

## Hidradenitis Suppurativa and Risk of Myocardial Infarction: A Plausible Association?

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### Introduction

Hidradenitis suppurativa (HS) is a chronic, inflammatory and relapsing skin disease. Cumulative evidence has established an association between HS and certain cardiovascular risk factors such as metabolic syndrome, diabetes, hypertriglyceridemia and obesity. However, whether HS is an independent risk factor for myocardial infarction (MI) remains controversial.

### Objectives

A systemic review and meta-analysis were conducted to accurately evaluate the association between HS and risk of MI. Eligible records comprised case-control, cross-sectional and cohort designs that compared rates of MI in HS to control groups. All studies without control groups (eg analysis of

prevalence or assessment of comorbidity burden) were excluded. Five databases (Pubmed, Scopus, Embase, Cochrane Library and [clinicaltrials.gov](http://clinicaltrials.gov)) were searched. Two authors extracted independently the data of the studies.

### Results

The search retrieved 15 records. After applying the inclusion criteria and removing duplicates, five records comprising 58,494 HS patients and 4,010,171 matched controls were finally analyzed. The complete flow chart of the literature search is depicted in Figure 1 and full references of the five included records are presented in the Supplementary File 1. The meta-analysis showed the relative risk of MI was similar among both cohorts: 1.00 (95%CI: 0.49-2.02,  $I^2=97%$ ; Figure 2). To test the robustness of the meta-analysis, the combined results were recalculated by excluding one study

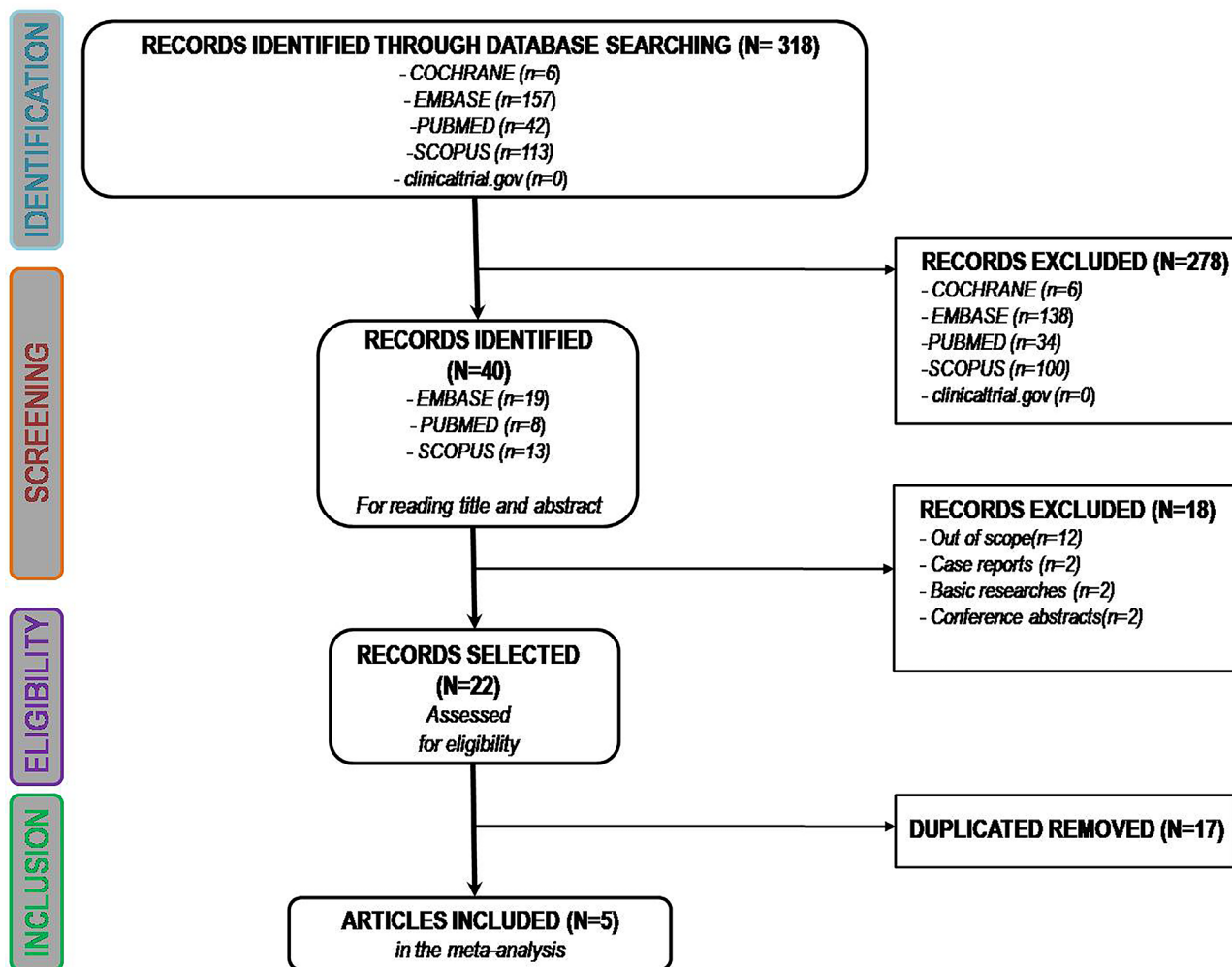


Figure 1. Flow chart of data extraction and selection process throughout the databases.

