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ORIGINAL ARTICLE

Combined Echocardiographic and Laboratory Indices as Predictors of In-hospital outcome of Acute Pulmonary Embolism

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ABSTRACT

Background: Progression of normotensive patients with acute pulmonary embolism (APE) to hemodynamic instability is thought to be associated with Mechanical and Inflammatory changes. **Objective:** To establish the ability of some new Echocardiographic and Inflammatory indices to predict the outcome of normotensive APE patients in an In-hospital setting. **Patients and Methods:** This prospective cohort study included 62 normotensive patients were diagnosed as APE. Six parameters were recorded for these patients: left ventricular outflow tract-velocity time integral (LVOT-VTI); Right/Left ventricle (RV/LV ratio); right atrium volume index (RAVI); neutrophil-lymphocyte ratio (NLR); platelet-lymphocyte ratio (NLR); and Troponin. All patients were followed up during hospital stay for development of adverse events. According to outcome they were divided into two groups, and the indices were compared in both groups. **Results:** The mean age of patients with AE (n=11) was 65.82y (p=0.062). The mean age of patients without AE (n=51) was 57.24y (p=0.062).There were a positive association between AE and NLR, PLR, RAVI, Troponin and RV/LV Ratio. There were a negative correlation between AE and LVOT VTI. Multiple logistic regression demonstrated that RAVI, NLR, PLR, Rv/Lv Ratio, LVOT VTI and troponin levels could be used as predictors for AE.
Conclusion: LVOT VTI, RV/LV basal diameter ratio, RAVI, NLR, PLR and troponin are all useful tools independently or combined to identify normotensive patients with APE who are at risk of developing in-hospital poor outcome.

Keywords: Pulmonary Embolism; Left Ventricular Outflow Tract; Velocity Time Integral

INTRODUCTION

Pulmonary embolism (PE), initially was reported in the 1980s, can be categorised as central or peripheral, submassive (25–50% obstruction) or massive (>50% obstruction) **[1].** Pulmonary vascular resistance rises dramatically in response to an acute pulmonary embolism. This causes harm to the right ventricle's systolic function and eventual right ventricular collapse. Hypoxia intensifies this vicious cycle of cardiogenic shock, which ultimately results in cardiovascular collapse **[2].**

The time between the beginning of symptoms and mortality is really short. After symptoms appear, 50% of patients with major pulmonary embolism pass away in 30 minutes, 70% in an hour, and over 85% in six hours **[3].** However, thrombogenesis, inflammation, abnormal hemodynamics, and endothelial cell dysfunction are all part of the pathogenesis of PE. These procedures influence how PE develops, progresses, and terminates **[4].** Activation of leukocytes, platelets, and endothelial cells leads to the generation of inflammatory cytokines and activates the coagulation system, which is most likely the cause of thrombosis **[5].** Furthermore, it has been demonstrated that having evidence of a documented embolism can also cause inflammation **[6].** Thrombosis and inflammation have a strong relationship and can trigger each other's activation **[7].**

Patients in the intermediate risk group for PE appear to have stable hemodynamics. But 10% of them go on to acquire significant PE, which has a high death rate. The range of short-term fatality rates for patients at intermediate risk is 2-10% **[8].** It is difficult to predict which intermediate-risk patients will have problems and need more intensive treatment.

Echocardiography is a useful tool for detecting acute RV pressure overload and RVD, despite the complicated anatomy of the RV. There is still debate over the relationship between different RVD echocardiographic characteristics and poor short-term outcomes in PE patients. Mortality rates for RVD, as determined by various echo parameters, range from 4.3% to 16.4% **[9].**

A measure of LV stroke volume called low left ventricular outflow tract velocity-time integral (LVOT VTI) has been demonstrated to predict adverse outcomes in individuals with acute PE **[10].** LVOT VTI is less prone to error than measuring the LVOT area and can be a more accurate estimate of cardiac output than ejection fraction when there are no anomalies in the LVOT **[11].**

Conversely, a study suggests that RA dilation is a finding that is significant to prognosis in patients with a range of cardiopulmonary disorder **[12].**

In terms of imaging, a study discovered that in patients who had a total hip prosthesis, NLR can considerably predict VTE. These results all emphasize the significance of inflammatory markers derived from hematology in the physiopathology of VTE **[13].** Remarkably, elevated cardiac troponins in PE were linked to the course and outcome of the illness rather than the diagnosis **[14].**

In this study we combined those Echocardiographic and inflammatory parameters (RV/LV basal diameter ratio, LVOT-VTI, right atrial volume indexed, NLR, PLR and troponin) to help predict progression and outcome of normotensive patients with acute PE in an in-hospital setting.

