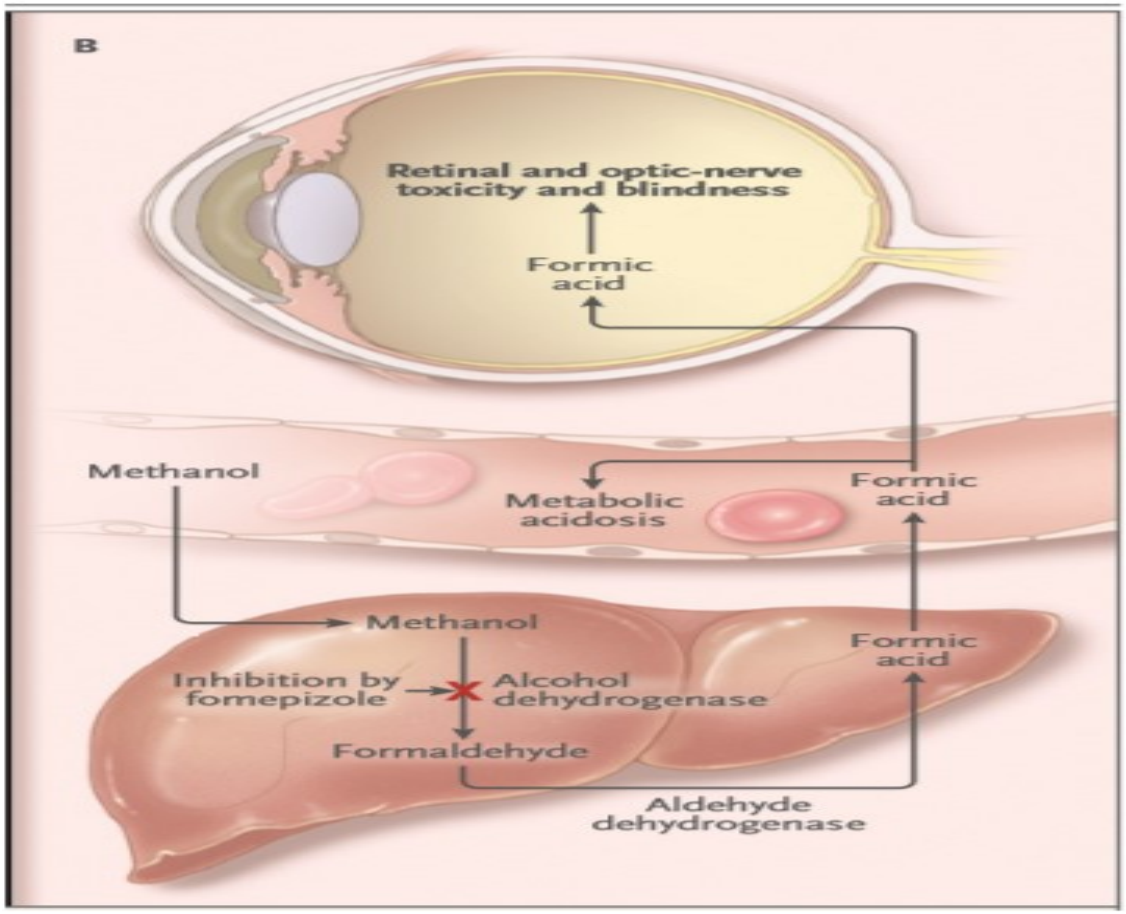
[a] Laboratory analysis by freezing point depression method only.

The NEW ENGLAND JOURNAL of MEDICINE

CLINICAL THERAPEUTICS

Fomepizole for Ethylene Glycol and Methanol Poisoning

Jeffrey Brent, M.D., Ph.D.



Intensive Care Med (2005) 31:189–195
DOI 10.1007/s00134-004-2521-0

REVIEW

Bruno Mégarbane
Stephen W. Borron
Frédéric J. Baud

Current recommendations for treatment of severe toxic alcohol poisonings

INDICATIONS OF RENAL REPLACEMENT THERAPY

Methanol poisoning:
Initial arterial pH <7.10[9] or 7.25–7.30 [16]
Drop in arterial pH >0.05 resulting in a pH outside the normal range despite bicarbonate infusion
Inability to maintain arterial pH >7.3 despite bicarbonate therapy
Decrease in bicarbonate concentration >5 mmol/l, despite bicarbonate therapy
Visual impairment
Renal failure
Deteriorating vital signs despite intensive supportive care
Initial plasma methanol concentration ≥ 0.5 g/l (15.6 mmol/l)[a]
Rate of methanol decline <0.1 g/l (3.1 mmol/l) per 24 h

[a] The recommendation for routine hemodialysis on the basis of serum concentrations alone has been recently called into question

Recommendations for the Role of Extracorporeal Treatments in the Management of Acute Methanol Poisoning: A Systematic Review and Consensus Statement

Darren M. Roberts, PhD, FRACP[1,2]; Christopher Yates, MD[3]; Bruno Megarbane, MD[4]; James F. Winchester, MD[5]; Robert Maclaren, PharmD[6]; Sophie Gosselin, MD[7]; Thomas D. Nolin, PharmD, PhD[8,9]; Valéry Lavergne, MD[10]; Robert S. Hoffman, MD[11]; Marc Ghannoum, MD[12]; on behalf of the Extracorporeal Treatments in Poisoning Workgroup

2015

TABLE 3. Role of Extracorporeal Treatment in the Treatment of a Patient With Methanol Poisoning

We recommend ECTR is initiated in the following circumstances:

1) Severe methanol poisoning (grade 1D), including any of:
   a) Coma (grade 1D)
   b) Seizures (grade 1D)
   c) New vision deficits (grade 1D)
   d) Metabolic acidosis from methanol poisoning
      i) Blood pH ≤ 7.15 (grade 1D)
      ii) Persistent metabolic acidosis despite adequate supportive measures and antidotes (grade 1D)
   e) Serum anion gap > 24 mmol/L (grade 1D); calculated by serum $[Na^+] - [Cl^-] - [Hco_3^-]$.

