Peer Review File

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Reviewer A

This is a well-written study. Some comments:

1. Title: The term "intracerebral hemorrhage" needs to be abbreviated before being used, i.e.: Comparison of Clinical Scores for Predicting Stroke Associated Pneumonia after Intracerebral Hemorrhage (ICH)：Potential Tools for Personalized Care and Clinical Trial in ICH

   Reply 1： Thanks for your suggestion. We modified the title and labeled it with yellow color .

2. Abstract: Same comment as above. The authors use the abbreviation without previously defining it.

   Reply 2： Thanks for your suggestion. We modified the abstract and labeled it with yellow color.

3. The Conclusions are rather short and weak. The authors need to elaborate on their Conclusions and add their recommendations about which risk model should be preferred and which should better be avoided.

   Reply 3： this is a indeed good suggestion. We modified the conclusion and labeled it with yellow color.

Reviewer B

There are some aspects of the manuscript that leave questions unanswered and thus still warrant revision:

1. Grammar should be revised, as well as some terms (e.g. "pneumatic" means "air/gas-related" not "pneumonia-related" and should not be used like it has been used in the text).

   Reply 1: thanks you very much. We modified the content and labeled it with yellow.

2. Clarify the definition of "spontaneous ICH". Which conditions were systematically seen as secondary causes and excluded? Did you consider anticoagulant-related hemorrhages or cerebral amyloid angiopathy associated hemorrhages to be spontaneous or secondary hemorrhages?

Reply 2: This is a good question. Spontaneous ICH is relative to traumatic ICH. In term of primary ICH, it refers to rupture of damaged small arteries or arterioles, most commonly secondary to either hypertension or cerebral amyloid angiopathy. Secondary ICH can result from coagulopathy, vascular malformation rupture, cerebral venous thrombosis, mycotic aneurysm rupture, moyamoya, tumor, hemorrhagic conversion of an ischemic stroke, or vasculitis. In the present study, we enrolled patients with spontaneous ICH, which included both primary (90.9%) and secondary (10.1%) ICH based on it potential etiologies.

3. How representative of a sample was your selection of 13 hospitals? Were there many hospitals which did not participate? Is this likely to cause bias in this study? If so, what kind?

   Reply 3: This is also a good question. When we select potential centers to participate the study, we evaluated the research capability and commitment to the registry of each hospital. Finally, 13 University hospital tertiary stroke center were included. In addition, our study included only hospitalized patients and those patients died in emergency department or treated in outpatient clinics were not included. Meanwhile, like most registries, our registry required informed consent and selection bias was inevitable. We added this as limitation.

4. How many patients underwent MRI? Can you estimate the proportion of cases in which cerebral amyloid angiopathy (CAA) was a significant underlying factor. Is there a correlation between CAA and SAP?

   Reply 4: In the Beijing Registration of Intracerebral Hemorrhage, the standard baseline neuroimaging is CT. however, we do have MRI for those patients enrolled by Beijing Tiantan hospital. We estimated the proportion of ICH due to CAA will be lower than that of western cohorts due to younger age of ICH onset in our cohort (mean age: 56.8+14.4). In term of the potential relationship between CAA and SAP, we can make a further investigation in the future. Thanks for your suggestion.

5. How long was your follow-up?

   Reply 5: We follow up the enrolled patients for one year after ICH.

6. Please provide some context on the SAP scores used: in what populations were they validated in previously (acute phase or later? patient nationality? spontaneous ICH or other types included as well? etc.).

   Reply 6: This is a good suggestion. We summarized the tested model and variables in the supplementary table 1. Meanwhile, we listed the predictive performance of these clinical scores in the derivation and internal validation cohorts in supplementary table 2.

7. Based on the mean age, your cohort was significantly younger than many cohorts e.g. in European and American populations. Please discuss do you think your results are generizable to other populations and why/why not.

   Reply 7: This is a good point. Though it is promising, caution need to be taken when interpreting the results: first, the study populations for derivation and validation of these models are different. The baseline characteristics of our study were different from those of

western cohorts, such as with younger age of ICH onset, less severity of neurological deficit, smaller hematoma volume, fewer intraventricular extension and lower rate of withdraw of care. Second, there might be complex genetic, social, economic factors as well as regional management philosophies and preferences that are difficult to account for when risk models are developed or applied to a distinct population. These models need to be further validated in more populations and larger samples in the future.

8. You observed that patients with LOS less than 72h were not at greater risk of developing SAP despite the fact that their condition was much more severe. You discussed that this might be due to these patients dying before SAP has chance to develop. Does your data support this claim? How long was the delay from admission to death in these patients, and how long was the delay from admission to SAP diagnosis in your overall cohort?

   Reply 8: It is important and interesting to figure out the potential reasons why all these clinical scores for predicting SAP performed better in patients survival beyond 48-72 hours after ICH. We compared the baseline characteristics between patients with LOS less than 72 hours and those longer than 72 hours. It was found that patients with shorter LOS had significantly more severe neurological deficit on admission, such as with higher NIHSS score (26 vs.10), lower GCS score (6 vs. 14) and larger hematoma volume (40.9ml vs. 15.0ml). Theoretically, these patients should have increased risk of in-hospital SAP after ICH. however, The rates of in-hospital SAP between two groups was not statistically different (30.2% vs. 29.2%). Further, we found that patients with shorter LOS had significantly higher proportion of in-hospital mortality (40.6% vs. 7.4%) and withdraw of medical care (18.4% vs. 5.7%). There are limited data on the time of SAP after stroke. Another cohort of our team (the iMCAS study) showed that the median times from onset to diagnosis of SAP after ICH, AIS and SAH were 3 days (IQR: 2-5), 4 days (IQR:2-7) and 5 days (IQR:3-7). Based on these data, we conjectured that the contradiction between neurological severity and risk of in-hospital SAP after ICH in patients with LOS less than 72 hours might be due to that patients died or left hospital before pneumonia occurred. The results was similar with that from the China National Stroke Registry (CNSR)(Stroke. 2014;45:2620-2628)

9. On Table 1, the 4th column title reads "with SPA", this should probably be "With SAP"? Please correct.

   Reply 9: Thanks for your suggestion. We modified the content and labeled it with yellow color.

10. Please provide a table presenting which variables are included in which SAP scores, as these are likely not known to most readers.

    Reply 10: This is a good suggestion. We summarized the tested model and variables in the supplementary table 1.

11. The AUROC values are notably smaller for all models in the category of patients with LOS 72h or less. Is this likely to be because of how the models have initially been validated or is your sample in this patient subgroup insufficient? The confidence intervals suggest that maybe a little bit of both? Please discuss.

    Reply 11: This is a good question and is related to question number 8. This phenomenon was similar with that from an independent cohort, the China National Stroke Registry

published in STROKE (CNSR) (Stroke. 2014;45:2620-2628). We think the different baseline characteristics and clinical course of patients with LOS less and longer than 78 might play key roles. The phenomenon should be tested in more population and larger samples.