ISSN 2308-3840 (online)

World Journal of *Meta-Analysis*

World J Meta-Anal 2019 March 31; 7(3): 66-119

Published by Baishideng Publishing Group Inc

WJMA

World Journal of Meta-Analysis

Contents

Irregular Volume 7 Number 3 March 31, 2019

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World Journal of Meta-Analysis

Volume 7 Number 3 March 31, 2019

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Responsible Electronic Editor: *Yun-Xiaojian Wu* **Proofing Editorial Office Director:** *Jin-Lei Wang*

NAME OF JOURNAL
World Journal of Meta-Analysis

ISSN
ISSN 2308-3840 (online)

LAUNCH DATE
May 26, 2013

FREQUENCY
Irregular

EDITORS-IN-CHIEF
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https://www.wjgnet.com/2308-3840/editorialboard.htm

EDITORIAL OFFICE
Jin-Lei Wang, Director

PUBLICATION DATE
March 31, 2019

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World J Meta-Anal 2019 March 31; 7(3): 96-100

DOI: 10.13105/wjma.v7.i3.96

ISSN 2308-3840 (online)

MINIREVIEWS

Early immune response in post endoscopic retrograde cholangiopancreatography pancreatitis as a model for acute pancreatitis

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Author contributions: Plavsic I and Hauser G designed and conduct research and are the guarantors of this work; Plavsic I, Hauser G, Zitinic I, Tulic V, Poropat G contributed to the discussion, and reviewed and edited the manuscript; Plavsic I, Marusic M and Hauser G analyzed collected data; Plavsic I, Hauser G and Zitinic I wrote the paper.

Supported by University of Rijeka grant, No. 18.04.2.1.01.

Conflict-of-interest statement: The author has no conflict of interest to declare.

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Abstract

This opinion review summarizes comparison of clinical presentation and immunology of post-endoscopic pancreatitis and acute pancreatitis (AP) of other etiology. The rationale for this topic was found in studies that mention differences in clinical presentation between these entities, stating that severe form of AP after endoscopic retrograde cholangiopancreatography was more severe than AP of other etiology. Found difference in clinical presentation may have a background in different immunology that needs to be further investigated.

Key words: Innate immunity; Pancreatitis immunology; Post endoscopic retrograde cholangiopancreatography pancreatitis

Manuscript source: Invited manuscript

Received: February 10, 2019
Peer-review started: February 12, 2019
First decision: March 20, 2019
Revised: March 27, 2019
Accepted: March 27, 2019
Article in press: March 28, 2019
Published online: March 31, 2019

P-Reviewer: Ierardi E, Kitamura K, Huan C
S-Editor: Dou Y
L-Editor: A
E-Editor: Wu YXJ

Core tip: Innate immunity plays an immense role in the development of acute pancreatitis (AP) and may determine the course of the disease. Information about the role of innate immunity in patients with post-endoscopic pancreatitis (PEP) is still deficient. PEP may serve as an ideal model for further research of innate immunity function in AP development.

Citation: Plavsic I, Zitinic I, Tulic V, Poropat G, Marusic M, Hauser G. Early immune response in post endoscopic retrograde cholangiopancreatography pancreatitis as a model for acute pancreatitis. *World J Meta-Anal* 2019; 7(3): 96-100
URL: https://www.wjgnet.com/2308-3840/full/v7/i3/96.htm
DOI: https://dx.doi.org/10.13105/wjma.v7.i3.96

INTRODUCTION

Acute pancreatitis (AP) is the most common gastrointestinal cause of morbidity and mortality, with a reported incidence that varies between 4.9 and 73.4 cases per 100000 worldwide[1]. The most common cause of AP are gallstones, followed by alcohol abuse as an independent risk factor.

Endoscopic retrograde cholangiopancreatography (ERCP) is an invasive diagnostic and therapeutic technique, which carries certain complication risks. Acute pancreatitis is the most common one. According to the European Society of Gastrointestinal Endoscopy guidelines, the reported incidence of post-endoscopic pancreatitis (PEP) is 3.5% in unselected patients[2].