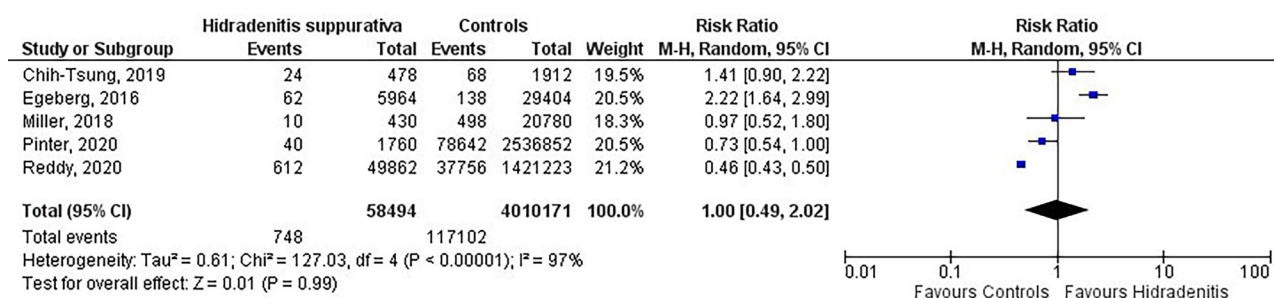


Figure 2. Forest plot of myocardial infarction in hidradenitis suppurativa individuals.

per iteration. The exclusion of any single study did not alter the overall pooled outcome.

## Conclusions

MI is mainly attributed to atherosclerosis. Atherosclerosis is a chronic inflammatory disease that shares precise pathways with HS such as overexpression of the T helper cell (Th)1/ (Th)17 [1]. Our work aggregates all available literature so far, and after pooling the largest cohort of HS patients, we

found a negative correlation between HS and MI. The trim and fill sensitivity analysis did not change the general result, suggesting our outcome is not an artefact. Though analyses of hazard ratio were conducted independently of HS severity, this does not seem to affect the final result: Pinter et al observed the cardiovascular risk of MI in HS patients is largely attributed to smoking and not to HS-related processes such as chronic systemic inflammation [2]. In fact, these authors found matching for nicotine dependence reduced the risk of MI in HS subjects to almost levels of the control participants.

Accordingly, other authors noticed smoking is more frequent in severe HS patients and smoking increased likelihood of suffering from more severe HS form [3,4]. Together, these data indicate any potential increased incidence of MI related to severity of HS is driven more to the smoking habit rather than the enhanced systemic inflammation in severe HS.

Along this line, a recent meta-analysis addressing this question have showed the odds ratio of MI of HS patients is similar to healthy controls, while the risk ratio from cohort studies cannot be assessed accurately due to the high heterogeneity of the current evidence [5].

In psoriasis which shares immunological characteristics with HS, such as Th1/Th17 pathways and involvement of TNF-alpha, a meta-analysis showed slight increased risk of MI with an overall relative risk of 1.22 [3,6]. Unlike psoriasis, this work shows HS does not influence the coronary arterial vessels, possibly due to differences in the pathological inflammatory pathways acting on blood vessels. In support of this, any potential clinical impact of HS to vascular function in precise areas of the arterial and venous vascular system such as brain or lower extremities can be soundly questioned according to recent reports in which hazards of stroke, arterial peripheral disease and venous thromboembolia were comparable to controls [7]. In sum, all these data indicate the HS-related chronic inflammation is not a trigger of cardiovascular comorbidities, but other factors and behaviors may play a more dominant role.

The major strength of our work is the sample size and the statistical power. On the basis of their characteristics, all included records can be considered good quality, well-conducted studies with long follow-up, reliable exposure and outcome assessment: most of patient groups comprised hospital-based individuals diagnosed of HS by dermatologists. Thus, potential misdiagnosis is minimized. Additionally, hospital and population-based patients differ from each other since the former may have more severe forms of HS and eventually, higher probability of cardiovascular comorbidities; hence, the significant negative association is stronger. Finally, four records of the meta-analysis consisted of general population instead of hospital controls, and although

the comparability of hospital versus population controls has not been explored in detail in case-control studies for HS, it is feasible to assume the matched-controls from general population are healthier than hospital controls, and thus, any negative correlation is even more robust.

On summary, the association between HS and MI is not evident from current literature and has yet to be elucidated. Further research is warranted to clarify as to whether the HS population have an increased trend of suffering from this major cardiovascular event.

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