Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPERS OF THE MONTH

Significance of the imidazoline receptors in toxicology

Lowry JA, Brown JT. Clin Toxicol 2014; online early:
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Introduction

The alpha-2 adrenergic (AA-2) receptor agonists and imidazolines are common exposures in the American Association of Poison Control Centers (AAPCC) National Poison Data System (NPDS). Although the interaction between the AA-2 receptor and imidazoline receptors has been extensively studied, it largely remains unknown to health-care professionals. This review describes these interactions and mechanisms by which agonists affect physiologic responses binding to these receptors.

Methods

Papers published in English from 1960 to 2013 were retrieved from PubMed. A total of 323 original articles were identified and 173 were included.

Background

The toxicity associated with clonidine (e.g., bradycardia, miosis, and hypotension) is largely assumed to be secondary to the functional overlap of the AA-2 receptors and the mu receptors. However, the effects at the AA-2 receptor could not fully account for these symptoms. Subsequently, clonidine was found to produce its pharmacologic effect in the central nervous system (CNS) by interaction not only with the AA-2 receptor but also on selective imidazoline receptors.

Imidazoline receptors

Since their discovery, three distinct classes of imidazoline receptors, also known as imidazoline binding sites or imidazoline/guanidinium receptive sites, have been characterized. Imidazoline-1 (I-1) receptors are involved in the hypotensive activity of clonidine and related compounds supporting the idea that the I-1 receptors are upstream from the AA-2 receptor and work in tandem for its effect on blood pressure. Additionally, stimulation of N-type Calcium-2 channels, G-protein inwardly rectifying potassium channel, adenosine receptors, phosphatidyl-choline-specific phospholipase C, and nicotinic receptors have been implicated to be involved. Previous studies have shown that I-1 receptors may also be involved in other physiologic responses beyond cardiac function. Imidazoline-2 (I-2)

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receptors interact with monoamine oxidase A and monoamine oxidase B leading to research that has focused on the effect of I-2 receptors and depression and the suggestion of a possible antidepressant action of the imidazolines. I-2 receptor ligands may have substantial antinociceptive activity and work synergistically with opioids in acute pain. Imidazoline-3 (I-3) receptors are located on the pancreatic β-cells and modulate glucose homeostasis.

Imidazoline ligands

Four endogenous compounds have been found to bind and include clonidine-displacing substance, agmatine, harmane, and imidazole acetic acid. Significant interest in developing new agents with higher selectivity and affinity for I-1 receptors has resulted.

Toxicology

Alpha-2 adrenoceptor and imidazoline receptor agonists such as clonidine and tetrahydrozoline are common ingestions reported to poison control centers. The most common toxic effects of clonidine are similar to those of the over-the-counter imidazolines and include CNS depression, bradycardia, hypotension, respiratory depression, miosis, hypothermia, and hypertension (early and transient). Based on their structure and subsequent studies, imidazoline receptors seem to be the primary binding site for these chemicals. Case reports typically illustrate rapid onset of action with serious side effects following ingestion of relatively small amounts. These agents have been reportedly used in drug-assisted sexual assaults.

Conclusion

Much of the toxicity associated with drugs such as clonidine, guanfacine, and tetrahydrozoline are due to their binding to imidazoline receptors. Knowledge of the imidazoline receptors may lead to new therapeutic agents and inform management of patients with imidazoline overdose.

Full text available from: http://dx.doi.org/10.3109/15563650.2014.898770

Neurotoxicity of European viperids in Italy: Pavia Poison Control Centre case series 2001–2011

Lonati D, Giampreti A, Rossetto O, Petrolini VM, Vecchio S, Buscaglia E, Mazzoleni M, Chiara F, Aloise M, Gentilli A, Montecucco C, Coccini T, Locatelli CA. Clin Toxicol 2014; online early:

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Context

Some clinical aspects about neurotoxicity after snakebites by European viper species remain to be elucidated.

Objective

This observational case series aims to analyze neurological manifestations due to viper envenomation in Italy in order to describe the characteristic of neurotoxicity and to evaluate the clinical response to the antidotic treatment, the outcome, and the influence of individual variability in determining the appearance of neurotoxic effects. Materials and methods. All cases of snakebite referred to Pavia Poison Centre (PPC) presenting peripheral neurotoxic effects from 2001 to 2011 were included. Cases were assessed for time from bite to PPC evaluation, Grade Severity Score (GSS), onset/duration of clinical manifestations, severity/time course of local, non-neurological and neurological effects, and antidotic treatment.

Results

Twenty-four were included (age, 3-75 years) and represented on average of 2.2 cases/year

(about 5% of total envenomed patients). The mean interval time of PPC evaluation from snakebite was 10.80 ± 19.93 hours. GSS at ED-admission was 0 (1 case), 1 (10 cases), and 2 (13 cases). All patients showed local signs: 41.6%, minor; 58.4%, extensive swelling and necrosis. The main systemic non-neurological effects were as follows: vomiting (86.7%), diarrhea (66.7%), abdominal discomfort (53.3%), and hypotension (20%). Neurotoxic effects were accommodation troubles and diplopia (100%), ptosis (91.7%), ophtalmoplegia (58.3%), dysphagia (20.8%), drowsiness (16.6%), cranial muscle weakness (12.5%), and dyspnea (4.2%). Neurotoxicity was the unique systemic manifestation in 9 cases; in 4 cases, they were associated with only mild local swelling. In 10 patients the onset of neurotoxic effects followed the resolution of systemic non-neurological effects. Antidote was intravenously administered in 19 (79.2%) patients. The mean duration of manifestations in untreated versus treated groups was 53.5 ± 62.91 versus 41.75 ± 21.18 hours (p = 0.68, local effects) and 9.77 ± 3.29 versus 8.25 ± 12.23 hours (p = 0.1, systemic non-neurological effects) and 43.4 ± 14.69 versus 26.58 ± 20.62 hours (p = 0.03, neurotoxic effects).

