

Diabetic Aspirin Users Versus Non-Users and their Risk of NASH and Liver Disease



Joanna Wieckowska, DO, Ashley Dababneh,
Ascension Genesys Hospital, Medical Education, Grand Blanc, MI

Introduction

- Non-alcoholic fatty liver disease (NAFLD) has been estimated to have a prevalence of 70% in the type II diabetic population, and it increases their risk for serious hepatic diseases, such as non-alcoholic steatohepatitis (NASH), cirrhosis, and hepatocellular carcinoma. [1,2]
- Recent evidence shows that daily aspirin use is associated with a lower risk of NASH and disease progression over time compared with non-aspirin use in the general population and may have a protective hepatocellular effect. [3]
- The goal of this study was to determine whether or not there is a protective hepatocellular effect of daily aspirin use in type II diabetics as related to their risk of developing NASH and serious liver disease.

Hypothesis

- The hypothesis of this research was that aspirin use in diabetics is associated with a lower incidence of NASH as predicted by the NAFLD fibrosis score.

Methods

- Retrospective chart review from July 1, 2012 to June 30, 2019, included patients who were 18 years and older who had diabetes mellitus type II. Those who took aspirin daily were assigned to the Exposure Group, and those who did not take aspirin were assigned to the Control Group. Patients' most recent set of labs were used to calculate their NAFLD fibrosis score. A score ≤ -1.455 indicated that fibrosis could be excluded with high accuracy, and a score ≥ 0.676 indicated that the presence of fibrosis could be diagnosed with high accuracy. Once this score was calculated for each patient, the two study groups were compared to see if diabetics who took aspirin daily had a lower NAFLD fibrosis score than those who did not.

Results

- There were 152 (50.7%) patients in the Exposure Group and 148 (49.3%) patients in the Control Group.
- Exposure Group had 6 (3.9%) patients with NAFLD fibrosis score ≤ -1.455 , 70 (46.1%) patients with indeterminate score, and 76 (50%) patients with score ≥ 0.675 (Figure 1).
- Control Group had 23 (15.5%) patients with score ≤ -1.455 , 56 (37.8%) patients with indeterminate score, and 69 (46.6%) patients with score ≥ 0.675 (Figure 1).
- Chi square analysis revealed a significant association between NAFLD fibrosis score and whether or not the patient uses aspirin, $p=0.002$.
- Exposure Group was 0.24 times less likely to have a score ≤ -1.455 , and Control Group was 4.2 times more likely to have score ≤ -1.455 than Exposure Group (Figure 2).