2) Serum methanol concentration
   a) > 700 mg/L or 21.8 mmol/L in the context of fomepizole therapy (grade 1D)
   b) > 600 mg/L or 18.7 mmol/L in the context of ethanol treatment (grade 1D)
   c) > 500 mg/L or 15.6 mmol/L in the absence of an ADH blocker (grade 1D)
   d) In the absence of a methanol concentration, the osmolal/osmolar gap may be informative (grade 1D)

3) In context of impaired kidney function (grade 1D)

To optimize the outcomes from ECTR, we recommend:

4) Intermittent hemodialysis is the modality of choice in methanol poisoning (grade 1D). Continuous modalities are acceptable alternatives if intermittent hemodialysis is not available (grade 1D).

5) ADH inhibitors are to be continued during ECTR for methanol poisoning (grade 1D) as well as folic acid

6) ECTR can be terminated when the methanol concentration is < 200 mg/L or 6.2 mmol/L and a clinical improvement is observed (grade 1D)

ECTR = extracorporeal treatment, ADH = alcohol dehydrogenase

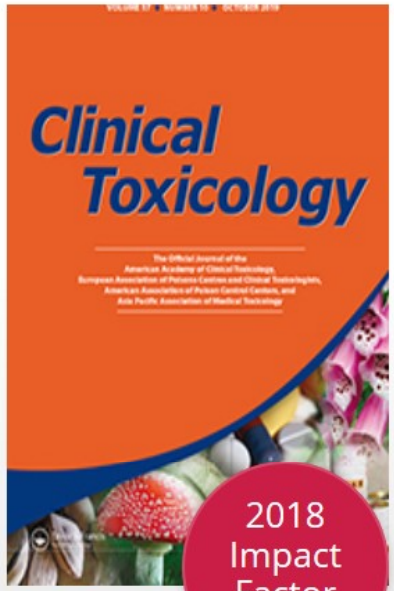
WHAT ARE
THE PROGNOSIS FACTORS ?

20 YEARS AGO…

- **Poor prognostic indicators include :**

  - **Serum formate concentrations >50mg/dL (>500mg/L).**
  - **pH < 7.0.**
  - **Coma Or Seizures on admission to the ED.**

Liu J.J. and col. Prognostic Factors in Patients with Methanol Poisoning. J. Toxicol. Clin. Toxicol. 1998.



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Methanol poisoning in Tunisia: Report of 16 cases

Nozha Brahmi M.D., Youssef Blel M.D., Nour Abidi M.D., Nadia Kouraichi M.D., Hafedh Thabet M.D., Abderrazek Hedhili & Mouldi Amamou M.D.

To cite this article: Nozha Brahmi M.D., Youssef Blel M.D., Nour Abidi M.D., Nadia Kouraichi M.D., Hafedh Thabet M.D., Abderrazek Hedhili & Mouldi Amamou M.D. (2007)

Table 1. Clinical features and laboratory chemistry at admission

Case	Sex/Age	SAPS II	APACHE II	Latent period (h)	Medical delay (h)	Symptoms	Ingested quantity (mL)	MeOH serum Level (g/L)	pH	HCO_3^- (mmol/L)	Outcome
1	M/16	14	8	8	26	Vertigo, headache, GI, VI	200	0.9	7.32	18	Survivor
2	M/17	12	8	6	36	Drowsiness, vertigo, GI	300	3.62	7.17	7	Survivor
3	M/24	12	9	4	6	Vertigo, VI, GI	500	3.56	7.14	7	Survivor
4	M/23	12	6	11	45	Headache, GI, VI	200	0.76	7.25	7.5	Survivor
5	M/29	84	36	–	36	Coma, BV, GI, shock	–	1.4	6.80	3.3	Died
6	M/20	14	8	7	12	Drowsiness, BV, GI	300	3.6	7.16	4	Survivor
7	M/20	81	35	–	30	Coma, BV, shock	–	2.5	6.99	6.9	Died
8	F/53	14	7	–	40	Vertigo, headache, VI	1000	1.3	7.42	22	Survivor
9	M/27	78	30	–	48	Coma, shock, BV, GI	1000	1.9	6.61	4	Died
10	M/23	11	6	–	36	Vertigo, blindness, chest tightness	300	1.9	7.19	15	Survivor
11	M/21	14	8	18	36	Nausea, dyschromatopsia	100	1.15	7.28	10	Survivor
12	M/17	12	8	–	36	None	70	0.24	7.34	15	Survivor
13	M/23	10	3	–	36	Vertigo, bradycrdia	30	3.56	7.14	7	Survivor
14	M/19	20	10	–	48	None	150	1.3	7.18	10.2	Survivor
15	M/21	12	7	18	36	Nausea, vomiting	80	0.19	7.35	16.8	Survivor
16	M/22	16	9	24	36	Nausea	100	1.6	7.26	10	Survivor

GI: gastrointestinal signs; VI: visual impairment; BV: blurred vision.

