Paciente de 56 años con intoxicación accidental con gasolina – 2018

Dr. Leonardo Ramírez Zambrano

Compañeros de foro buenas tardes. ¿Alguno de Uds ha leído sobre Miocarditis provocada por consumo accidental de gasolina?

Tenemos un paciente de 56 años hipertenso controlado sin más y sin síntomas previos quien consumió accidentalmente alguna cantidad de gasolina e hizo una Neumonitis química de la cual se esta recuperando pero concomitantemente a su cuadro respiratorio hizo derrame pericárdico leve e hipocinesia lateral con FEVI 45% que no tenía hace 1 año. El ECG sólo muestra HVI sin más. Troponina I positiva y una discreta elevación de CKT y Mb.

Se recupera satisfactoriamente pues llegó en Insuficiencia Respiratoria.

Muchas gracias a todos.

Leonardo Ramírez Zambrano

OPINIONES DE COLEGAS

Querido Leonardo:

No tengo experiencia personal en este tipo de intoxicaciones.

En una primera búsqueda bibliográfica encuentro esto sobre complicaciones cardiológicas:

Sensibilización miocárdica a las catecolaminas, arritmias, miocarditis y muerte (más frecuente con H. aromáticos y halogenados).

En los inhaladores por la hipoxia (uso de la bolsa), el stress e hipocalemia (por el tolueno) se llega más rápidamente a fibrilación ventricular y paro cardíaco.

Aquí tienes el link al artículo original

http://www.mendoza.gov.ar/wp-content/uploads/sites/16/2014/10/Recomendaciones-Hidrocarburos-Destilados-Del-Petroleo.pdf

Un abrazo

Edgardo Schapachnik

Lo revisare esta misma noche Profesor. Muchas gracias. Saludos para todos.

Leonardo Ramírez Zambrano

Queridos amigos:

Si bien la gasolina tiene una composición química más compleja, realicé esta estrategia de búsqueda con TOLUENO
Search: ("Toluene/toxicity"[Mesh]) AND "Cardiovascular Diseases"[Mesh]

Los resultados son los siguientes; espero haya alguna cita que sea de utilidad

Un abrazo

Edgardo Schapachnik

PubMed Results
Items 1 - 8 of 8 (Display the 8 citations in PubMed)

1. Int J Mol Sci. 2016 Nov 17;17(11). pii: E1925.
3D Visualization of Developmental Toxicity of 2,4,6-Trinitrotoluene in Zebrafish Embryogenesis Using Light-Sheet Microscopy.

Eum J, Kwak J, Kim HJ, Ki S,Lee K, Raslan AA, Park OK, Chowdhury MA, Her S, Kee Y, Kwon SH, Hwang BJ.

Environmental contamination by trinitrotoluene is of global concern due to its widespread use in military ordnance and commercial explosives. Despite known long-term persistence ingroundwater and soil, the toxicological profile of trinitrotoluene and other explosive wastes have not been systematically measured using in vivo biological assays. Zebrafish embryos are idealmodel vertebrates for high-throughput toxicity screening and live in vivo imaging due to their small size and transparency during embryogenesis. Here, we used Single Plane Illumination Microscopy (SPIM) / light sheet microscopy to assess the developmental toxicity of explosive-contaminated water in zebrafish embryos and report 2,4,6-trinitrotoluene-associated developmental abnormalities, including defects in heart formation and circulation, in 3D. Levels of apoptotic cell death were higher in the actively developing tissues of trinitrotoluene-treated embryos thancontrols. Live 3D imaging of heart tube development at cellular resolution by light-sheet microscopy revealed trinitrotoluene-associated cardiac toxicity, including hypoplastic heart chamber formation and cardiac looping defects, while the real time PCR (polymerase chain reaction) quantitatively measured the molecular changes in the heart and blood development supporting the developmental defects at the molecular level. Identification of cellular toxicity in zebrafish using the state-of-the-art 3D imaging system could form the basis of a

sensitive biosensor for environmental contaminants and be further valued by combining it with molecular analysis.

PMCID: PMC5133921Free PMC Article
PMID: 27869673[Indexed for MEDLINE]

Conflict of interest statement
The authors declare no conflict of interest.

2. BMC Pharmacol Toxicol. 2013 Jan 24;14:8. doi: 10.1186/2050-6511-14-8.
Unusual case of severe arrhythmia developed after acute intoxication with tosylchloramide.

Lariccia V, Moraca A, Marini M, Nasti AA, Battistoni I, Amoroso S, Perna GP.

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Abstract

BACKGROUND:

Drugs not commonly considered to be cardioactive agents may cause prolongation of the QT interval with resultant torsades de pointes and ventricular fibrillation. This form of drug toxicity oftencauses cardiac arrest or sudden death.

