

Protective and Therapeutic Properties of Obestatin in Experimental Models of Acute Pancreatitis

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Rec date: Aug 17, 2015; Acc date: Sep 01, 2015; Pub date: Sep 7, 2015

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Keywords: Obestatin; Therapeutic properties; Acute pancreatitis; Histological mechanisms; Pancreas

Short Communication

Acute pancreatitis (AP) still remains associated with high mortality rates reaching as high as 30% despite substantial improvements in the management of the disease [1,2]. This is due to complex disease aetiology, its diverse clinical course, as well as, the lack of targeted treatment owing to the poor understanding of its pathogenesis. A number of histological mechanisms have been associated with AP and are responsible for permanent morphological and structural changes of the gland in the course of severe AP [3]. The article "Pretreatment with obestatin reduces the severity of ischemia/reperfusion-induced acute pancreatitis in rats" provides fascinating data how administration of obestatin inhibits the development of ischemia/reperfusion-induced AP [4]. Authors' conclusions, together with previous findings, suggest that protective effect of obestatin in the pancreas is universal and independent of the primary cause of acute pancreatitis. The observations are in agreement that different initial causes can damage pancreas through same mechanisms or even through different mechanisms, which, however, share same end-point biochemical and histological markers (very uncommon in human biology), as obestatin probably expresses its protective effect, according to the results, through controlling all these biochemical and histopathological parameters which definitely express a specific mechanism of pancreatic damage (oxidative stress). The study prompts a question about what should be an acceptable possible way of obestatin to fail to express its protection in case of ischemia/reperfusion-induced AP. The phenomenon of obestatin protection seems to be oxidative stress- and dose-dependent. Accordingly, the reason for the statistically insignificant blood flow improvement although statistically significant oxidative stress scavenging, remains unknown. Relative changes in the expression of these results do not alter, however, the core issue of this important question.

In another study authors investigated the effect of obestatin treatment on the severity of acute ischemia-reperfusion induced pancreatitis [5]. Treatment with the exogenous obestatin reduced severity of ischemia/reperfusion-induced AP and accelerated its recovery as evidenced by reduced pancreatic oedema, vacuolisation of acinar cells, haemorrhages, acinar necrosis, and leukocyte infiltration

of the gland. Although the protective effect of obestatin has been demonstrated earlier in the caerulein induced AP model, the study is sound and described significant observations since obestatin has been used as post treatment, and has more clinical significance compared to the pretreatment administration. Since this pancreatitis model seemed to be focal the paper rightly provided different magnifications for each time point, and appropriately used a 0-5 scoring for the histological evaluation.

The authors have recently reported the effect of ghrelin on the ischemia/reperfusion induced AP, and it was important that the researchers increased herein the amount of data, to better understand the mechanisms of the protective effects of obestatin [6]. Additionally, authors hypothesized that obestatin may protect pancreas through GLP-1R and rightly mentioned that it has been shown previously that genetic and pharmacological interference with GLP-1R does not affect severity of pancreatitis in cerulein model.

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