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Expression patterns of c-met in pancreatic atrophy after pancreatic duct ligation in the mouse

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Canine pancreatic acinar atrophy (PAA) is defined as chronic pancreatic disease characterized by progressive atrophy of acinar cells in absence of inflammatory response. There are no animal models which correctly reflect pathophysiology of canine PAA, but the pancreatic duct ligation (PDL) model in the mouse resembles canine PAA histopathologically.

Activation of the receptor c-met stimulates motility, mitosis, morphogenesis, processes involved in organ regeneration, or progression of malignancies.

In this study, I investigated the time-dependent expression of c-met in the pancreas after PDL in the mouse by RT-PCR and Western blotting. In addition, immunohistochemical detection of c-met was carried out.

Histopathological findings showed that artophic degeneration including subsequent loss of acinar cells occurred from 3 days after the ligation. At 7 days after the ligation, pancreatic lobules were markedly atrophied and replaced by duct like structure consisting of group of duct cells varying in size. Expression of c-met mRNA was detected in pancreas after the ligation. And the tendency to time-dependent increase of c-met protein expression was observed until 7 days after the ligation. Immunohistochemical studies showed that both acinar and islet cells in the pancreas after the ligation were positive for c-met. At 7 days after the ligation, cells composed of duct like structure and cells accumulated adjacent to duct like structure were positive.

These results showed that the c-met mRNA and protein were up-regulated in the pancreas after severe acinar cell damage. It was suggested that centroacinar cells, intercalated duct cells and the cells composed of duct like structure might contribute to tissue repair of the pancreas injured by PDL. Furthermore, it appeared that analysis of the c-met expression patterns in canine PAA may be useful to elucidate the pathogenesis of canine PAA.