

# Use of a Non-Invasive Pulmonary Gas-Exchange Analyzer to Improve the Pretest Probability of Pulmonary Embolism in a Patient Classified as “Low Risk”

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## Introduction

Pulmonary embolism (PE) results in impaired gas transfer of oxygen and carbon dioxide across the affected region of the lung due to ventilation and perfusion mismatch caused by reduced perfusion (D'Angelo, 1997). PE is one of the leading causes of death in the US and the third leading cause of death in hospitalized patients (Ouellette & Patocka, 2012). PE is difficult to diagnose due to a wide spectrum of medical presentations and does not have a defining historical feature, physical examination finding, laboratory test, or diagnostic modality that can independently and confidently exclude its possibility (Stein et al., 2007). Given the possibility of asymptomatic and atypical presentation, it is generally accepted that many cases of PE go undiagnosed (Ouellette & Patocka, 2012). The Wells Score is used to determine the pretest probability of patients having pulmonary embolism and is scored from low (<2) to high (>6). A negative D-Dimer in the setting of Well's Score of 0-1.5 has good sensitivity and negative predictive value to rule out a PE. However, there are numerous causes to have a positive D-Dimer, including advanced age. Given the risk of a “false” positive, it is common practice to order D-Dimer tests judiciously to avoid more invasive investigations including Computed Tomography (CT).

Therefore, there is a need for adjunct tools to enhance recognition of PE.

## Patient Case

An 81-year-old male presented to the emergency department with a four-week history of shortness of breath.

He was alert and oriented with a GCS of 15.

Medical history included myocardial infarction with placement of a cardiac stent.

Patient presentation and medical history suggested cardiac ischemia.

Non-invasive pulmonary gas-exchange analysis was conducted.

Due to the high O<sub>2</sub> Deficit, PE was suspected, and the patient was sent for a CT scan which revealed prominent thrombotic load within the lobar and segmental pulmonary arteries.

## Clinical Testing

Given this patient's presentation and medical history an Acute Coronary Syndrome (ACS) / non-ST Elevation Myocardial Infarction (NSTEMI) or possible missed “silent MI” would need to be considered among other diagnosis including, but not limited to, anemia, upper respiratory tract infection (including COVID-19 infection), malignancy, and PE.

A cardiac panel and routine bloodwork, including cardiac enzymes, was ordered.

The initial 12 lead EKG revealed slight T-wave inversion, not present a year previous.



The T-wave inversion was suspicious for cardiac ischemia, but not infarction. Because of his cardiac history, an Acute Coronary Syndrome diagnosis could not be ruled out. Subsequent positive cardiac troponin enzymes of 0.169 ug/l, chest x-ray, and a normal hemoglobin supported the diagnosis of an ACS / NSTEMI.



Test	Result
D-Dimer	6573 ng/mL
Troponin I	0.169 µg/L

## Differential Diagnosis

With respect to PE as a differential diagnosis one must consider the clinical picture and incorporate probability assessment scores such as the Well's Criteria for PE.

After assessment, it was found that the patients Well's score was 0. If the patients use of a cane/walked would predispose him to the possibility of an “immobility” risk factor that would give him a Well's score of 1.5, which still makes the diagnosis of PE unlikely. A negative D-Dimer in the setting of Well's Score of 0-1.5 has good sensitivity and negative predictive value to rule out a PE.

However, there are numerous causes to have a positive D-Dimer, including advanced age, among others. Given the risk of a “false” positive, it is common practice to order D-Dimer tests judiciously to avoid more invasive investigations including CT scans that include intravenous contrast and may be harmful to the patient.

## Gas-Exchange Analysis

A non-invasive pulmonary gas-exchange analyzer (MediPines AGM100™) was used to administer a non-invasive pulmonary gas-exchange analysis while waiting for the patient's bloodwork results to be available.

The non-invasive pulmonary gas-exchange analysis showed a large O<sub>2</sub> Deficit (A-a gradient) of 65 mmHg, pPaO<sub>2</sub> of 60 mmHg, ETCO<sub>2</sub> of 19 mmHg, respiratory rate of 29, and SpO<sub>2</sub> of 94%.



## Discussion

This patient presented with an O<sub>2</sub> Deficit of 65 mmHg, a high level of gas-exchange impairment, which gave evidence that the diagnosis of ACS / NSTEMI was not as plausible and further investigation was needed to determine the cause of gas-exchange impairment and shortness of breath.

In this case, O<sub>2</sub> Deficit was used as a respiratory marker in conjunction with the clinical presentation and physical judgement.

Having an objective measure of pulmonary gas exchange impairment can factor into the diagnosis and treatment decisions that must be made in a time sensitive manner.

This is the first reported case of a non-invasive pulmonary gas-exchange analysis being used to improve pre-test probability of PE.

## Conclusion

The AGM100™ demonstrated to be a helpful adjunct tool in the diagnosis of PE and is ideally suited because of the quick and precise measurements it provides while being non-invasive and easy to use. The O<sub>2</sub> Deficit measurement can be highly informative in scenarios similar to this case and more research is needed to fully understand its potential in improving the probability of identifying PE in patients classified as “Low Risk”.

## References

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