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PEER-REVIEW REPORT

Name of journal: World Journal of Clinical Cases

Manuscript NO: 55851

Title: Primary myelofibrosis with concurrent CALR and MPL mutations: A case report

Reviewer's code: 00068723
Position: Editorial Board
Academic degree: MD, PhD
Professional title: Doctor, Occupational Physician
Reviewer's Country/Territory: Japan
Author's Country/Territory: China

Manuscript submission date: 2020-04-05
Reviewer chosen by: Jin-Lei Wang
Reviewer accepted review: 2020-09-17 06:47
Reviewer performed review: 2020-09-20 04:28
Review time: 2 Days and 21 Hours

Scientific quality	[ ] Grade A: Excellent [ ] Grade B: Very good [ ] Grade C: Good [Y] Grade D: Fair [ ] Grade E: Do not publish
Language quality	[ ] Grade A: Priority publishing [ ] Grade B: Minor language polishing [Y] Grade C: A great deal of language polishing [ ] Grade D: Rejection
Conclusion	[ ] Accept (High priority) [ ] Accept (General priority) [ ] Minor revision [Y] Major revision [ ] Rejection
Re-review	[ ] Yes [Y] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

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SPECIFIC COMMENTS TO AUTHORS

The authors reported a case of myelofibrosis subjected to next generation sequencing. They found mutations in CALR, MPL, and PIK3RI. The results were intriguing, but the presented clinical data were immature. It was hard to imagine the patient with the presented data. Results of blood examination were absent. They should be presented in Tables. CT was performed, but the images were absent. The authors said that the patient had splenomegaly. It would be better to show the spleen with the CT. Bone marrow biopsy was performed. The photos of bone marrow should be presented. The reason was not clear how ruxolitinib was administered. Brief information on treatment strategy for primary myelofibrosis was necessary. Not all the readers are specialist on hematology. Part of Introduction should be spent on the information on CALR, MPL and PIK3RI. How would the authors speculate the biological significance of the mutations of CALR, MPL and PIK3RI. Did the authors conclude that ruxolitinib failed? If so, was the mutations related with the treatment failure? If ruxolibinit improved the clinical cause of patient, did the mutations affect the effects of the agent?

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PEER-REVIEW REPORT

Name of journal: World Journal of Clinical Cases

Manuscript NO: 55851

Title: Primary myelofibrosis with concurrent CALR and MPL mutations: A case report

Reviewer's code: 01036411

Position: Peer Reviewer

Academic degree: MD

Professional title: Doctor

Reviewer's Country/Territory: Italy

Author's Country/Territory: China

Manuscript submission date: 2020-04-05

Reviewer chosen by: Jin-Lei Wang

Reviewer accepted review: 2020-09-17 06:35

Reviewer performed review: 2020-09-20 15:16

Review time: 3 Days and 8 Hours

Scientific quality	[ ] Grade A: Excellent [ ] Grade B: Very good [Y] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Language quality	[ ] Grade A: Priority publishing [Y] Grade B: Minor language polishing [ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection
Conclusion	[ ] Accept (High priority) [ ] Accept (General priority) [ ] Minor revision [Y] Major revision [ ] Rejection
Re-review	[ ] Yes [Y] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

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SPECIFIC COMMENTS TO AUTHORS

The authors reported on a case of primary myelofbrosis in whom NGS analysis found mutations in CALR, MPL and PIK3RI genes. The case deserves mention since both the mutations combination is rare and MPL mutation is atypical. Major criticisms - The description of the case deserves more information. One would expect the report of serum LDH, and cholesterol; one expect classification in term of prognostic score (IPSS). On expect a more detailed description of bone marrow biospy, in particular presence of clusters of megakaryocytes, description of nuclear anomalies of megakaryocytes, and myeloid cellularity. - One common challenge of NGS is to differentiate acquired somatic mutations from germline pathogenic variants. This may be done by mutation detection in germline control samples (e.g., skin fibroblasts, saliva). Also high and stable VAF (e.g., 40–50%) at follow-up may be indicative for germline alteration. The doubt about the possibility of a germline mutation of MPL must be solved.