

PEER-REVIEW REPORT

Name of journal: *World Journal of Gastrointestinal Surgery*

Manuscript NO: 84302

Title: Idiopathic hypereosinophilic syndrome with hepatic sinusoidal obstruction syndrome: A case report and literature review

Provenance and peer review: Unsolicited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 03322877

Position: Peer Reviewer

Academic degree: MD, MSc

Professional title: Professor

Reviewer's Country/Territory: Turkey

Author's Country/Territory: China

Manuscript submission date: 2023-03-08

Reviewer chosen by: Geng-Long Liu

Reviewer accepted review: 2023-03-29 05:59

Reviewer performed review: 2023-03-29 07:21

Review time: 1 Hour

Scientific quality	<input type="checkbox"/> Grade A: Excellent <input type="checkbox"/> Grade B: Very good <input checked="" type="checkbox"/> Grade C: Good <input type="checkbox"/> Grade D: Fair <input type="checkbox"/> Grade E: Do not publish
Novelty of this manuscript	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Good <input type="checkbox"/> Grade C: Fair <input type="checkbox"/> Grade D: No novelty
Creativity or innovation of this manuscript	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Good <input type="checkbox"/> Grade C: Fair <input type="checkbox"/> Grade D: No creativity or innovation

Scientific significance of the conclusion in this manuscript	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Good <input type="checkbox"/> Grade C: Fair <input type="checkbox"/> Grade D: No scientific significance
Language quality	<input type="checkbox"/> Grade A: Priority publishing <input checked="" type="checkbox"/> Grade B: Minor language polishing <input type="checkbox"/> Grade C: A great deal of language polishing <input type="checkbox"/> Grade D: Rejection
Conclusion	<input type="checkbox"/> Accept (High priority) <input type="checkbox"/> Accept (General priority) <input checked="" type="checkbox"/> Minor revision <input type="checkbox"/> Major revision <input type="checkbox"/> Rejection
Re-review	<input type="checkbox"/> Yes <input checked="" type="checkbox"/> No
Peer-reviewer statements	Peer-Review: <input checked="" type="checkbox"/> Anonymous <input type="checkbox"/> Onymous
	Conflicts-of-Interest: <input type="checkbox"/> Yes <input checked="" type="checkbox"/> No

SPECIFIC COMMENTS TO AUTHORS

Dear authors, -As the meaning of the sentence in conclusion part of the Abstract is obscure (There is a possibility of combined HVOD and IHES and our drug regimen case can be used as a reference.) it must be rephrased. -The authors should explain why they did not use defibrotide in treatment of HSOS. -As the HSOS is preferred in new nomenclature they should use HSOS instead of HVOD. -The last status of the patient is not clearly defined.

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Reviewer's code: 03699961

Position: Associate Editor

Academic degree: MD, PhD

Professional title: Professor

Reviewer's Country/Territory: Japan

Author's Country/Territory: China

Manuscript submission date: 2023-03-08

Reviewer chosen by: Geng-Long Liu

Reviewer accepted review: 2023-03-29 14:58

Reviewer performed review: 2023-03-30 12:13

Review time: 21 Hours

Scientific quality	<input type="checkbox"/> Grade A: Excellent <input type="checkbox"/> Grade B: Very good <input checked="" type="checkbox"/> Grade C: Good <input type="checkbox"/> Grade D: Fair <input type="checkbox"/> Grade E: Do not publish
Novelty of this manuscript	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Good <input type="checkbox"/> Grade C: Fair <input type="checkbox"/> Grade D: No novelty
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Conclusion	<input type="checkbox"/> Accept (High priority) <input type="checkbox"/> Accept (General priority) <input type="checkbox"/> Minor revision <input checked="" type="checkbox"/> Major revision <input type="checkbox"/> Rejection
Re-review	<input checked="" type="checkbox"/> Yes <input type="checkbox"/> No
Peer-reviewer statements	Peer-Review: <input checked="" type="checkbox"/> Anonymous <input type="checkbox"/> Onymous
	Conflicts-of-Interest: <input type="checkbox"/> Yes <input checked="" type="checkbox"/> No

SPECIFIC COMMENTS TO AUTHORS

Title: Idiopathic hypereosinophilic syndrome with hepatic veno-occlusive disease: A case report and literature review Xu-Tao Xu, Bing-Hong Wang, and Yang-Jie Guo, et al.