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Table 2. Risk factors of visual disturbances

Risk factors	Presence of visual disturbances (n = 11)	Absence of visual disturbances (n = 5)	P
Latent period (h)	9.2 ± 5.5	16.6 ± 8.08	0.04
Recovery time (h)	32 ± 13.5	36 ± 6.9	NS
Ingested quantity of methanol (g)	462 ± 350	105 ± 60	0.0001
GCS	11.2 ± 5.7	14.8 ± 0.4	NS
SBP	12.1 ± 4.1	12.5 ± 1.2	NS
pH	7.10 ± 0.2	7.30 ± 0.07	0.04
HCO_3^- (mmol/L)	8.7 ± 5.8	14 ± 3.7	NS
Methanol serum level (g/L)	2.2 ± 1.1	0.84 ± 0.62	0.01
Urea (mmol/L)	6.6 ± 3.7	5.45 ± 1.8	NS

GCS: Glasgow Coma Scale; SBP: systolic blood pressure.

Clinical Toxicology (2012), **Early Online:** 1–9

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DOI: 10.3109/15563650.2012.728224

informa
healthcare

RESEARCH ARTICLE

Risk factors related to poor outcome after methanol poisoning and the relation between outcome and antidotes – a multicenter study

RAIDO PAASMA,[1] KNUT ERIK HOVDA,[2] HOSSEIN HASSANIAN-MOGHADDAM,[3] NOZHA BRAHMI,[4] REZA AFSHARI,[5] LEIV SANDVIK[6,7] and DAG JACOBSEN[8]

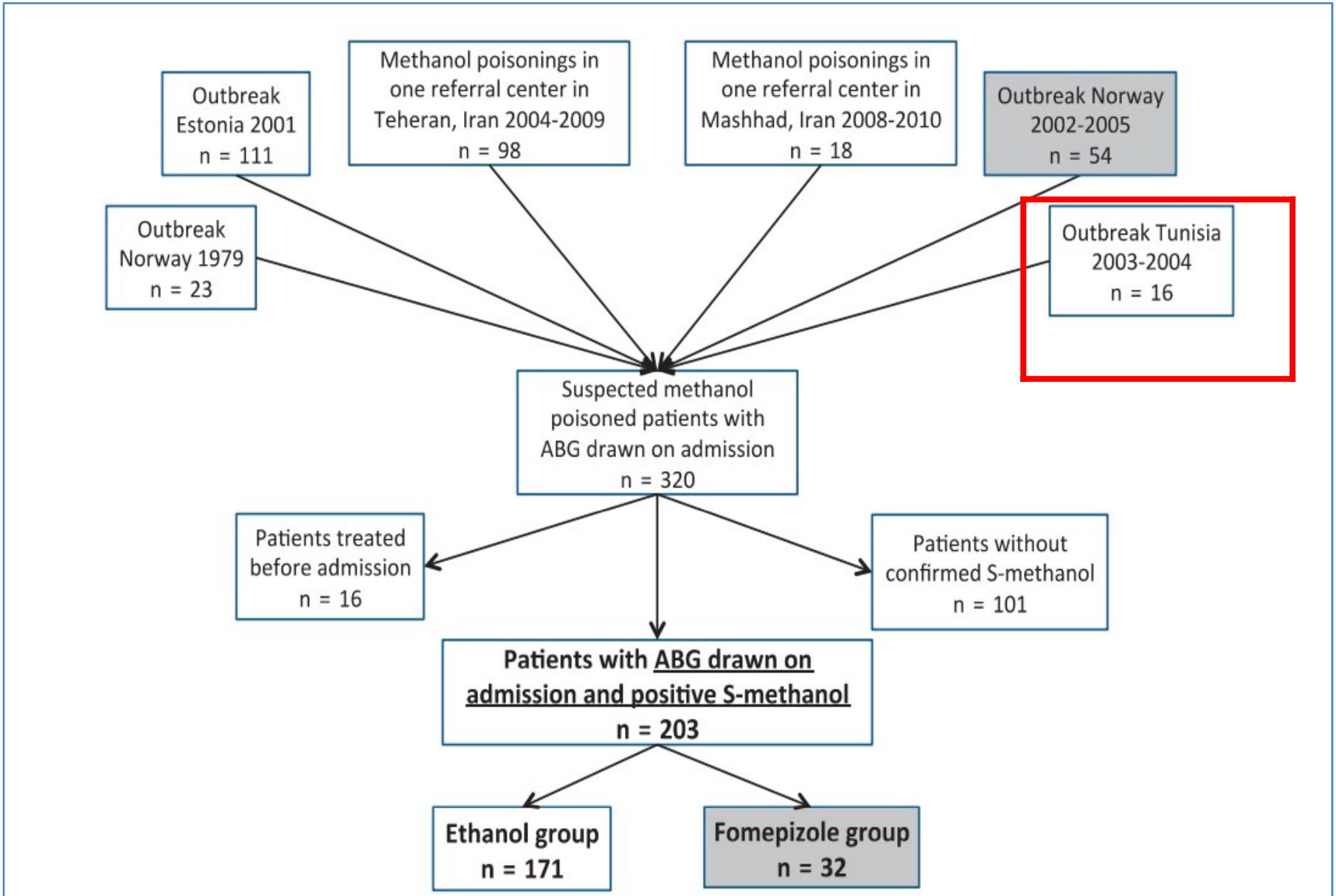
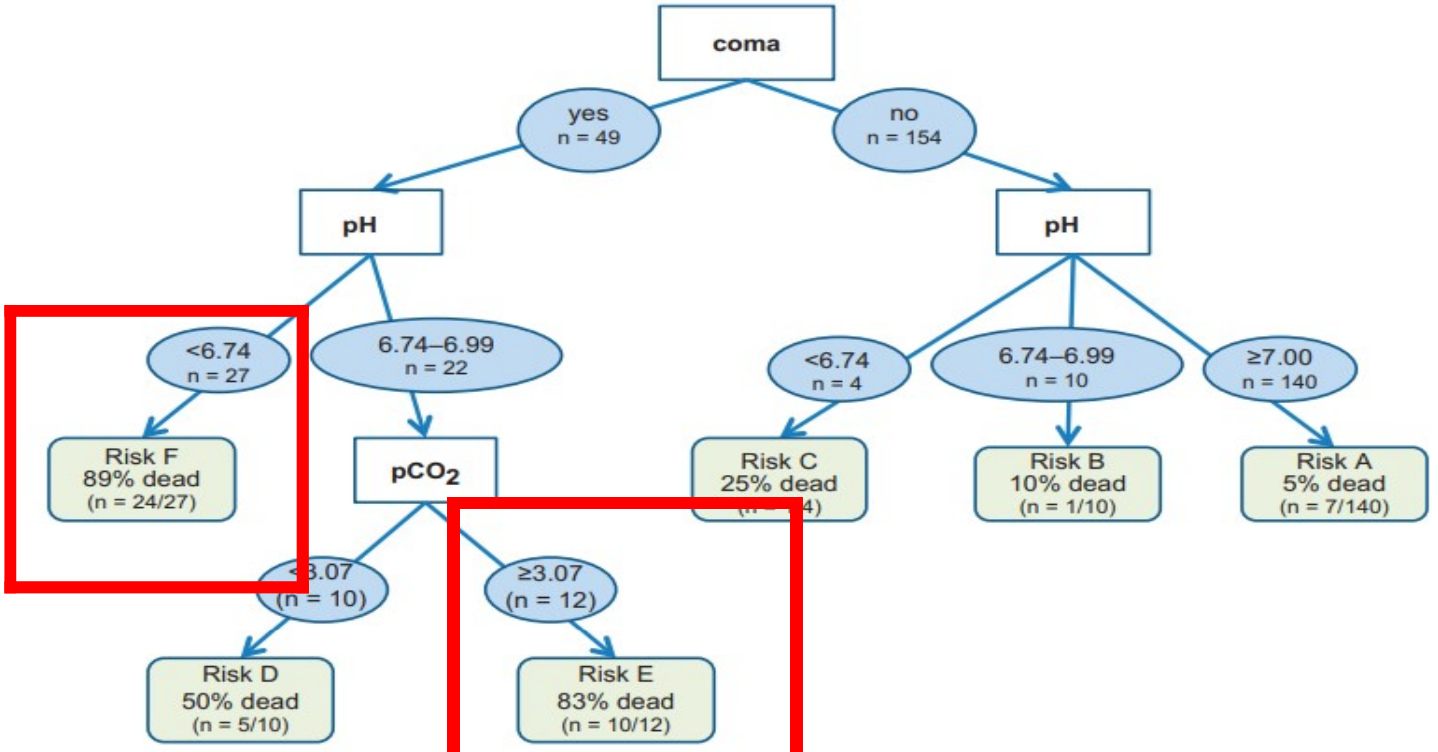