METHODS

Eighty patients who were consecutively admitted to the cardiology department, faculty of medicine, Zagazig university hospitals, and Alahrar Teaching Hospital with an acute PE diagnosis were included in this prospective cohort study.

Inclusion criteria

The diagnosis of acute PE was confirmed by computed tomographic pulmonary angiography (CTPA) with contrast enhancement. Only those with a systolic blood pressure (BP) of at least 90 mmHg and no hemodynamic assistance were eligible to participate in this trial.

Exclusion criteria:

Patients suffering from moderate-to-severe aortic regurgitation with dynamic LVOT blockage, hypovolemia, sepsis, and hypertrophic obstructive cardiomyopathy. Individuals with active cancer, inflammatory and autoimmune diseases, inadequately traceable LVOT VTI, left ventricular outflow tract stenosis, poor echocardiographic data, and persistent pulmonary hypertension are also excluded.

Ethical Consideration:

An approval of the study was obtained from Zagazig University Academic and Ethical Committee. Written informed consent of all the participants was obtained. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Every patient was observed for:

- **Complete history taking:** Factors such as age, history of heart attack, high blood pressure, diabetes, and other cardiac diseases were recorded.
- **Clinical evaluation:** Measurements of BP, HR, basal rales, and body mass index.
- **Laboratory investigations:** cardiac enzymes (troponin, CK-MB), Complete blood count was taken on admission.
- **Twelve lead surface ECG**.
- **Echocardiography:** a full study was done to each patient focusing on:

The right ventricle's (RV) dilatation was assessed in the apical four chamber view by measuring the LV and RV end-diastolic diameters at the level of the mitral and tricuspid valve tips. The RV/LV ratio was calculated (RV/LV ratio > 1 was considered high).

The boundary of the Doppler spectrum of the LVOT systolic flow was traced from the apical five- or three-chamber view using pulsed-wave Doppler. Inside the LVOT, around one centimeter below the aortic valve, was where the sample volume was placed. To obtain the best VTI with the least degree of spectral widening, the subaortic flow was aligned with the pulsed-wave Doppler sample volume **[15].** Patients with atrial fibrillation had an average of five VTI seconds because of beat-to-beat fluctuation in VTI. Generally speaking, an LVOT VTI of less than 15 cm is regarded as low.

Measurements of RA were assessed from the apical 4-chamber viewpoint. The RA area was measured

using planimetry at the end of ventricular systole (maximum volume), tracking the RA endocardium from the septal aspect to the lateral aspect of the tricuspid annulus.

All patients received treatment in compliance with ESC guidelines, and during their hospital stay, they were kept an eye out for the following adverse events:

- **Death:** In the absence of a different diagnosis or soon after a clinically severe PE, a death attributable to PE was confirmed.
- **Cardiac arrest:** The term "cardiac arrest" refers to the condition in which cardiopulmonary resuscitation is required.
- **The development of hemodynamic instability**: cardiogenic shock or the need for systemic rescue thrombolysis, was defined as the occurrence of pressor support-related or sustained reduction of systolic blood pressure < 90 mmHg for at least 15 minutes, or a systolic blood pressure drop ≥ 40 mmHg for more than 15 minutes that was not brought on by sepsis, hypovolemic, or new-onset arrhythmia.
- **The need for systemic rescue thrombolysis**: was based upon the attending physician's or the on-call physician's recommendation and it was characterized as an escalation of therapy due to hemodynamic instability.

All patient echocardiographic data and laboratory indicators were compared between patients who experienced adverse events and those that did not. Multivariate analysis was used to assess the predictive value of the parameters that showed a significant difference between the groups.

STATAITICAL ANALYSIS

Data collected and analyzed using Microsoft Excel software. Data were then imported into Statistical Package for the Social Sciences (SPSS version 20.0) software for analysis. According to the type of data qualitative represent as number and percentage, quantitative continues group represent by mean± SD. Differences between quantitative independent multiple by ANOVA. P value was set at <0.05 for significant results &<0.001 for high significant result.

