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DIAGNOSTIC LIMITATIONS OF D-DIMER IN PULMONARY EMBOLISM AMONG COVID-19 PATIENTS: A COMPARATIVE ANALYSIS OF 146 CASES

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Relevance. A key diagnostic method for **Pulmonary Embolism (PE)** is **D-Dimer.** However, a number of illnesses other than PE, such as infections, inflammation, and cancer, can raise D-dimer. Even in the absence of thrombosis, COVID-19 causes a severe hypercoagulable and pro-inflammatory condition that raises D-dimer levels. This poses a crucial question: Does the infection itself result in false-positive increases, or can we rely on D-dimer as the primary diagnostic technique for PE in COVID-19 patients?

Aim: to prove that D-dimer is not a valid primary diagnostic tool for pulmonary embolism since it can be markedly raised by other causes; in particular, to show that COVID-19 infection alone results in D-dimer levels that are identical to those in patients with confirmed PE.

Materials and methods. One hundred and forty-six covid patients were analyzed and divided into two groups – pulmonary embolism positive and pulmonary embolism negative using MS Excel.

Results and their discussion. Both groups had similarly high median D dimer levels. The median D dimer level in the pulmonary embolism positive group was 1484 ng/mL. The median D dimer was 1022 ng/mL in the pulmonary embolism negative group. The two groups did not differ in a way that was statistically significant. Remarkably, 80% of individuals without pulmonary embolism had D dimer levels higher than diagnostic cutoff of 500 ng/mL. D dimer levels that would normally prompt additional testing for pulmonary embolism were seen in almost all patients without pulmonary embolism. Therefore, D dimer increases caused by COVID-19 infection alone fully overlapped with those in patients who really experienced a pulmonary embolism. As a result, D dimer was unable to distinguish between the two groups. Wells scores, on the other hand, varied considerably between the groups, with the pulmonary embolism positive group having higher values. Eight patients in the pulmonary embolism positive group and eight in the pulmonary embolism negative group died, indicating a similar mortality rate between the two groups.

Conclusions. Even in COVID-19 patients without pulmonary embolism, D dimer is markedly high; 80% of these patients have levels over the typical diagnostic cutoff of 500 ng/mL. There is no discernible difference in D dimer levels between COVID-19 patients with and without pulmonary embolism, indicating that D dimer cannot accurately differentiate between elevation caused by infection and actual thrombosis. D dimer is often raised by other reasons, such as COVID-19 infection, making it an unreliable main diagnostic tool for pulmonary embolism. Rather than relying solely on isolated D dimer thresholds, diagnostic imaging judgements should be guided by clinical probability assessment utilising the Wells score.