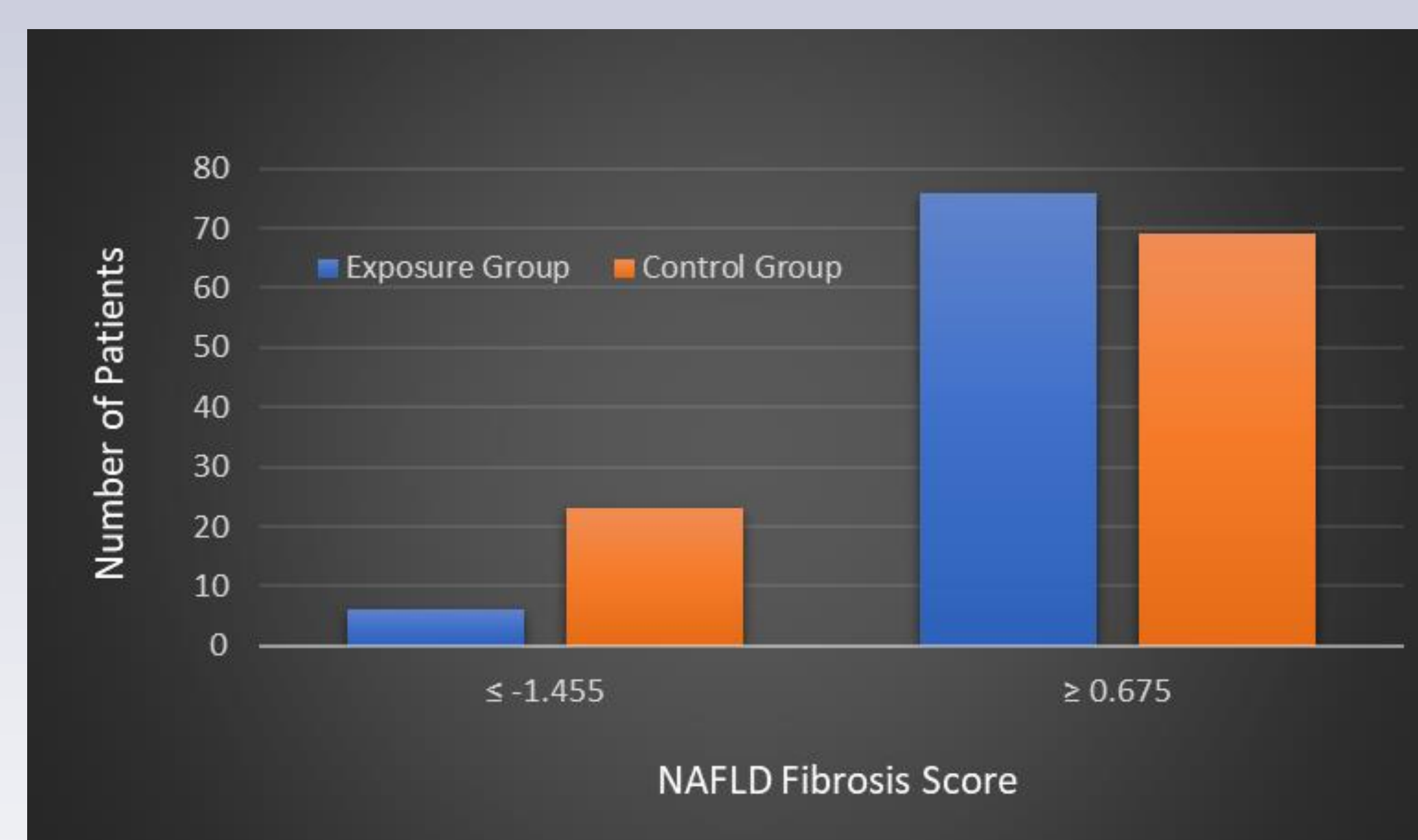


Figure 1. NAFLD Fibrosis Scores between Exposure and Control Groups

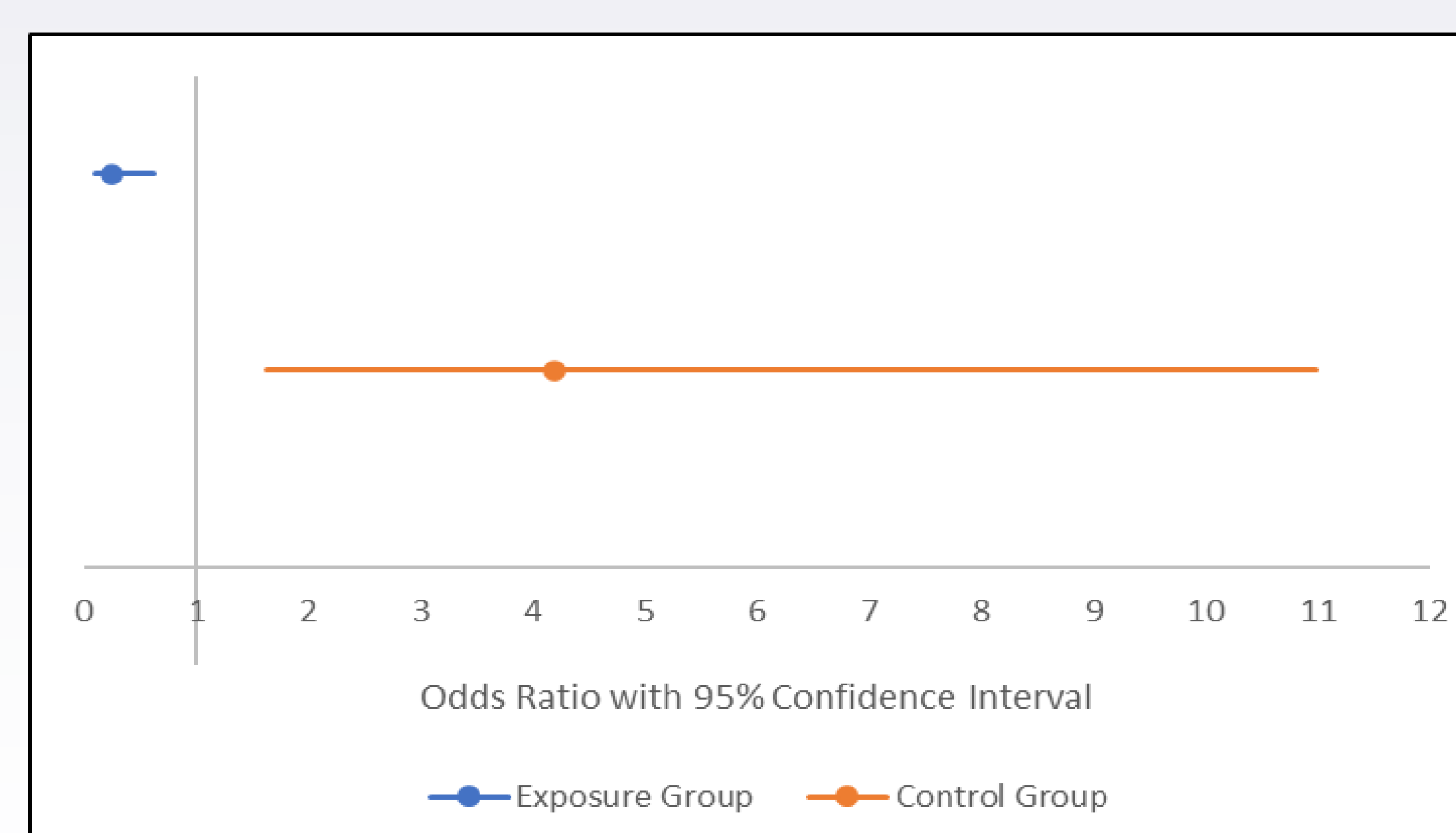


Figure 2. Scatter Plot of Odds Ratio of NAFLD Fibrosis Score ≤ -1.455 between Exposure and Control Groups

Discussion

- It is the diabetic non-aspirin users who have higher odds of not developing NASH compared to their aspirin user counterparts. Aspirin users actually seem to have a worse NAFLD fibrosis score compared to non-aspirin users (i.e., mean score 1.0 (SD: 1.7) and 0.59 (SD: 2.02), respectively).
- This may be explained by the mean number of comorbidities between diabetic non-aspirin users and aspirin users—1.5 (SD: 1.3) and 3.2 (SD: 1.4), respectively—with those having 1-2 comorbidities attaining a lower NAFLD fibrosis score than those having ≥ 3 comorbidities, p -value = 0.003.
- Given the multiple diseases associated with NAFLD and their influence on its progression [4], it may be inferred that diabetics with multiple comorbidities will be less likely to not develop NASH due to the impact that these comorbidities can have on the structure and function of the liver.

Conclusion

- Aspirin in the diabetic population may not render a protective hepatocellular effect.