RESULTS

Several 84 patients were admitted with the diagnosis of acute pulmonary embolism. 80 normotensive patients were included and after final evaluation and application of exclusion criteria, 62 Patients were included in our study **(Figure 1)**.

Echocardiographic parameters (RV/LV basal diameter ratio, LVOT VTI and RAVI) as well as laboratory parameters (NLR, PLR and Troponin) were directly recorded in 62 normotensive patients with acute PE. And every patient was followed up during hospital stay for the development of adverse events.

They were further classified into two groups according to development of adverse events during hospital stay:

Group 1: 11 patients who developed adverse outcome which was one or more of the following:

-Hemodynamic instability
-Need of rescue thrombolysis
-In-hospital death
-Resuscitated cardiac arrest

Adverse events were present in 11 patients (17.74%) as follows:

- 11 patients developed hemodynamic instability (17.74%).
- 11 patients required rescue thrombolysis (17.74%).
- PE related death was registered in 2 patients (3.22%).
- Resuscitated cardiac arrest occurred in 3 patients (4.83%).

Group 2: 51 patients who didn't develop any of the mentioned adverse events (82.26%).

An adverse outcome (including hemodynamic instability, need for rescue thrombolysis, in-hospital death, and cardiac arrest). Of these, 11 patients (17.74%) experienced hemodynamic instability, and all of them (17.74%) required rescue thrombolysis. Two patients (6.45%) died from PE-related causes, and three patients (4.83%) experienced resuscitated cardiac arrest **(Table 1)**.

In 62 normotensive patients with acute PE, the echocardiographic parameters RV/LV ratio, LVOT VTI, and RAVI as well as the laboratory parameters NLR, PLR, and Troponin to predict adverse events related to PE, such as death, cardiac arrest, hemodynamic instability, and the need for rescue thrombolysis while hospitalized, were directly compared **(Table 2)**.

As presented, the 6 parameters proved to have statistically significant value in comparing both groups **(Table 3)**. Multiple logistic regressions demonstrated that RAVI, NLR, PLR, RV/LV Ratio, LVOT VTI and troponin levels could be used as predictors for AE (**Table 4**).

RV/LV ratio >1 was the most specific parameter to expect adverse outcome with a specificity of 88.24% (AUC was 0.840 with 95% CI 0.724 to 0.920, p <0.0001) **(Figure 2).**

NLR was the highest sensitive parameter with sensitivity of 100% and relatively low specificity

(74.5%). AUC = 0.840 (95% CI 0.793 to 0.960, *p*< .0001) **(Figure 3).**

Table.1: Baseline characters and incidence of adverse events in included patients

	Mean	SD
Age (years)	58.76	12.638
Gender	**N**	**%**
Male	35	56.5
Female	27	43.5
Adverse Events (Ae)	11	17.7
In-Hospital Death	2	3.2
Resuscitated Cardiac Arrest	3	4.8
Hemodynamic Instability	11	17.7
Rescue Thrombolysis	11	17.7

N=Number ; %= Percentage; Ae = adverse events

Table (2): Echocardiography and laboratory parameters among the studied patients

	Total (N=62)	**Without AE (N=51)**	**With AE (N=11)**	**P value**
Rv/Lv Ratio	0.85 [0.7875, 1.0175]	0.83 [0.77, 0.97]	1.07 [1.04, 1.2]	.011
LVOT VTI	17.635 [14.903, 19.193]	18 [16.18, 19.54]	14.5 [13.6, 17.6]	.000
RAVI	27.76 [23.103, 33.568]	26.91 [22.2, 29.77]	35.99 [31.76, 57.89]	.000
Laboratory examination:				
NLR	2.815[1.87, 4.28]	2.47 [1.79, 3.63]	4.75 [3.75, 5.8]	.000
PLR	121.11[98.913, 182.84]	105.88 [97.13, 162.72]	193.25 [158.67, 207.5]	.001
Troponin	0.021[0.01, 0.0708]	0.018 [0.01, 0.034]	0.52 [0.06, 0.815]	.000

Data were represented as median(IQR), unless otherwise mentioned.
RV= right ventricle; LV=left ventricle. LVOT-VIT=left ventricular outflow tract-velocity time integral; RAVI= right atrium volume index; NLR= neutrophil-lymphocyte ratio; PLR= platelet-lymphocyte ratio