Fig. 1. Overview of the patients in the study. The patients treated with fomepizole and their origin is shaded.

Table 2. The results of the multivariate analysis on the factors associated with a poor outcome.

Independent variable	OR	95% confidence interval (CI)	p-value
Coma yes vs. no	10.2	3.3–32.0	$p < 0.001$
pH 0.1 unit increase	0.58	0.46–0.75	$p < 0.001$

Clinical interpretation: If the pH is increased by 0.1 units, the odds for poor outcome is reduced by 42%.



Risk group	Name on figure	Number in total	Dead in group	Total mortality risk	Odds ratio (95%CI)*
1	A and B	150/203	8/150	5%	1
2	C	4/203	1/4	25%	6 (1–64)
3	D	10/203	5/10	50%	18 (4–74)
4	E	12/203	10/12	83%	89 (17–475)
5	F	27/203	24/27	89%	142 (35–573)

*Odds ratio (OR) and 95% confidence intervals (CI) for death for all groups compared to risk group 1.

PLOS | ONE

RESEARCH ARTICLE

The Methanol Poisoning Outbreaks in Libya 2013 and Kenya 2014

Morten Rostrup[1,2,3], Jeffrey K. Edwards[4,5], Mohamed Abukalish[6], Masoud Ezzabi[7], David Some[4], Helga Ritter[4], Tom Menge[8], Ahmed Abdelrahman[9], Rebecca Rootwelt[1], Bart Janssens[10], Kyrre Lind[11], Raido Paasma[12], Knut Erik Hovda[11,13]*

