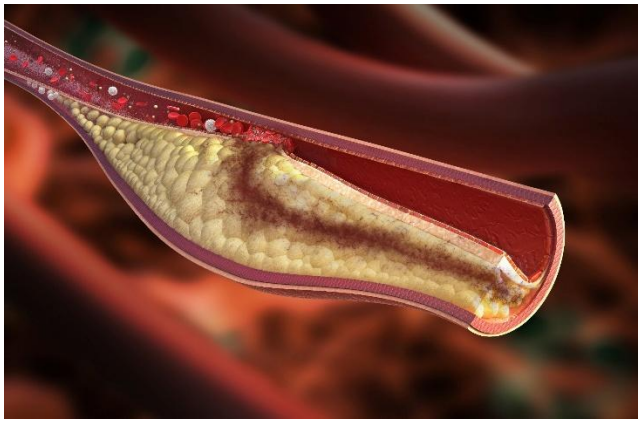
Artery Plaque Growth, Raising Heart Disease Risk Speeds Up after COVID-19

A research team from China revealed that individuals who had severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections experience faster progression of atherosclerotic plaques in their arteries, increasing their risk of heart attacks and strokes.



Study

To explore the potential link between COVID-19 and worsening heart health, the team conducted a retrospective analysis of data from 803 patients who had undergone at least two coronary computed tomography (CT) angiography (CCTA) scans between 2018 and 2023. The study focused on comparing plaque progression and coronary inflammation between patients with and without prior SARS-CoV-2 infection.

The researchers analyzed 2,588 coronary artery lesions using a specialized imaging technique to measure percent atheroma volume (PAV) — a key indicator of plaque buildup. They assessed the annual change in total and non-calcified PAV, the presence of high-risk plaques (defined as those exhibiting at least two of the following characteristics: positive remodeling, low attenuation, and spotty calcifications), and changes in pericoronary adipose tissue (PCAT) attenuation, a key marker of coronary inflammation.

Notably, only patients who managed their SARS-CoV-2 infection without requiring hospitalization were included, ensuring that the observed effects were not due to severe acute illness. The lesions were classified based on their plaque composition into non-calcified, fibrous, and calcified categories.

The study also examined whether prior SARS-CoV-2 infection increased the likelihood of target lesion failure — a composite measure that includes cardiac death, myocardial infarction, and the need for revascularization. The statistical models used in the study accounted for confounding factors such as age, hypertension, diabetes, and lipid levels. Additionally, a causal mediation analysis was performed to assess whether coronary inflammation played a role in mediating plaque progression.

Results

The researchers found that individuals who had COVID-19 experienced more rapid plaque growth and higher inflammation levels compared to those without prior infection. The findings suggested that COVID-19 may trigger long-term cardiovascular changes, increasing the risk of future heart-related complications.

SARS-CoV-2 infections were found to significantly accelerate the progression of atherosclerotic plaques in coronary arteries. Compared to individuals without prior infection, those who had COVID-19 showed a faster annual increase in total PAV (0.90% vs. 0.62%) and noncalcified PAV (0.78% vs. 0.42%). In contrast, calcified plaque progression was slower in COVID-19 patients (0.12% per year vs. 0.20% per year), suggesting that SARS-CoV-2 infection primarily accelerates the growth of non-calcified, more vulnerable plaques.

They were also more likely to develop high-risk plaques (21.0% vs. 15.8%), particularly those with positive remodeling and low attenuation, which are associated with an increased risk of rupture.

Furthermore, the levels of coronary inflammation, as indicated by elevated PCAT attenuation (above -70.1 Hounsfield units), were also higher in individuals with prior SARS-CoV-2 infections than in those without (27.1% vs. 19.8%). Importantly, causal mediation analysis revealed that coronary inflammation accounted for approximately 10.3% of the total plaque volume increase and 5.7% of the noncalcified plaque progression, indicating that inflammation may partially drive these cardiovascular changes.

Recovered COVID-19 patients also had a 10.4% incidence of target lesion failure, which was greater than the 3.1% observed in individuals without prior COVID-19 and suggested that they were nearly three times more likely to experience severe cardiovascular events such as heart attacks and the need for surgical intervention.

The study also explored potential mechanisms behind these effects, finding that persistent low-grade inflammation, even after mild COVID-19, may contribute to worsening heart health. This suggests that the cardiovascular risks associated with COVID-19 are not necessarily confined to individuals with pre-existing conditions but could extend to a broader population.

Limitations

The study had some limitations, including its retrospective design and relatively short follow-up period (median of 9 months), which indicated the need for more research to determine whether these changes are reversible and how best to mitigate long-term cardiovascular risks in post-COVID patients. Additionally, the study was conducted at a single center in China, which may limit generalizability to other populations. The researchers also acknowledged that factors such as reinfection risk, vaccination status, and behavioral differences (e.g., medication adherence) could not be fully accounted for.

Conclusion

Overall, the study added to the growing body of evidence linking COVID-19 to long-term cardiovascular risks. Even in non-hospitalized patients, the virus appears to accelerate atherosclerosis and increase inflammation, raising concerns about future heart complications.

These findings highlighted the importance of post-COVID health monitoring, early cardiovascular interventions, and strategies to manage persistent inflammation to reduce long-term risks. The researchers believe that further research is needed to develop strategies for mitigating these effects and protecting heart health.

Source:

https://www.news-medical.net/news/20250205/COVID-19-speeds-up-artery-plaque-growth-raising-heart-disease-risk.aspx