Table (3): Risk factors for AE among the studied patients

	B	**S.E.**	**Exp(B)**	**95% C.I for EXP(B)**		**P value**
RAVI	**0.099**	**0.042**	**1.104**	**1.017**	**1.197**	**0.017**
NLR	**1.226**	**0.415**	**3.407**	**1.511**	**7.684**	**0.003**
PLR	**0.022**	**0.007**	**1.023**	**1.008**	**1.038**	**0.003**
Rv/Lv Ratio	**5.792**	**2.533**	**327.799**	**2.288**	**46964.045**	**0.022**
LVOT VTI	**-0.781**	**.328**	**.458**	**.241**	**.871**	**0.017**
Troponin	**3.260**	**1.262**	**26.050**	**2.198**	**308.791**	**0.000**

*PE: Pulmonary embolism; RAVI= right atrium volume index; NLR= neutrophil-lymphocyte ratio; PLR= platelet-lymphocyte ratio**;** LVOT VTI: low left ventricular outflow tract velocity-time integral;*

Table (4): Accuracy of predictors with AE among the studied patients

Adverse outcomes	AUC	95% CI	Cut-off	Sensitivity	Specificity	Youden index J	P value
LVOT-VTI	0.746	0.619 to 0.848	≤14.95	72.73%	84.3%	0.5704	0.0043
RAVI	0.840	0.724 to 0.92	>31.48	81.82%	84.31%	0.6613	<0.0001
PLR	0.834	0.718 to 0.917	>147.19	90.91%	74.51%	0.6542	<0.0001
Troponin	0.891	0.786 to 0.956	>0.059	90.91	80.39	0.7130	<0.0001

LVOT VTI: low left ventricular outflow tract velocity-time integral; RAVI= right atrium volume index; PLR= platelet-lymphocyte ratio

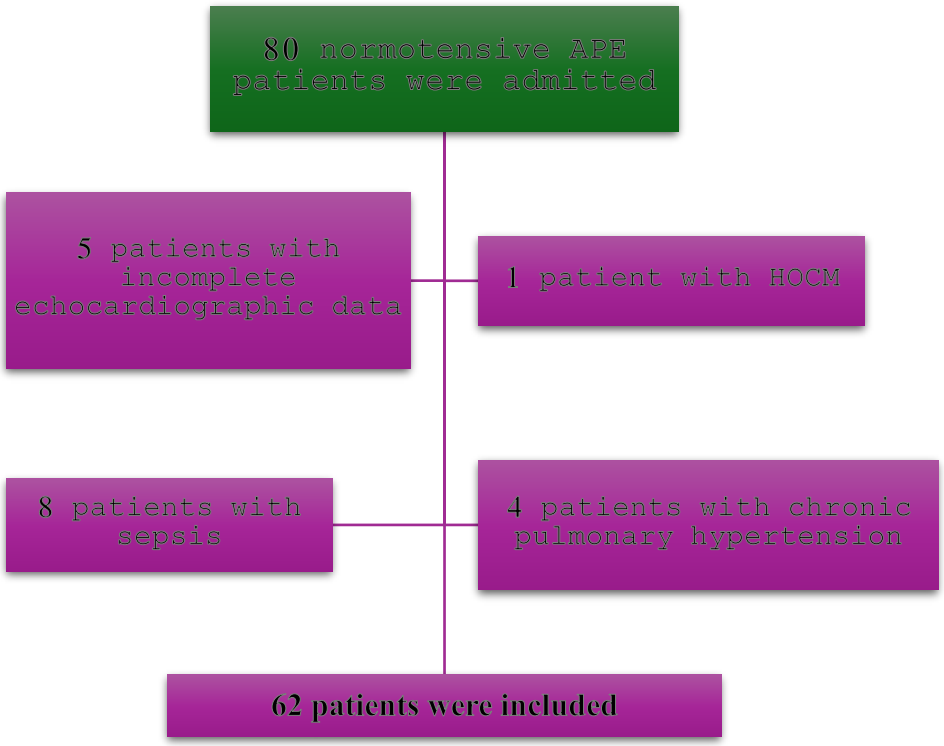

Figure (1): Study flow diagram of the studied patients (*APE= Acute pulmonary embolism; HOCM= hypertrophic obstructive cardiomyopathy*)