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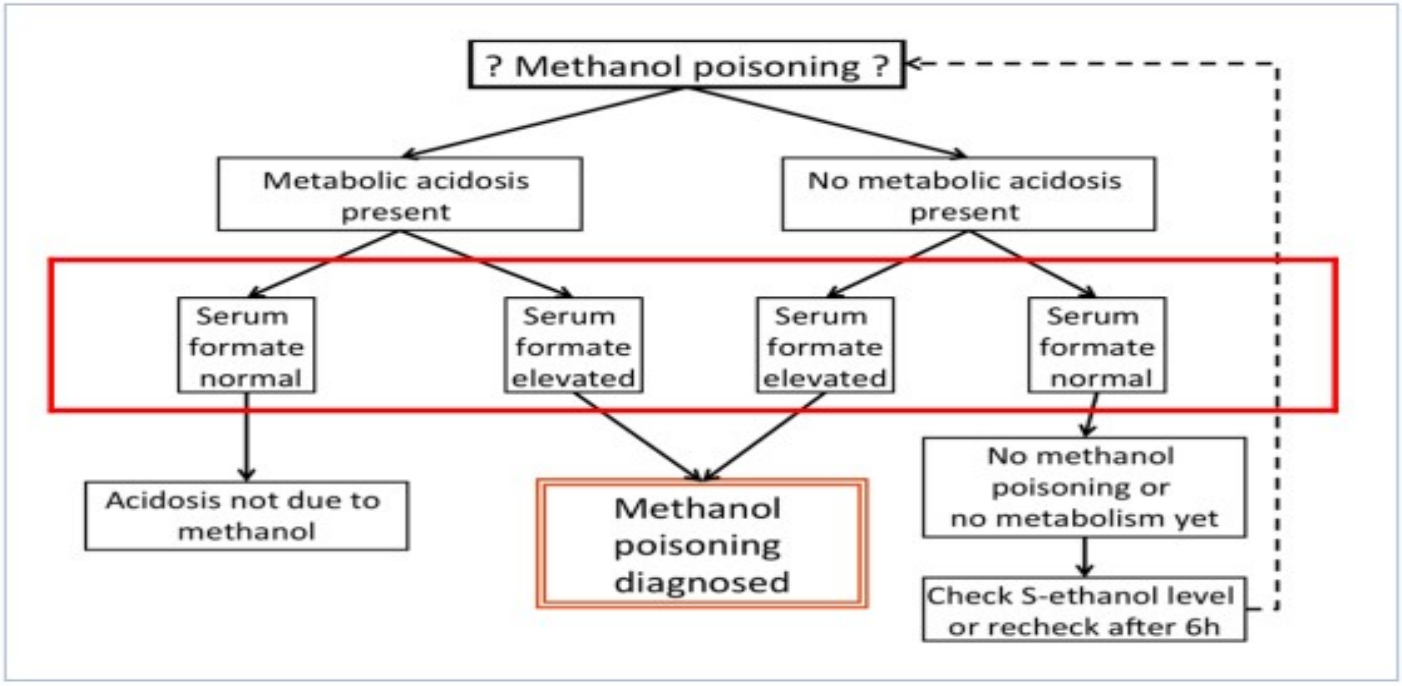
CrossMark
click for updates

Algorithm 2016…



ORIGINAL ARTICLE

ALCOHOLISM CLINICAL & EXPERIMENTAL RESEARCH

COVID-19 pandemic and methanol poisoning outbreak in Iranian children and adolescents: A data linkage study

Seyed Amirhosein Mahdavi[1] | Ali-Asghar Kolahi[2] |
Maryam Akhgari[1] | Farzad Gheshlaghi[3] | Narges Gholami[4] |

Archives of Toxicology (2020) 94:2259–2260
https://doi.org/10.1007/s00204-020-02795-2

LETTER TO THE EDITOR, NEWS AND VIEWS

Check for updates

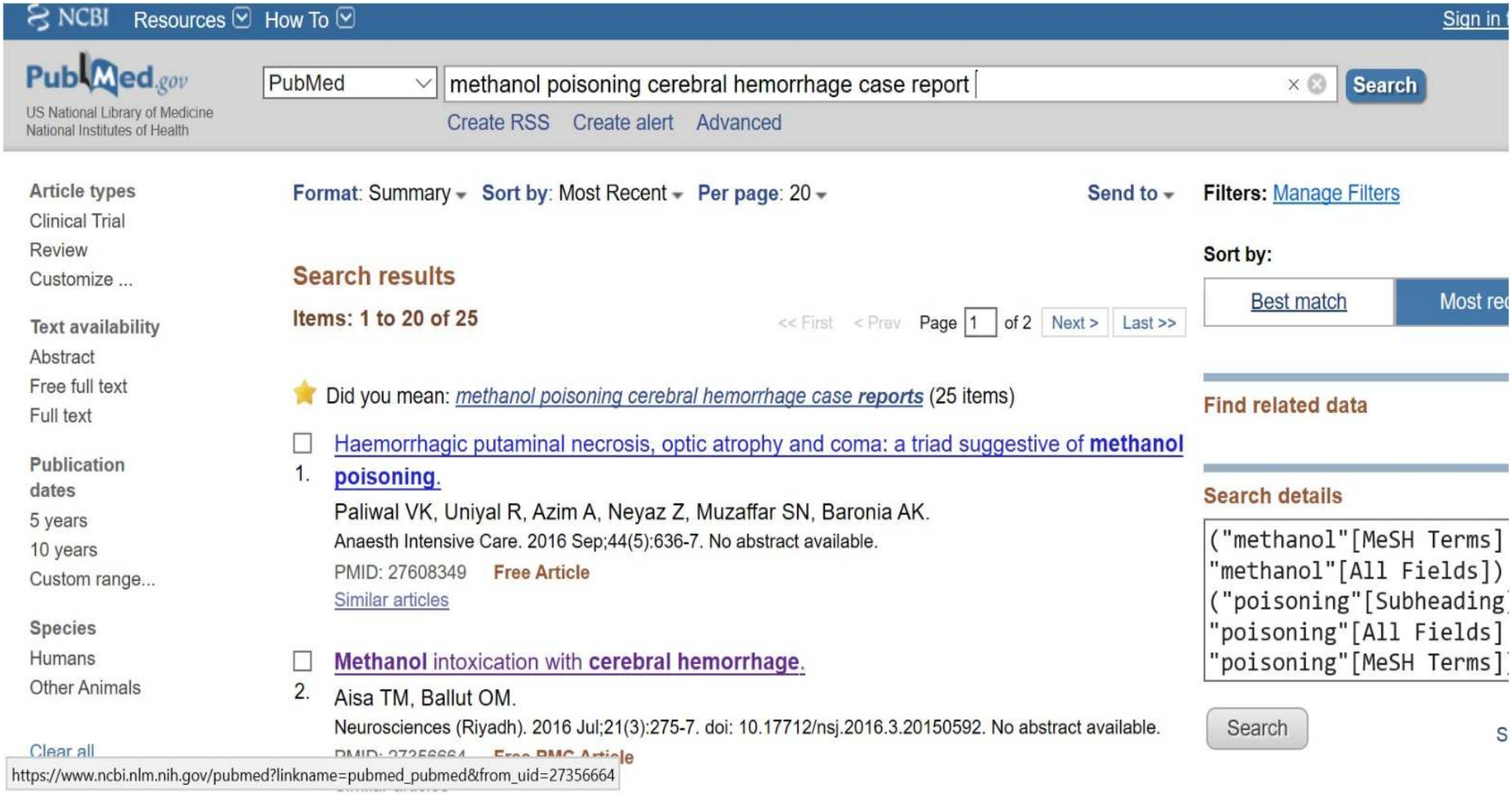
Toll of acute methanol poisoning for preventing COVID-19