- Currently, therapeutic strategies are limited to disease prevention with treatment of the underlying cause, which entails lifestyle changes and strict hyperglycemic control. [5]

References

1. Dharmalingam, M., & Yamasandhi, P. G. (2018). Nonalcoholic Fatty Liver Disease and Type 2 Diabetes Mellitus. *Indian journal of endocrinology and metabolism*, 22(3), 421–428. doi:10.4103/ijem.IJEM_585_17
2. Kim, D. et al. (2013). Association between noninvasive fibrosis markers and mortality among adults with nonalcoholic fatty liver disease in the United States. *Hepatology (Baltimore, Md.)*, 57(4), 1357–1365. doi:10.1002/hep.26156
3. Simon, T.G., et al. (2019). Daily aspirin use associated with reduced risk for fibrosis progression in patients with nonalcoholic fatty liver disease. *Clin Gastroenterol Hepatol*. doi: 10.1016/j.cgh.2019.04.061.
4. Glass, L. M., Hunt, C. M., Fuchs, M., & Su, G. L. (2019). Comorbidities and Nonalcoholic Fatty Liver Disease: The Chicken, the Egg, or Both?. *Federal practitioner : for the health care professionals of the VA, DoD, and PHS*, 36(2), 64–71.
5. Bhatt, H. B., & Smith, R. J. (2015). Fatty liver disease in diabetes mellitus. *Hepatobiliary surgery and nutrition*, 4(2), 101–108. doi:10.3978/j.issn.2304-3881.2015.01.03