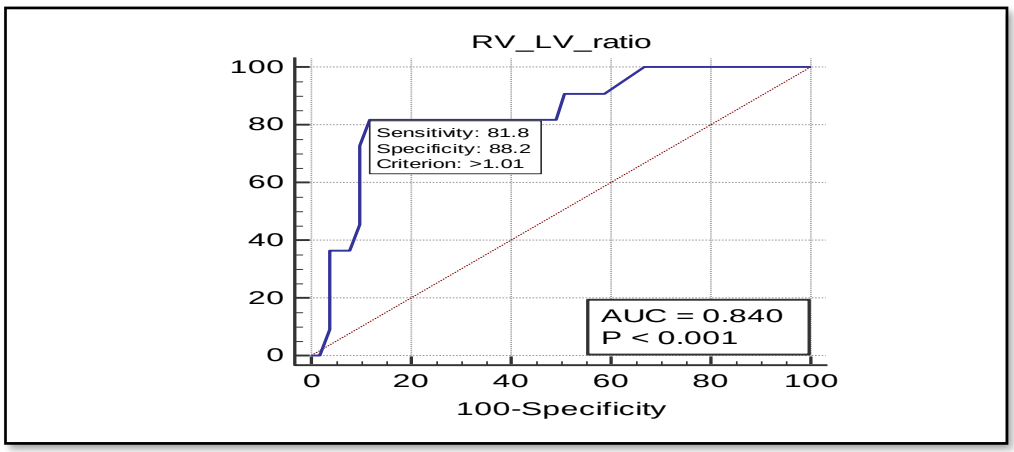

Figure (2): ROC curve for the accuracy of RV/LV ratio in the predication of adverse events (*AUC= Area under the curve).*

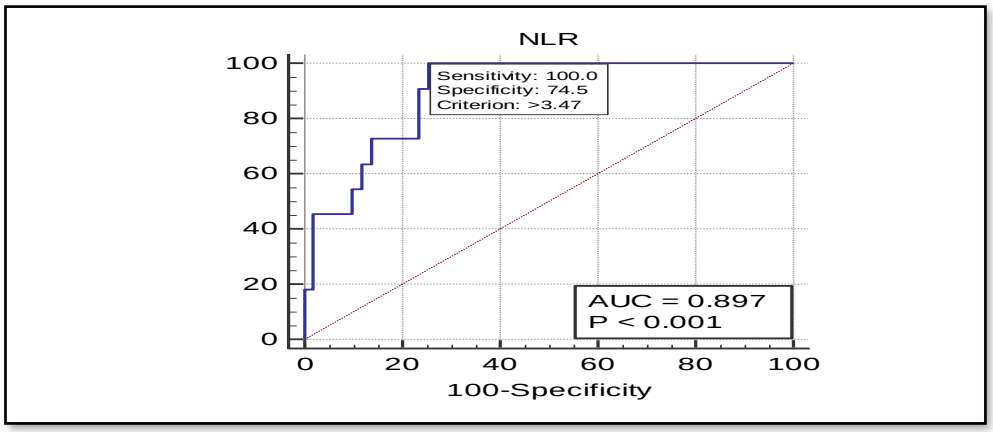

Figure (3): ROC curve for the accuracy of NLR in the prediction of adverse events (*AUC= Area under the curve).*

DISCUSSION

The present study showed that some echocardiographic and laboratory parameters could strongly predict acute pulmonary embolism course and outcome.

To our knowledge, this is the first study to combine both inflammatory and mechanical obstruction pathophysiology and correlate parameters of both mechanisms in order to predict in-hospital outcome of pulmonary embolism and normal blood pressure on admission. The main results of this investigation revealed six indices that may independently predict the unfavorable in-hospital prognosis of an acute pulmonary embolism.