Omid Mehrpour[1,2] · Mahmood Sadeghi[2]

Received: 24 April 2020 / Accepted: 20 May 2020 / Published online: 27 May 2020


مستشفى ابن الجزار
PITAL IBN JAZZAR

CASE REPORTS



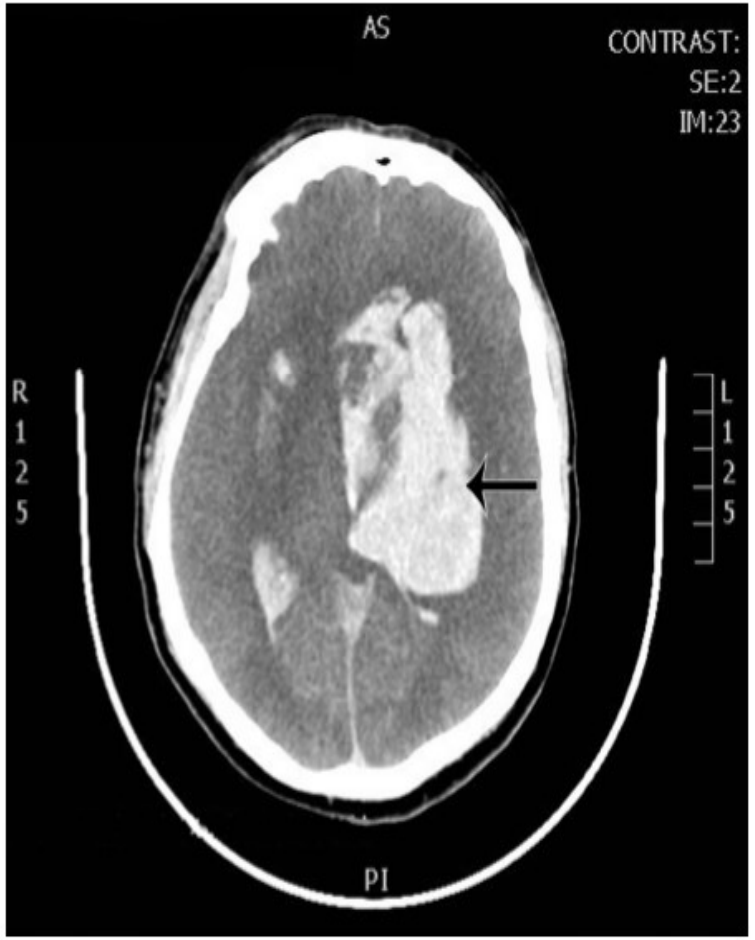
Methanol intoxication with cerebral hemorrhage

Tharwat M. Aisa, MD, EDIC,
Omar M. Ballut, MBBS, FRCP (Edin)

Neurosciences 2016

Cerebral Haemorrhage in methanol poisoning...

- The precise mechanism of necrosis and haemorrhage remains a matter of debate...
- It could be due to the direct toxic effect of methanol and its metabolite "formic acid" or secondary to anoxia and acidosis.
- Some authors suggested the mechanism of ischemia reperfusion injury causing haemorrhage in necrotic areas of the brain...?
- Heparinization during HD?



American Journal of
Emergency & Critical Care Medicine

Case Report

Severe Intracerebral Hemorrhage and Decompressive Craniectomy in a Case of Severe Methanol Intoxication

Nov 2020

Hassen Ben Ghezala[1*], Eslam E Abdelshafey[2], Mohammad Fawzy[3], Mohammad Ahmed Rashwan[3], Ibrahim Khalid Abdulal[3] and Ashraf Shamekh Al Tayar[4]

