

RGH Pharmacy E-Bulletin

Volume 35 (4): August 17, 2009

A joint initiative of the Patient Services Section and the Drug and Therapeutics Information Service of the Pharmacy Department, Repatriation General Hospital, Daw Park, South Australia. The RGH Pharmacy E-Bulletin is distributed in electronic format on a weekly basis, and aims to present concise, factual information on issues of current interest in therapeutics, drug safety and cost-effective use of medications.

Editor: Assoc. Prof. Chris Alderman, University of South Australia – Director of Pharmacy, RGH

© Pharmacy Department, Repatriation General Hospital, Daw Park, South Australia 5041

Drug-induced acute pancreatitis

Drug-induced acute pancreatitis (AP) is rare and the incidence ranges from 0.3% to 2% of AP cases. Most of the literature available consists of anecdotal evidence and case reports. The causes of AP include gallstones, alcohol, hypertriglyceridemia, hypercalcaemia, infection, trauma, idiopathic, genetic mutations, pancreatic tumours, and drugs. Drug-induced AP is implicated after excluding the common causes of AP, and when pancreatitis resolves after stopping the offending drug or recurs after re-challenge.

The cause of drug-induced pancreatic injury is unknown. Proposed mechanisms include metabolic effects, direct cellular toxicity, pancreatic duct constriction and an immune-mediated response. Pancreatitis may occur within a few weeks of starting a drug or after many months of drug therapy depending on the medication involved.

In a review article by Badalov et al there were 120 drugs classified as potentially causing pancreatitis. Agents were classified as either;

- class Ia (at least one case report with positive rechallenge excluding all other causes such as gallstones, alcohol, hypertriglyceridaemia, hypercalcaemia and other drugs)
- class Ib (at least one case report with positive rechallenge but other causes not ruled out)
- class II drugs (at least 4 cases with consistent latency ie time interval between starting drug and onset of AP in at least 75% of cases)
- class III (at least 2 cases, no consistent latency among cases and no rechallenge)
- class IV (not fitting into earlier classes, single case report without rechallenge).

Only class Ia, Ib and II drugs are listed below .

Class Ia		Class Ib		Class II
Methyl dopa	Olsalazine	All-trans retinoic acid	Norethindronate/ mestranol	Paracetamol
Bezafibrate	Pentamidine	Azathioprine	Omeprazole	Chlorthiazide
Cannabis	Pravastatin	Clomiphene	Sulfamethoxazole	Clozapine
Carbimazole	Procainamide	Dexamethasone	Trimethoprim- sulfamethazole	Didanosine
Codeine	Pyritinol	Ifosfamide		Erythromycin
Cytosine	Simvastatin	Lamivudine		Estrogen
Arabinoside	Stibogluconate	Losartan		L-asparaginase
Dapsone	Sulfamethoxazole	Lynesterol/ Methoxyethinylestradiol		Pegasparagase
Enalapril	Sulindac	6-Mercaptopurine		Propofol
Frusemide	Tetracycline	Meglumine		Tamoxifen
Isoniazid	Valproic acid	Methimazole		
Mesalazine		Nelfinavir		
Metronidazole				

Drugs associated with AP can be difficult to identify, but if a drug is highly suspected as a causative agent, it is recommended to hold therapy until further investigations are made. If patients need to restart therapy they should be carefully monitored, and the suspect medication discontinued if symptoms of AP recur.

Acknowledgment – This E-Bulletin is based on work by Irene Heng, Clinical Pharmacist, RGH.

FOR FURTHER INFORMATION – CONTACT THE PHARMACY DEPARTMENT ON 82751763 or email: chris.alderman@rgh.sa.gov.au
Information in this E-Bulletin is derived from critical analysis of available evidence – individual clinical circumstances should be considered when making treatment decisions. You are welcome to forward this E-bulletin by email to others you might feel would be interested, or to print the E-Bulletin for wider distribution. Reproduction of this material is permissible for purposes of individual study or research.

