Sampling bias, sample size, ordinal regression model, and competing risk in evaluating association between ABO blood group and severity of liver fibrosis in Hepatitis C virus patients

Sir,
I read with interest the paper by Shavakhi A et al., about association between severity of fibrosis in Hepatitis C virus (HCV) patients and ABO blood group.[1]

According to the only meta-analysis of the prevalence of HCV, total prevalence in general population of Iran is 0.16%.[2] There are some sources for sampling bias. Authors did not include those who had received anti-HCV treatments. By excluding cases who were treated for HCV, we are excluding more severe cases and may underestimate the association between severity of liver fibrosis in HCV patients and blood group. I was interested to know if there is any change in severity of this association if we consider the situation of these cases before their enrollment in the treatment and include them in analysis? On the other hand, the probability of selecting more severe cases increases when patients are selected from University clinics in comparison with private clinics.

However, it is not obvious which sample size formula has been hired. We cannot calculate sample size only according to the number of sample size of previous study. However, based on information from previous studies like percentages or standard deviation we calculate needed sample size. The sentence “With an alpha of 0.05 and power of 80% according to previous studies, 200 consecutive patients were enrolled” makes no sense.

Since this study compares cases with O and non-O blood group, it is preferred that sampling be based on such groups with equal number of persons to increase the power. If not, they should explain about the details of sampling. A sampling frame cannot be assumed. Maybe the possibility of observing significant differences increases between cases with O blood group when they recruit more samples with O blood group. It is a cross-sectional study without control group which authors have compared the results of its subgroups (cases with O and non-O blood group). In their discussion, it is preferred to explain that power of such design for evaluating mentioned hypothesis is very low.

Also, details of multivariate model such as “which method has been selected?” and “which variables have been entered into the model?” are not determined. These information affect on the results. For example, why gender with P-value = 0.763 has been entered in the ordinal regression model? It is not logical except when we need gender adjusted results. Moreover, goodness of fit and P-value of the ordinal regression model are not mentioned. Authors have not any explanations about the coefficients of the regression model: meaning of the coefficients and their clinical implication.

Competing risks like duration of infection, risky behaviors (intravenous drug usage), and alcohol consumption should be considered when we are searching about the most important risk factors for more severe fibrosis in HCV patients. It seems that the data are gathered retrospectively and some information were not accessible at the time of data collection.

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