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Etiological diagnosis rate of acute pancreatitis can be improved by means of PDCA cycle

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Aim: To explore whether the PDCA cycle method (plan, do, check and act) is helpful to improve the etiological diagnosis rate of acute pancreatitis.

Methods: The existing etiological diagnosis data of acute pancreatitis were analyzed from 2005 to 2008 according to the database of acute pancreatitis in our department. The PDCA cycle method was applied to analyze the reason of low etiological diagnosis rate, and then improvement plan was formulated and executed, and the effect was assessed. The etiological diagnosis rate for each year from 2009 to 2014 was calculated and analyzed.

Results: The process of etiological diagnosis was standardized by means of PDCA cycle. The ratio of idiopathic acute pancreatitis (IAP) was on a generally downward trend, from 26.8% in 2008 to 6.1% in 2014. Accordingly, the etiological diagnosis rate of acute pancreatitis was on a generally upward trend, from 73.2% in 2008 to 93.9% in 2014.

Conclusion: PDCA cycle method is helpful to improve the etiological diagnosis rate of acute pancreatitis.

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Novel therapeutic targets for acute pancreatitis

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Acute pancreatitis is a common clinical condition. Excessive systemic inflammatory response syndrome (SIRS) in acute pancreatitis leads to distant organ damage and multiple organ dysfunction syndromes (MODS), which is the primary cause of morbidity and mortality in this condition. Development of in vivo experimental models of acute pancreatitis and associated systemic organ damage has enabled us to study the role played by inflammatory mediators in the pathogenesis of acute pancreatitis and associated systemic organ damage. Using these models, recent studies have established the critical role played by inflammatory mediators in acute pancreatitis and the resultant MODS. Hydrogen sulfide (H2S) plays an important role in cardiovascular, central nervous and gastrointestinal systems and has been shown to act as a vasodilator. We have also shown that H2S acts as a mediator of inflammation. Substance P is 11 amino-acid neuropeptide that is released from nerve endings in many tissues. Subsequent to its release, substance P binds to neurokinin-1 (NK-1) receptors on the surface of effector cells. Using experimental models, recent studies in our laboratory have established the critical role played by H2S and substance P in acute pancreatitis. Furthermore, early results point to the clinical relevance of this research. Studies with experimental animal models of disease will therefore help define the role of these mediators in the pathogenesis of acute pancreatitis and can lead to the development of novel therapeutic approaches for this condition.

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