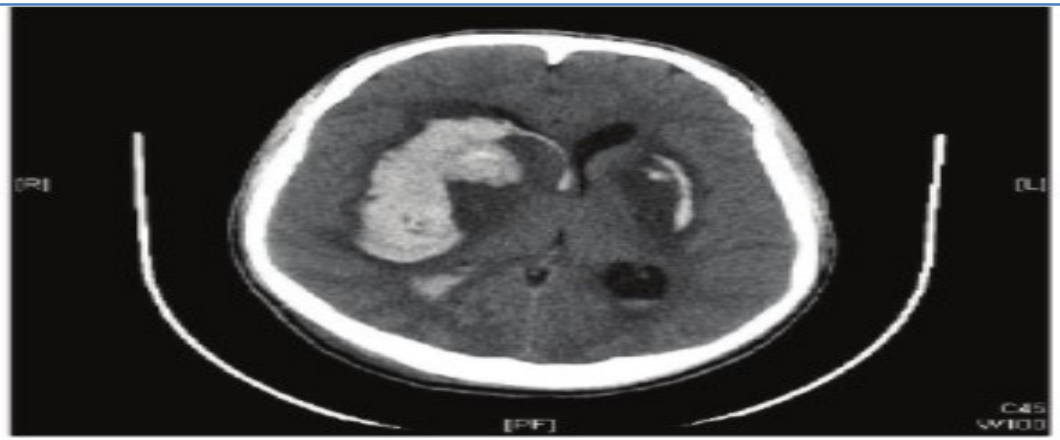


Figure 3: Follow up CT brain on day 4: Bilateral hemorrhagic transformation more on right side with mass effect.

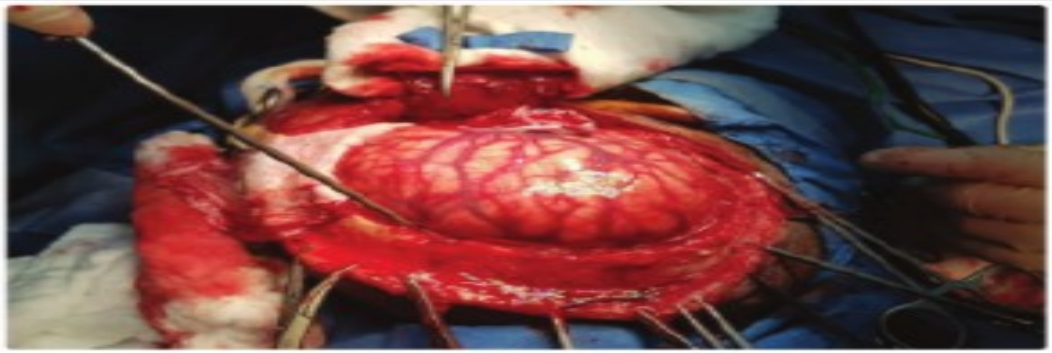


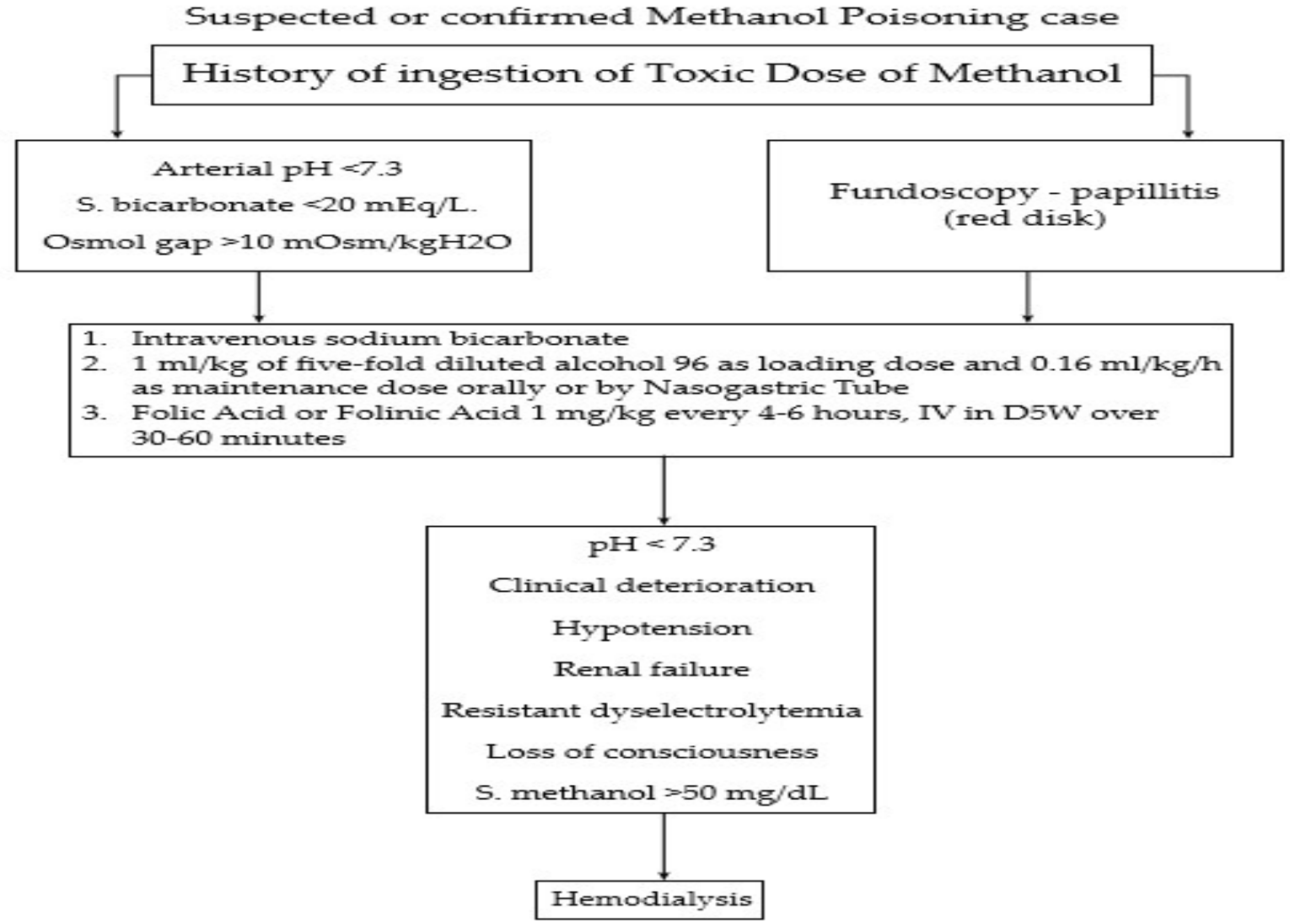
Figure 4: Per-operative intra-cerebral hematoma evacuation.

