

# Jacobs Journal of Gastroenterology and Hepatology

Letter to Editor

## Drug-induced Acute Pancreatitis – ‘Scope for Secondary Prevention’

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Received: 10-07-2016

Accepted: 10-16-2016

Published: 10-20-2016

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I read with interest the article by Chang et al. [1] in which the authors adopt a systematic approach (including taking into account binary clinical conditions such as the presence of hypertriglyceridemia, alcohol and / or tobacco use, gallstone, obesity and bilio-pancreatic cancers, estimating dose response effects and performing sensitivity analyses) to tease out the relationship of various anti-diabetic drugs and the risk of Acute pancreatitis (AP). In the end, they provide compelling evidence that sulfonylureas carry a more significant risk of causing AP compared to metformin which in turn carry a more significant risk that dipeptidyl peptidase-4.

The study [1] reaffirms the role played by drugs in the causation of AP. It is well appreciated that diabetic patients are at an increased risk of developing AP and are more likely to suffer the severe form of the disease [2]. Hyperglycaemia, coupled with the factors influencing insulin resistance (tumour necrosis- $\alpha$ , NF $\kappa$ B, amylin) which cause an increase in reactive oxygen species generation in acinar cells, have been hypothesized to play a role in the pathophysiology of AP in patient with diabetes mellitus [3].

While literature is abound with case reports, case series and even reviews on drug-induced AP there continue to be doubts raised as to the extent to which drugs actually play in causing an episode of AP [4]. Such arguments are very valid as reports on drug-induced AP may result from an incomplete aetiological work-up. The more concerning offshoot of presumptuously arriving at such a diagnosis, though, is the risk that in the haste to label a patient with drug-induced AP, a more sinister cause of AP (eg. a tumour) may be over-

looked.

Nevertheless, we must acknowledge that AP is a universal health problem [5] contributing majorly to health care expenditure [6]. What is more important is that AP often runs an unpredictable course once the wheels of the disease have been set into motion and, to this day, the treatment is only supportive in the absence of a specific therapy [7]. And while at the present time we may be unable to explain how or why some patients react differently to their prescription medications and develop an episode of AP while a million other patients on the same drug remain unaffected, clinicians must remember that drug-induced AP does exist [8] and possibly represents one of the few instances in AP where an astute clinician can make a world of difference to the patient's life. Patients with drug-induced AP generally have a mild disease in the initial presentation [8]. Thus, all it takes is for the clinician, be it the general practitioner, the endocrinologist, the emergency room doctor or the gastroenterologist to recognise the possibility of one of the prescription medications [9] being a potential cause for the episode of AP (after ruling out every other cause [10]), and to effect a simple switch in therapy to a drug with less potential for causing AP [9]. Studies such as the one Chang et al. [1] should be encouraged as they present clinicians with exactly this information. There are very few instances in AP wherein the clinician may have the luxury for secondary prevention of the disease. Performing a cholecystectomy following an episode of mild biliary AP is one; whilst recognising and appropriately managing a patient with drug-induced AP represents the other.

**Commentary on the manuscript:** Chang HY, Hsieh CF, Singh S, Tang W, Chiang YT, Huang WF. Anti-diabetic therapies and the risk of acute pancreatitis: a nationwide retrospective cohort study from Taiwan. *Pharmacoepidemiol Drug Saf.* 2015

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