The severity of AP can be divided into mild, moderately severe or severe based on the presence or absence of persistent organ failure and local and systemic complications. In 90% of cases, PEP is of a mild or moderate severity[2].

Although, the prevalence of the severe form of PEP is reported to be low, Testoni *et al*[3] in their study reported that the overall and severe pancreatitis-related mortality was approximately double after ERCP in comparison with AP of other causes. Also, the length of hospital stay in severe cases was longer for post ERCP pancreatitis.

Treatment of AP regardless of the cause, is primarily supportive and implies a certain economic burden to the healthcare system worldwide. Even more, if it develops into a severe form[4].

More thorough clarification of the disease pathogenesis is needed, in order to find an adequate immune target to predict and consequently prevent the severe form of the disease[5].

CLINICAL PRESENTATION

Admission in hospital varies between patients who develop AP, therefore the exact time of injury is not known. In post-ERCP AP there is the opposite situation, the exact time of injury can be foreseen. Messmann *et al*. concluded that post-ERCP AP represents an adequate model for the evaluation of early immune response in AP. The researchers linked higher values of IL-6 and CRP with the development of AP after ERCP, and concluded that the severity of the disease is not only reflected by a higher, but also earlier peaking, IL-6 serum concentration[6]. The role of IL-6 in predicting disease severity was also recognised in patients with AP[7,8]. Time is a limit for IL-6, since most of the studies confirmed its effectiveness as a disease severity marker between 12-24 h after ERCP[9,10]. Opposite to Il-6 and CRP values, amylase and lipase values were unselectively elevated in all patients after ERCP with an earlier peak in their higher values[6,10]. Amylase and lipase are released into the systemic circulation due to a disturbance in transport and an increase in ductal permeability; however, they are not thought to be responsible for inducing further inflammation. They can't discriminate between patients with the potential to develop the mild or severe form of the disease[10]. According to the present guidelines, use of the Cotton criteria is recommended and amylase values are evaluated after 24 h for the diagnosis of PEP[2].

Differences between the mild and severe form of PEP and non-ERCP were found, they are summarised in Table 1. (With permission: Plavsic *et al*[5])

Testoni *et al*[3] reported that clinicians may overestimate the presence of true

Table 1 Differences in clinical presentation of post-endoscopic pancreatitis *vs* acute pancreatitis

	Post-endoscopic pancreatitis	Acute pancreatitis	Conclusion
Fung *et al*[11] endoscopic retrograde cholangiopancreatography - induced acute necrotising pancreatitis *vs* acute necrotising pancreatitis induced by other causes.	Higher APACHE II scores on admission	Lower APACHE II scores on admission	acute necrotising pancreatitis is more severe when induced by endoscopic retrograde cholangiopancreatography
	More extensive pancreatic necrosis	Less extensive pancreatic necrosis	
	Higher rate of infected necrosis	Lower rate of infected necrosis	
Testoni *et al*[3] endoscopic retrograde cholangiopancreatography induced acute pancreatitis *vs* non endoscopic retrograde cholangiopancreatography induced acute pancreatitis	No statistical difference: (1) the severity of the pancreatitis; (2) the mortality rate (double in severe post-endoscopic pancreatitis); (3) hospitalisation		
	In the mild form of acute pancreatitis, serum amylase fell by 50% in 38.9 h. Peak serum amylase halved within 48 h in 92% of patients	In the mild form of acute pancreatitis, serum amylase fell by 50% in 46.4 h. Peak serum amylase halved within 48 h in 73.6% of patients	There was a statistical difference ($P < 0.001$). Mild form of post-endoscopic pancreatitis, a sort of pancreatic reaction, instead of a true episode of acute pancreatitis
Abid *et al*[12] mild form: Endoscopic retrograde cholangiopancreatography induced acute pancreatitis *vs* non endoscopic retrograde cholangiopancreatography induced acute pancreatitis	Shorter duration of pain; Shorter time of intravenous hydration; Shorter time to the resumption of an oral diet; Shorter hospital stay. ($P < 0.001$).		Endoscopic retrograde cholangiopancreatography-induced acute pancreatitis mild attacks run a significantly shorter and milder course than non- endoscopic retrograde cholangiopancreatography related mild attacks

pancreatic acute damage in mild PEP, based on significant differences found in the dynamics of serum amylase values measured in patients with PEP and non- ERCP AP. Severe PEP was associated with a higher mortality rate and a longer hospital stay, although with no significant differences.

