

Current Awareness in Clinical Toxicology

Editors: Damian Ballam MSc and Allister Vale MD

March 2018

ANNOUNCEMENT

For more than 25 years, *Current Awareness in Clinical Toxicology* has been circulated monthly to staff of the UK National Poisons Information Service and, via the International Clinical Toxicological Societies [the American Academy of Clinical Toxicology, The European Association of Poisons Centres and Clinical Toxicologists and, more recently, to the Asia-Pacific Association of Medical Toxicology], to readers in Poisons Centres worldwide. Spontaneous and regular comments from readers have testified to the value of this monthly citation of the published literature.

The publication of *Current Awareness in Clinical Toxicology* has been made possible by the generous financial support of the UK Departments of Health, via Public Health England most recently. The decision has now been taken that this support cannot continue as there are greater financial priorities within the National Poisons Information Service. This issue of *Current Awareness in Clinical Toxicology* is therefore the last.

I would like to acknowledge the massive support of Sarah Cage and Damian Ballam in ensuring the timely publication of *Current Awareness in Clinical Toxicology* over three decades and to thank readers for their strong support.

If readers would like a digital version of the underlying archive to March 2018, which contains some 130,000 citations, please let me know. This can be offered either in Reference Manager™ or EndNote™ format.

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Drug-associated pulmonary arterial hypertension

McGee M, Whitehead N, Martin J, Collins N. Clin Toxicol 2018; online early: doi: 10.1080/15563650.2018.1447119:

Introduction

While pulmonary arterial hypertension remains an uncommon diagnosis, various therapeutic agents are recognized as important associations. These agents are typically categorized into "definite", "likely", "possible", or "unlikely" to cause pulmonary arterial hypertension, based on the strength of evidence.

Objective

This review will focus on those therapeutic agents where there is sufficient literature to adequately comment on the role of the agent in the pathogenesis of pulmonary arterial hypertension.

Methods

A systematic search was conducted using PubMed covering the period September 1970-2017. The search term utilized was "drug induced pulmonary hypertension". This resulted in the identification of 853 peer-reviewed articles including case reports. Each paper was then reviewed by the authors for its relevance. The majority of these papers (599) were excluded as they related to systemic hypertension, chronic obstructive pulmonary disease, human immunodeficiency virus, pulmonary fibrosis, alternate differential diagnosis, treatment, basic science, adverse effects of treatment, and pulmonary hypertension secondary to pulmonary embolism.

Agents affecting serotonin metabolism (and related anorexigens)

Anorexigens, such as aminorex, fenfluramine, benfluorex, phenylpropanolamine, and dexfenfluramine were the first class of medications recognized to cause pulmonary arterial hypertension. Although most of these medications have now been withdrawn worldwide, they remain important not only from a historical perspective, but because their impact on serotonin metabolism remains relevant. Selective serotonin reuptake inhibitors, tryptophan, and lithium, which affect serotonin metabolism, have also been implicated in the development of pulmonary arterial hypertension.

Interferon and related medications

Interferon alfa and sofosbuvir have been linked to the development of pulmonary arterial hypertension in patients with other risk factors, such as human immunodeficiency virus co-infection.

Antiviral therapies

Sofosbuvir has been associated with two cases of pulmonary artery hypertension in patients with multiple risk factors for its development. Its role in pathogenesis remains unclear.

Small molecule tyrosine kinase inhibitors

Small molecule tyrosine kinase inhibitors represent a relatively new class of medications. Of these dasatinib has the strongest evidence in drug-induced pulmonary arterial hypertension, considered a recognized cause. Nilotinib, ponatinib, carfilzomib, and ruxolitinib are newer agents, which paradoxically have been linked to both cause and treatment for pulmonary arterial hypertension.

Monoclonal antibodies and immune regulating medications

Several case reports have linked some monoclonal antibodies and immune modulating therapies to pulmonary arterial hypertension. There are no large series documenting an increased prevalence of pulmonary arterial hypertension complicating these agents; nonetheless, trastuzumab emtansine, rituximab, bevacizumab, cyclosporine, and leflunomide have all been implicated in case reports.

Opioids and substances of abuse

Buprenorphine and cocaine have been identified as potential causes of pulmonary arterial hypertension. The mechanism by which this occurs is unclear. Tramadol has been demonstrated to cause severe, transient, and reversible pulmonary hypertension.

Chemotherapeutic agents

Alkylating and alkylating-like agents, such as bleomycin, cyclophosphamide, and mitomycin have increased the risk of pulmonary veno-occlusive disease, which may be clinically indistinct from pulmonary arterial hypertension. Thalidomide and paclitaxel have also been implicated as potential causes.