1) General Comments In this case report, a 70-year-old male is presented to show the pathological association between hepatic veno-occlusive disease and eosinophilia in the peripheral blood during the course of treatment using corticosteroids, which suggests that eosinophils induce endothelial cell damage of the hepatic sinusoids. This type of rare case is crucial to learn fundamental mechanisms and functions of biology through specific pathophysiology. In this case, the liver biopsy specimen did not reveal the infiltration of eosinophils. Therefore, it is inevitable to discuss the mechanism of veno-occlusive disease in a case with the hypereosinophilic syndrome to make this case report valuable.

RE-REVIEW REPORT OF REVISED MANUSCRIPT

Name of journal: *World Journal of Gastrointestinal Surgery*

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Title: Idiopathic hypereosinophilic syndrome with hepatic sinusoidal obstruction syndrome: A case report and literature review

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Peer-review model: Single blind

Reviewer's code: 03699961

Position: Associate Editor

Academic degree: MD, PhD

Professional title: Professor

Reviewer's Country/Territory: Japan

Author's Country/Territory: China

Manuscript submission date: 2023-03-08

Reviewer chosen by: Li Li

Reviewer accepted review: 2023-04-18 23:40

Reviewer performed review: 2023-04-19 07:36

Review time: 7 Hours

Scientific quality	<input type="checkbox"/> Grade A: Excellent <input type="checkbox"/> Grade B: Very good <input type="checkbox"/> Grade C: Good <input checked="" type="checkbox"/> Grade D: Fair <input type="checkbox"/> Grade E: Do not publish
Language quality	<input type="checkbox"/> Grade A: Priority publishing <input checked="" type="checkbox"/> Grade B: Minor language polishing <input type="checkbox"/> Grade C: A great deal of language polishing <input type="checkbox"/> Grade D: Rejection
Conclusion	<input type="checkbox"/> Accept (High priority) <input type="checkbox"/> Accept (General priority) <input type="checkbox"/> Minor revision <input checked="" type="checkbox"/> Major revision <input type="checkbox"/> Rejection
Peer-reviewer	Peer-Review: <input checked="" type="checkbox"/> Anonymous <input type="checkbox"/> Onymous

statements

Conflicts-of-Interest: [] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

Title: Idiopathic hypereosinophilic syndrome with hepatic veno-occlusive disease: A case report and literature review Xu-Tao Xu, Bing-Hong Wang, and Yang-Jie Guo, et al.

1) General Comments In this case report, a 70-year-old male is presented to show the pathological association between hepatic veno-occlusive disease and eosinophilia in the peripheral blood during the course of treatment using corticosteroids, which suggests that eosinophils induce endothelial cell damage of the hepatic sinusoids. This type of rare case is crucial to learn fundamental mechanisms and functions of biology through specific pathophysiology. In this case, the liver biopsy specimen did not reveal the infiltration of eosinophils. Therefore, it is inevitable to discuss the mechanism of veno-occlusive disease in a case with the hypereosinophilic syndrome to make this case report valuable. Response: The pathogenesis of HSOS involves the injury of endothelial cells and hepatocytes in the hepatic sinusoids caused by HSCT, chemotherapeutic agents, PA, etc., as well as locally released cytokines that also induce the activation of cell adhesion molecules on endothelial cells, leading to local cell damage and shedding, resulting in activation of the coagulation cascade, the formation of blood clots, and the loss of thrombus-fibrinolytic balance. Not only do eosinophils cause tissue damage, they can also be rapidly recruited to the site of injury for platelet adhesion to form thrombus and are activated through direct interaction with platelets. Activated eosinophils contribute to platelet activation, inhibit the function of thrombomodulin, and promote thrombus formation. It is speculated from the pathogenesis that hypereosinophilia may lead to venous thrombosis and hepatic venule occlusion through imbalance of the coagulation fibrinolysis balance caused by endothelial cell injury. R1 comment: The discussion made by the authors in the rebuttal

pertains to the general mechanisms connecting hypereosinophilia and endothelial cell damage in veins. However, the discussion should focus on the reasons why endothelial cell damage occurs specifically in the hepatic sinusoids, even though the liver biopsy specimen did not reveal eosinophil infiltration.