Journal of The Association of Physicians of India ▪ Vol. 66 ▪ April 2018

REVIEW ARTICLE

Methanol Poisoning

DB Kadam[1], Sonali Salvi[2], Ajay Chandanwale[3]



PREVENTION

- Public Education.
- Public/Government Health Problem..
- Control of false Information and rumors (COVID 19…)..
- Policies and Procedures….
- Labelling and Packaging (Article 17 European Chemical Agency Guidance).
- Legislative Control. ...Addiction..

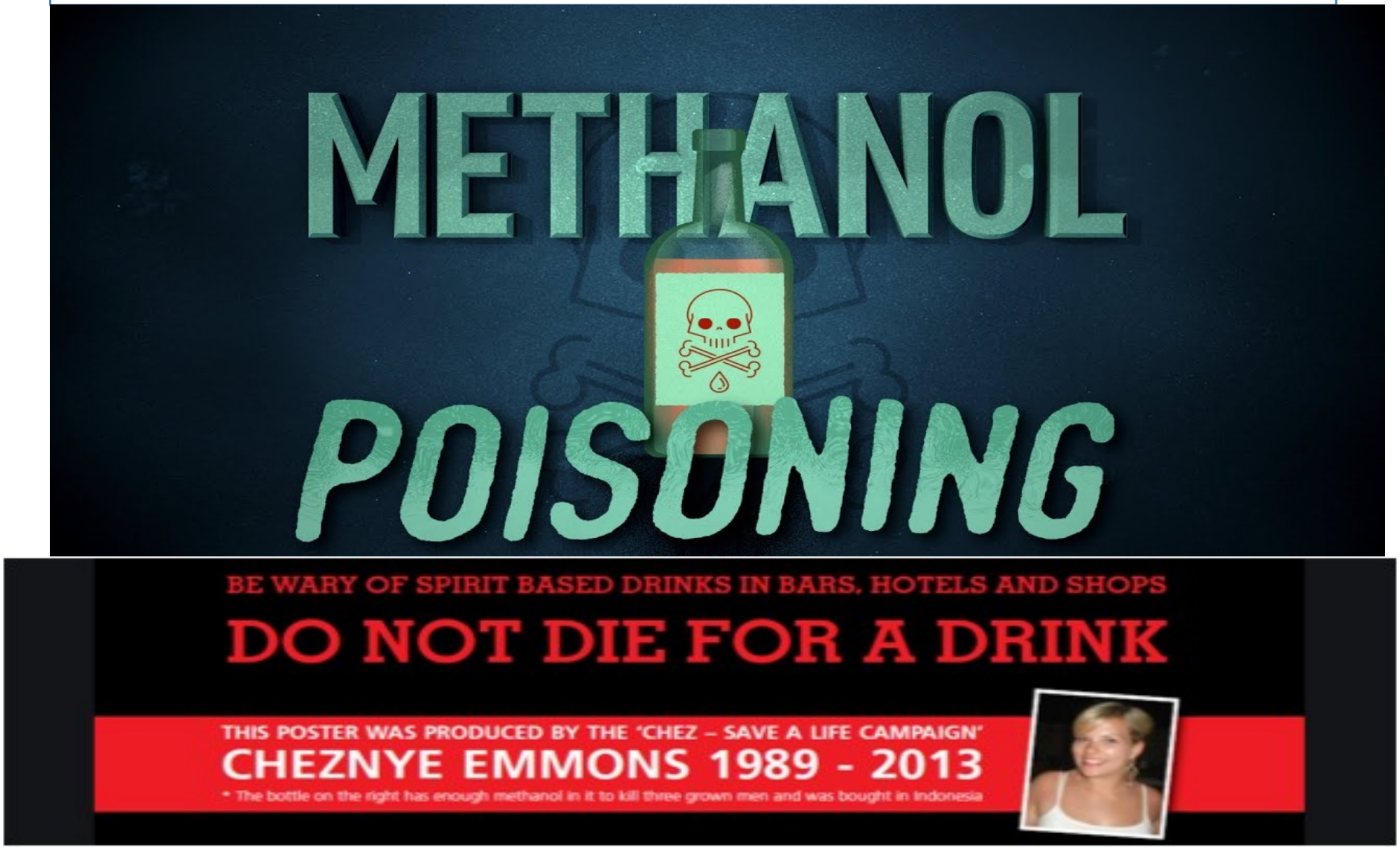
Loi 92-52 18/05/1992 relative aux stupefiants.

Take Home Messages

- ✓ *The silent METABOLIC killer.*
- ✓ *Fomepizole (Ethanol) to be included in the Hospital drug formulary among antidotes….*
- ✓ *Fomepizole Versus Ethanol? Prospective Study…*
- ✓ *Renal RRT and Formic acid? Versus Antidote?*
- ✓ ***Local Protocols.***
- ✓ *Public education about methanol consumption..*
- ✓ *Legislative control..*

METHANOL KILLS

DO NOT DIE FOR A DRINK.



Merci pour votre Attention