IMMUNOLOGY

Disease immunology was extensively studied in both groups of patients and certain components of the immune system emerged as a potential disease severity marker. (Table 2)

Systemic inflammatory response syndrome causes the activation of the compensatory anti-inflammatory response syndrome (CARS). Too strong a CARS, paradoxically leads to immunosuppression and a higher possibility of infection[22]. A fall in the co-expression of HLA-DR on CD14+ monocytes is considered a standard laboratory indicator of a CARS[23] Analysis of this immune component is linked to the severe form of AP and immunosuppression.

Dysregulated host inflammatory response was included in the new sepsis definition by the Society for Critical Care Medicine and the European Society of Intensive Care in 2016. A recent review article analysed the role of the major innate lymphocyte population, Natural Killer (NK) cells, in the dysregulated host inflammatory response on infection. They concluded that NK cells appear to be critical for the elimination of pathogens during the early phase of sepsis and lead to the prevention of secondary infection during the immunosuppressive phase. This opinion suggests that they may be suitable as new immunotherapeutic agents[24].

Infection is considered to be the most important prognostic factor for disease severity in AP, regardless of the cause. In non-ERCP AP, infection is considered to be the secondary event, while in PEP it's considered to be the primary event[3].

CONCLUSION

It has been proven that innate immunity plays an immense role in AP and the imbalance of innate immunity may determine the severity of the disease early in the course of the disease[16,25,26]. As Table 2 shows, most of the studies that researched the role of immune cells in innate immunity, used patients with AP as the research subjects. Answers about the role of immune cells in patients with PEP are still insufficient.

On the contrary, the role of different cytokines in both groups of patients was extensively studied.

Table 2 Role of immune components in predicting disease severity

	Acute pancreatitis	Post-endoscopic pancreatitis
Monocytes and macrophages	(1) Expression of HLA-DR on monocytes gives a good insight into monocyte function; (2) Decreased monocyte HLA-DR expression may serve as an indicator of immunosuppression[13]; and (3) Decreased monocyte HLA- DR expression predicts the development of organ dysfunction in severe acute pancreatitis[13].	
T cells	(1) $CD4^+$ lymphocytes are reported to have a direct cytotoxic effect on acinar cells[14]; (2) Depletion of $CD4^+$ lymphocytes reduces the severity of acute pancreatitis[15]; and (3) Reduction in the number of cytotoxic T lymphocytes (CD3+CD8+) in severe form of acute pancreatitis[16].	
Natural Killer cells	(1) Depletion of the natural killer cell population on the first day of severe acute pancreatitis[16]; and (2) No significant change in natural killer cell number in mild acute pancreatitis[16].	
IL-10	Predictive marker of organ failure in severe acute pancreatitis[17].	Conflicting results about reducing the incidence of post endoscopic retrograde cholangiopancreatography acute pancreatitis after IL-10 usage[18,19].
IL- 6	Independent factor for predicting severity in acute non- endoscopic retrograde cholangiopancreatography pancreatitis[7].	(1) Peak value 24-48 h after clinical expression of post endoscopic pancreatitis; and (2) In necrotising post endoscopic pancreatitis, the peak levels of IL-6 occur after 24 h[6].
IL-1β	(1) Required for full pancreatic and distal organ injury and inflammation[20]; and (2) Values peak after 24 h and are larger in patients with severe acute pancreatitis compared to mild acute pancreatitis, although a strong correlation with acute pancreatitis severity in humans wasn't found[21].	

Further investigation of innate immunity cells and their function in PEP is important. Especially, as already mentioned, the exact time of injury in PEP is known and therefore it may represent a good model for evaluation of the early immune response in AP[6,27].

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