Miscellaneous medications

Protamine appears to be able to cause acute, reversible pulmonary hypertension when bound to heparin. Amiodarone is also capable of causing pulmonary hypertension by way of recognized side effects.

Conclusions

Pulmonary arterial hypertension remains a rare diagnosis, with drug-induced causes even more uncommon, accounting for only 10.5% of cases in large registry series. Despite several agents being implicated in the development of PAH, the supportive evidence is typically limited, based on case series and observational data. Furthermore, even in the drugs with relatively strong associations, factors that predispose an individual to PAH have yet to be elucidated.

Full text available from: <https://doi.org/10.1080/15563650.2018.1447119>

Interventions for paracetamol (acetaminophen) overdose

Chiew AL, Glud C, Brok J, Buckley NA. Cochrane Database Syst Rev 2018; 2: CD003328.

Abstract and full text available from: <https://doi.org/10.1002/14651858.CD003328.pub3>

Acute carbon monoxide toxicity in a paediatric cohort: analysis of 10 boys poisoned during a scuba diving lesson

McDermott JH, Reynard C, Perry J, Dear JW, Child F, Jenner R. Clin Toxicol 2018; online early: doi: 10.1080/15563650.2018.1444175:

Background

Recent public health strategies have contributed towards a significant reduction in the

incidence of carbon monoxide (CO) poisonings. When events do occur, symptoms can vary dramatically depending on the carboxyhaemoglobin level and individual factors. Most reports to date focus on individual cases or larger retrospective reviews of diverse cohorts. There are very few reports of CO exposure related to scuba diving activities.

Methods

We describe the clinical sequelae experienced by 10 children who were exposed to CO during a scuba diving lesson. We collate patient data in the context of a severely affected individual and employ exponential decay calculations to estimate half-life.

Results

Six of the patients exposed to CO were symptomatic. The most severely affected individual suffered multi-organ effects, including myocardial damage, and required intensive care unit admission. The remaining cohort demonstrated notable clinical variability. The half-life of carboxyhaemoglobin on high flow oxygen in this cohort was 75 min, in line with previous estimates.

Conclusion

This work described an uncommon clinical presentation, representing the largest single cohort of its kind. This work exemplifies the variable symptomatology of CO toxicity, of which clinicians should be alert to if patients fall ill after scuba diving.

Full text available from: <https://doi.org/10.1080/15563650.2018.1444175>

Alterations in mitochondrial respiration and reactive oxygen species in patients poisoned with carbon monoxide treated with hyperbaric oxygen

Jang DH, Khatri UG, Shortal BP, Kelly M, Hardy K, Lambert DS, Eckmann DM. Intensive Care Med Exp 2018; 6: 4.

Abstract and full text available from: <https://doi.org/10.1186/s40635-018-0169-2>

High dose insulin for beta-blocker and calcium channel-blocker poisoning: 17 years of experience from a single poison center

Cole JB, Arens AM, Laes JR, Klein LR, Bangh SA, Olives TD. Am J Emerg Med 2018; online early: doi: 10.1016/j.ajem.2018.02.004:

Abstract and full text available from: <https://doi.org/10.1016/j.ajem.2018.02.004>

Risks linked to accidental inoculation of humans with veterinary vaccines: a 7-year prospective study.

Meyer G, Jegou F, Hamel J-F, Boels D. Clin Toxicol 2018; online early: doi: 10.1080/15563650.2018.1445261:

Aim

Accidental inoculation of humans with veterinary vaccines can lead to early and late complications. The aim of our study is to describe these complications and their risk factors.

Methods

Prospective observational study conducted from 2007 to 2014 at Angers University Hospital's Poison Control Centre. The endpoints examined were: early and late locoregional

complications, surgical treatment, and absence from work. The statistical analysis was based on a multivariate analysis.

Discussion

The presence of mineral oil adjuvants, the injection of the vaccine under pressure and injection in joint and tendon of the hand significantly increased early locoregional complications and surgery but only the presence of mineral oil adjuvant increased significantly late locoregional complications at one month. Absence from work is significantly correlated to the site of injection and the presence of mineral oil adjuvant.

Conclusion

It is important to know about the contents of the veterinary vaccine in order to anticipate early and late complications that may arise (particularly due to the presence of mineral oil adjuvants). Special attention must also be given do the site of injection. We think that any accidental injection of veterinary vaccine into humans, especially those containing mineral oils, must lead to an early medical consultation. This must also be indicated on the product

Full text available from: <https://doi.org/10.1080/15563650.2018.1445261>

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