Conclusions

Neurotoxicity may appear late (11 hours after the bite in 58.3% of cases), in contrast with the data reported in medical literature. Neurotoxic effects have been reversible in all cases and may be the unique systemic manifestation of envenomation. Neurotoxic effects are shorter in treated group. The antidotic treatment of patients considered as GSS 2 only for neurotoxic effects (with mild local effects) may not be necessary. Variable factors such as different amount of venom injected, concentration of PLA2 component, and individual susceptibility may explain the less percentage of patients presenting neurotoxic effects.

Full text available from: http://dx.doi.org/10.3109/15563650.2014.904046

Does diisocyanate exposure result in neurotoxicity?

Hughes MA, Carson M, Collins MA, Jolly AT, Molenaar DM, Steffens W, Swaen GMH. Clin Toxicol 2014; online early:

doi: 10.3109/15563650.2014.898769:

Context

Diisocyanates have been associated with respiratory and dermal sensitization. Limited number of case reports, and a few case studies, media, and other references suggest potential neurotoxic effects from exposures to toluene diisocyanate (TDI), 1,6 hexamethylene diisocyanate (HDI), and methylene diisocyanate (MDI). However, a systematic review of the literature evaluating the causal association on humans does not exist to support this alleged association.

Objective

To perform systematic review examining the body of epidemiologic evidence and provide assessment of causal association based on principles of the Sir Austin Bradford Hill criteria or considerations for causal analysis.

Methods

A comprehensive search of public databases for published abstracts, case reports, cross-sectional surveys, and cohort studies using key search terms was conducted. Additional searches included regulatory reviews, EU IUCLID and EU Risk Assessment databases, and unpublished reports in the International Isocyanate Institute database. An expert panel consisting of physicians, toxicologists, and an epidemiologist critically reviewed accepted papers, providing examination of epidemiologic evidence of each report. Finally, the Hill criteria for causation were applied to the summative analysis of identified reports to

estimate probability of causal association.

Results

Twelve papers reporting exposed populations with a variety of neurological symptoms or findings suitable for analysis were identified, including eleven case or case series reports, and one cross-sectional study. Three papers reported on the same population. Each of the papers was limited by paucity of diisocyanate exposure estimates, the presence of confounding exposures to known or suspected neurotoxicants, a lack of objective biological measures of exposure or neurotoxic effects, and lack of relative strength of association measures. Additionally, reported health symptoms and syndromes lacked consistency or specificity. No plausible mechanism of toxicity was found. Application of a predictive mathematical model for determining probability of causal association for neurotoxicity was calculated to be 21%.

Conclusion

There is insufficient evidence for a causal association of neurotoxic effects and diisocyanate exposure based on lack of evidence in all categories of the Hill criteria for causality except for temporal association of reported symptoms and alleged exposure. Future reports should attempt to address more rigorous exposure assessment and control for confounding exposures.

Full text available from: http://dx.doi.org/10.3109/15563650.2014.898769

Four-hour acetaminophen concentration estimation after ingested dose based on pharmacokinetic models

Villeneuve E, Gosselin S, Whyte I. Clin Toxicol 2014; online early: doi: 10.3109/15563650.2014.902956:

Introduction

The United Kingdom has recently changed the indications for N-acetylcysteine treatment for acetaminophen intoxication. Any ingestion over 75 mg/kg is now referred to the hospital. A model based on pharmacokinetic parameters was developed to predict 4-h acetaminophen concentration for this and other ingested doses.

Methodology

EMBASE and Medline were searched to obtain values for volume of distribution, absorption, and elimination constants and bioavailability for acetaminophen. Four-hour concentrations were calculated for ingestion doses currently recommended for hospital referral in different countries. Calculated plasma concentrations at 4 h for several doses were plotted against the Rumack-Matthew and the United Kingdom treatment lines.

Results

Six articles were used for the calculations (4 adult and 2 pediatric). In order to achieve a 4-h acetaminophen concentration of 100 mg/L, doses (mg/kg ± 99.9CI) of 180.5 ± 43.2 for adults and 396.1 ± 115.5 for children were calculated.

Discussion

A dose of 75 mg/kg would likely yield a 4-h acetaminophen concentrations well below 100 mg/L. Medical toxicologists and poison information specialists are left without evidence-based guidance for which patients or which ingestion history would now warrant referral to hospital for acetaminophen concentration measurement. Larger toxicokinetic studies in acetaminophen overdose are needed to define ingestion dose for referral to hospital.

Full text available from: http://dx.doi.org/10.3109/15563650.2014.902956

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