CASE PRESENTATION:

After accidental ingestion of tosylchloramide a caucasian 77-year-old woman, with a family history of cardiovascular disease and hypertension, was admitted to the intensive care unit following episodes of torsades de pointes with a prolonged QT/QTc interval (640/542 ms). The patient received an implantable cardioverter-defibrillator, was discharged from the hospital with normal QT/QTc interval and did not experience additional ventricular arrhythmias during one year of follow-up.

CONCLUSION:

This is the first report concerning an unusual case of torsades de pointes after accidental intoxication by ingestion of tosylchloramide. The pronounced impact of the oxidyzing agent tosylchloramide on the activity of some of the ion channels regulating the QT interval was identified as a probable cause of the arrhythmia.

PMCID: PMC3566980Free PMC Article
PMID: 23347670[Indexed for MEDLINE]

3. Gig Sanit. 2005 Sep-Oct;(5):75-8.
[A combined effect of toluene and general vibration in a chronic toxicological experiment].

[Article in Russian]
Vlasov VN.
PMID: 16277004[Indexed for MEDLINE]

4. J Physiol Pharmacol. 2005 Sep;56 Suppl 4:215-21.
Effects of p53 inhibitor on survival and death of cells subjected to oxidative stress.

Strosznajder RP, Jesko H, Banasik M,Tanaka S.

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Abstract

Our previous data indicate that ischemia and amyloid beta peptide (A beta) cause an oxidative damage to macromolecules. In the present study we investigated the role of p53 protein in cell survival and death after administration of A beta. The experiments were carried out on pheochromocytoma cells (PC-12) and cortical primary neurons in culture. The cortical neurons wereexposed (48 h, 10 microM) to the action of a short A beta 25-35 neurotoxic fragment and the involvement of p53 was evaluated after addition of the p53 inhibitor pifithrin-alpha. Changes in cell morphology were evaluated by 4', 6-diamidino-2-phenylindole staining and the concentration-dependent effect of pifithrin-alpha on cells viability was determined. Additionally, we studied the effect of pifithrin-alpha on neuronal survival in vivo after a 5-min global brain ischemia followed by 7 days' reperfusion in gerbils. We found that A beta enhanced apoptotic cell death in cortical primary neurons. Pifithrin-alpha, at a 10 microM final concentration, protected the neuronal cells from the apoptotic death. However, at concentrations of 0.1 and 1 mM, the p53 inhibitor decreased PC-12 cells' viability in a dose-dependent manner. In in vivo experiments we did not observe any neuroprotection by pifithrin-alpha in the CA1 hippocampal layer, which suggests that its effects strongly depend on the duration and type of an ischemic insult. Our data indicate that pifithrin-alpha affects neuronal cells in a dual manner. It has a protective effect at a low concentration, but becomes neurotoxic at higher concentrations.

Free Article
PMID: 16204796[Indexed for MEDLINE]

5. Arch Toxicol. 2005 Dec;79(12):705-10. Epub 2005 Jul 16.
2,4,6-Trinitrotoluene inhibits endothelial nitric oxide synthase activity and elevates blood pressure in rats.

Sun Y, Iemitsu M, Shimojo N, Miyauchi T, Amamiya M, Sumi D, Hayashi T, Sun G, Shimojo N, Kumagai Y.

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Abstract

2,4,6-Trinitrotoluene (TNT), which is widely used in explosives, is an important occupational and environmental pollutant. Human exposure to TNT has been reported to be associated with cardiovascular dysfunction, but the mechanism is not well understood. In this study, we examine the endothelial nitric oxide synthase (eNOS) activity and blood pressure value following TNT exposure. With a crude enzyme preparation, we found that TNT inhibited the enzyme activity of eNOS in a concentration-dependent manner (IC50 value = 49.4 microM). With an intraperitoneal administration of TNT (10 and 30 mg/kg) to rats, systolic blood pressure was significantly elevated 1 h after TNT exposure (1.2- and 1.3-fold of that of the control, respectively). Under the conditions, however, experiments with the inducible NOS inhibitor aminoguanidine revealed that an adaptive response against hypertension caused by TNT occurs. These results suggest that TNT is an environmental chemical that acts as an uncoupler of constitutive NOS isozymes, resulting in decreased nitric oxide formation associated with hypertension in rats.

PMID: 16025313[Indexed for MEDLINE]

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PMID: 3402806[Indexed for MEDLINE]

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[Cardiotoxic effect of solvents].

[Article in German]
Elster I.
PMID: 4117980[Indexed for MEDLINE]

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[Article in Italian]
PENDINI A, ODESCALCHI CP.

PMID: 14485077[Indexed for MEDLINE]