The optimal echocardiographic markers that can indicate unfavorable outcomes are still up for debate, even though it is widely known that RVD impacts the short-term prognosis. It's still difficult to categories the risk of hemodynamically stable PE patients. If RVD is present in normotensive individuals with PE, there is a predicted increased risk of hemodynamic compensation **[16].** Mortality rates ranging from 4.3% to 16.4% are also linked to RVD diagnosed using distinct echo parameters **[17].**

A higher RV to LV ratio was linked to short-term negative outcomes such as cardiac arrest, death, hemodynamic decline, and the requirement for rescue thrombolysis. After multivariate analysis in our study, a ratio of ≥ 1 continued to be an independent predictor of in-hospital complications with an HR of 4.1. AUC of 0.840 (95% CI 0.724 – 0.920 p = 0.022) with 81.8% sensitivity and 88.2% specificity was revealed by ROC analysis.

Cardiac output can still be measured with the 2D Simpson method; however, LVOT VTI seems to be more accurate and can be used as a substitute for the stroke volume. A number ≤ 15 cm denotes a reduced left ventricular stroke volume, whereas a value > 18 cm denotes an appropriate stroke volume **[18].**

According to **Yuriditsky et al. [19]** patients at intermediate-high risk exhibited low VTI in 58%

of cases. These findings guided risk stratification and management. Low LVOT VTI was linked to a poor outcome in PE patients and demonstrated a prognostic value in patients with intermediate risk PE. The entire group's in-hospital death or cardiac arrest (HR 6.95, 95% CI 2–17.9, p = 0.0014) and shock or the need for reperfusion (HR 23.3, 95% CI 6.6–82.1, p < 0.0001) were associated with a low LVOT VTI. Low LVOT VTI was a better indicator of shock than cardiac arrest or death.

In our investigation, comparable outcomes were noted. An independent predictor of unfavorable hospital outcomes (such as death, cardiac arrest, development of cardiogenic shock, and requirement for rescue thrombolysis) was LVOT VTI ≤ 15 cm. The results of the ROC analysis indicated an AUC of 0.746, good sensitivity (72.7%), good specificity (84.3%), and good accuracy in predicting an unfavorable outcome.

Previous study has shown a correlation between the mean pulmonary artery pressure and the degree of pulmonary embolic obstruction and the mean RA pressure **[20].**

An analysis conducted recently on 1640 consecutive TTE patients revealed a correlation between larger RV/RA pressure gradients and higher RA dilatation **[21].**

A study including 636 PE patients found that patients with a RA/LA volume ratio >1.2 on CTPA had a greater 30-day death risk **[22].**

In the present study, patients with an in-hospital adverse outcome had larger RA volumes on Echocardiography and a calculated patient-cohort optimized RA volume indexed cut-off value of 31.48 ml/m^2 was able to predict an in-hospital adverse outcome.

There is now strong evidence linking the pathophysiology of venous thromboembolism (VTE) to the advancement of thrombosis through tissue factors generated by inflammatory cytokines **[23].**This correlation can be found in a number of cardiovascular conditions, such as coronaries **[24].**

In the current study NLR was the most sensitive parameter to expect AE with a sensitivity of 100% however, its specificity was relatively lower in comparison with LVOT VTI (74.51% specificity). The cut off value of NLR was >3.47 with (95CI 0.793 to 0.960, p< .0001). On the other hand, PLR showed sensitivity of 90.91% and specificity of 74.51% with (AUC=0.834, 95CI 0.718 to 0.917, p=0.0001). The cut-off value for AE was >147.19. Among both groups there was significant difference in the mean value of both NLR and PLR, where mean NLR for AE group was 4.75 while being 2.47 in the group without adverse events. Regarding PLR, the mean value in AE group was 193.25 and in the group with no adverse events it was 105.88.

Despite diagnosis, the course and prognosis of PE were related to increased cardiac troponins **[25].**When comparing sub-massive PE patients to non-massive PE patients, higher cTnI levels were identified.

Similarly, **Kucher et al. [26]** found that compared to patients with non-massive cases, those with big PE had higher cTnI levels. Elevated levels of troponin are thought to be caused by mechanical damage instead of to an inflammatory reaction **[27-28].**

A combination of the aforementioned measures demonstrated a higher predictive value for bad in-hospital outcomes associated with acute pulmonary embolism in normotensive individuals.

CONCLUSION

Our results suggest that LVOT VTI, RV/LV basal diameter ratio, RAVI, NLR, PLR and troponin are all useful tools independently or combined to identify normotensive patients with APE who are at risk of developing in-hospital poor outcome.

Further studies on a larger scale should be performed to establish the accurate relationship of these parameters and outcome and possibility of a change of management plan in these patients.

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