Austin Pancreatic Disorders

Open Access

Austin Publishing Group

Short Communication

Smoking and Acute Pancreatitis - It is about Time we Extinguish the Cigarette

Barreto SG*
Hepatobiliary and Oesophagogastric Unit, Division of Surgery and Perioperative Medicine, Australia

***Corresponding author:** Savio George Barreto, Hepatobiliary and Oesophagogastric Unit, Division of Surgery and Perioperative Medicine, Australia

Received: January 27, 2017; **Accepted:** February 03, 2017; **Published:** February 06, 2017

Keywords

Disease; Pancreas; Inflammation; Public health

Short Communication

Alcohol and gallstones are the better known causes of acute pancreatitis (AP) the world over [1-3]. However, recent studies have unearthed a likely association between smoking and AP [4-7].

In an attempt to tease out the exact relationship between the two, I reviewed the evidence linking smoking, more specifically, the constituents of cigarette smoke, and their potential to trigger an attack of AP [8]. This led to the appreciation that the two main metabolites of cigarette smoke, nicotine and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), are capable of inducing functional and histological changes within the pancreas consistent with AP. The pathways involved in the pathogenesis of smoking-induced AP include the sympathetic (via the α-7 nicotinic acetylcholine receptor and β-adrenergic receptors) and parasympathetic systems (through nicotinic preganglionic receptors), as well as, cholecystokinin (CCK) through their actions on acinar cells and zymogen secretion. Besides, nicotine with its inherent vasoconstrictive potential (inhibitory effect on endothelial nitric oxide synthase or stimulatory effect on endothelin-1) can act on the pancreatic microvasculature worsening AP that has been triggered off by another cause (alcohol or gallstones). Nicotine and acrolein have the potential (yet to be conclusively proven) to influence ductal secretion by contributing to cystic fibrosis trans membrane regulator (CFTR) dysfunction [8].

Edwin Thrower eloquently dissected out the cellular mechanisms by which the chemical constituents of cigarette smoke can alter calcium signalling, zymogen activation as well as affect thiamin levels in the acinar cell contributing to the development of AP [9].

Both these manuscripts provided a strong base to stimulate further research on the association between smoking and acute pancreatitis. More importantly, though, they lent credibility to the clinical concerns [4-7] raised about smoking being involved in the causation of AP. This has led to AP being added to the already long list of diseases (cardiovascular, metabolic and respiratory) caused by tobacco use. In the pancreas, itself, smoking has been acknowledged to be not only an important cause for chronic pancreatitis [10], but a leading risk factor for pancreatic cancer, including early onset disease [11].

We are well aware that smoking is a major contributor to the global burden of disease [12]. Recent reports have provided sufficient evidence that tobacco use and exposure begins at an early age [13]. As pancreatologist, we can no longer ignore the risks of smoking. We cannot afford to turn a blind eye and opt to sit out this 'war against tobacco' [14]. It is everyone's problem!

The question remains, 'how can we contribute?'. Adopting negative strategies such as Tobacco Industry Denormalization (TID) is less likely to yield success, owing to the lack of interest in such approaches by heavy smokers [15]. More so, these are strategies best left to policy makers. Instead, as clinicians we can adopt a more positive approach not only with patients, but our acquaintances as well, focusing on the 'good' effects of the cessation of smoking [15,16]. The use of such simple, but significant, strategies in our clinics, hospitals and everyday lives, needs to be more readily embraced if we are to do our bit in reducing the ever growing burden of tobacco-related diseases.

References

1. Barreto SG, Jardine D, Phillips P, Bhatia M, Saccone GT. Can by-products in country-made alcohols induce acute pancreatitis? Pancreas. 2010; 39: 1199-1204.
2. Barreto SG, Rodrigues J. Acute pancreatitis in Goa--a hospital-based study. J Indian Med Assoc. 2008; 106: 575-576, 578.
3. Barreto SG, Saccone GT. Alcohol-induced acute pancreatitis: the 'critical mass' concept. Med Hypotheses. 2010; 75: 73-76.
4. Majumder S, Gierisch JM, Bastian LA. The Association of Smoking and Acute Pancreatitis: A Systematic Review and Meta-analysis. Pancreas. 2015; 44: 540-546.
5. Sun X, Huang X, Zhao R, Chen B, Xie Q. Meta-analysis: Tobacco smoking may enhance the risk of acute pancreatitis. Pancreatology. 2015; 15: 286-294.
6. Yuhara H, Ogawa M, Kawaguchi Y, Igarashi M, Mine T. Smoking and risk for acute pancreatitis: a systematic review and meta-analysis. Pancreas. 2014; 43: 1201-1207.
7. Ye X, Lu G, Huai J, Ding J. Impact of smoking on the risk of pancreatitis: a systematic review and meta-analysis. PLOS One. 2015; 10: e0124075.
8. Barreto SG. How does cigarette smoking cause acute pancreatitis? Pancreatology. 2016; 16: 157-163.
9. Thrower E. Pathologic cellular events in smoking-related pancreatitis. Cancers (Basel). 2015; 7: 723-735.
10. Yadav D, Slivka A, Sherman S, Hawes RH, Anderson MA, Burton FR, et al. Smoking is under recognized as a risk factor for chronic pancreatitis. Pancreatology. 2010; 10: 713-719.
11. McWilliams RR, Maisonneuve P, Bamlet WR, Petersen GM, Li D, Risch HA, et al. Risk Factors for Early-Onset and Very-Early-Onset Pancreatic Adenocarcinoma: A Pancreatic Cancer Case-Control Consortium (PanC4) Analysis. Pancreas. 2016; 45: 311-316.
12. Global, regional and national comparative risk assessment of 79 behavioural,

Citation: Barreto SG. Smoking and Acute Pancreatitis - It is about Time we Extinguish the Cigarette. Austin Pancreat Disord. 2017; 1(1): 1001.

environmental and occupational and metabolic risks or clusters of risks, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet. 2016; 388: 1659-1724.

13. Xi B, Liang Y, Liu Y, Yan Y, Zhao M, Ma C, et al. Tobacco use and second-hand smoke exposure in young adolescents aged 12-15 years: data from 68 low-income and middle-income countries. Lancet Glob Health. 2016; 4: 795-805.

14. Cole H, Fiore M. The War against Tobacco: 50 years and counting. JAMA. 2014; 311: 131-132.

15. Kushnir V, Selby P, Cunningham JA. Association between tobacco industry denormalization beliefs, tobacco control community discontent and smokers' level of nicotine dependence. Addict Behav. 2013; 38: 2273-2278.

16. Cataldo JK, Hunter M, Petersen AB, Sheon N. Positive and instructive anti-smoking messages speak to older smokers: a focus group study. Tob Induc Dis. 2015; 13: 2.

Citation: Barreto SG. Smoking and Acute Pancreatitis - It is about Time we Extinguish the Cigarette. Austin Pancreat Disord. 2017; 1(1): 1001.