REVIEW OF RESEARCH
ISSN: 2249-894X
IMPACT FACTOR : 5.7631(UIF)
UGC APPROVED JOURNAL NO. 48514
VOLUME - 8 | ISSUE - 9 | JUNE - 2019

GEO EPIDEMIOLOGY OF PULMONARY EMBOLISM: A REVIEW

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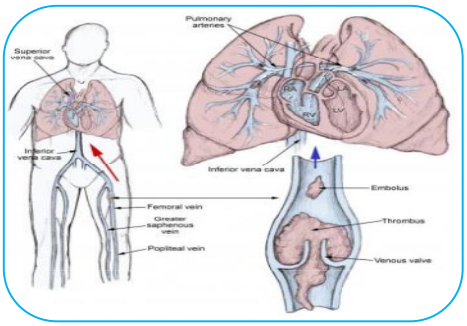
ABSTRACT:



Pulmonary embolism "(PE) is a common and life-threatening form of venous thromboembolic disease. It is the third most common cause of cardiovascular death and is associated with multiple inherited and acquired risk factors, as well as advanced age. 90% of pulmonary emboli (PE) are derived from deep venous thrombi of the lower extremities. Pulmonary embolism is the third most common cardiovascular disease. The most common symptoms of PE include: dyspnea, chest pain, cough, syncope, and hemoptysis. PE is classified as high risk (massive), intermediate risk (submissive), or low risk. Catheter-based interventions are used if systemic therapy has failed or is contraindicated. The prognosis of PE depends on the degree of obstruction and the hemodynamic effects of PE, and an understanding of the pathophysiology helps patients stratify risk and determine treatment. Although the natural history of thrombus is resolution, a subset of patients has a chronic residual thrombus, which contributes to post-PE syndrome. The clinical presentation of acute PE varies from asymptomatic and discovered by chance to massive PE causing immediate death. This review focuses on the epidemiology, risk factors, pathophysiology, and natural history of PE. It is clearly impossible to present data on the geo-epidemiology of all pulmonary embolisms. Instead, we try to generate interest among immunologists to generate articles that are stimulating, but also contemporary reviews. "

KEYWORDS: *Pulmonary embolism (PE) , inherited and acquired risk factors.*

1. OVERVIEW

Pulmonary embolism " (PE) and deep vein thrombosis (DVT) can be considered on the spectrum of venous thromboembolic disease (VTE). There are no definitive scientific data on the overall incidence of VTE in the general population, but a recent study estimates that the incidence is between 1 and 5 / 1,000 in the general population [1]. In the surgical population, the prevalence can reach more than 50% in the absence of thromboprophylaxis [1]. "

Worldwide, "more than 50% of all hospitalized patients are at risk for VTE and surgical patients are at higher risk than medical patients [2]. The incidence of PE represents 5 to 10% of hospital deaths, making this condition the most common preventable cause of hospital death [3-6]. Furthermore, VTE and associated complications contribute substantially to patient morbidity" and treatment costs [7, 8].

Within "the trauma and orthopedics discipline, the prevalence of DVT and PE has been estimated at 1.16% and 0.93% respectively [9]. Mortality

rates have been reported to vary between 0.38% and 13.8% [10, 11]. The main risk factors include an injury severity score (ISS) greater than 50 and more than two surgical procedures [9]. PE appears to be the most common cause of mortality in patients who survive the first 24 hours after trauma, and retrospective postmortem data showed that of an overall mortality of 13.8%, 1.6% was a consequence of death. PE [10]. In the elective orthopedic clinical setting, PE is the second most frequent cause of death in patients undergoing total joint arthroplasty of the lower extremity [11]. "

Despite "existing data reporting the overall prevalence of PE in the traumatic and orthopedic population, there is a common belief that since clinical signs and symptoms are nonspecific and often silent, " this complication can still be misdiagnosed [12].

This study has "several limitations, including the retrospective nature of data collection, case notes and electronic databases, small sample size, short study period, and the absence of a control group. Furthermore, our data set has documented events that occur only during the hospital stay (primary or readmission). We are aware that part of our study population may have been admitted or treated elsewhere, as the researchers treat several patients referred to the tertiary sector and, as such, we may have lost some patients who developed PE. "

RISK FACTORS

In "the mid-19th century, Rudolph Virchow identified the triad of risk factors that contribute to thrombosis: blood flow stasis, vascular endothelial damage, and hypercoagulability. All VTE risk factors reflect these underlying pathophysiological processes, and in general, patients experiencing VTE have at least one risk factor. Risk factors can be divided into hereditary and acquired factors: "

Inherited risk factors

- "Factor V Leiden"
- "Prothrombin gene mutation"
- "Antithrombin deficiency"
- "Protein C deficiency"
- "Protein S deficiency"

Acquired risk factors

- "Trauma"
- "Surgery"
- "Malignancy"
- "Peripartum state"
- "Estrogen therapy"
- "Aging"
- "Obesity"

PATHOGENESIS

The "emboli detach from their point of origin and travel through the systemic venous system, through the chambers on the right side of the heart, and lodge in the pulmonary arterial system. The physiological and clinical consequences of PE range from asymptomatic to hemodynamic collapse and death. PE contributes to gas exchange abnormalities and hypoxemia, but it is mainly the hemodynamic consequences of PE that are responsible for the increased morbidity and mortality. Understanding the pulmonary pathophysiology of PE is important in patients with risk stratification to determine treatment with anticoagulants themselves or to consider catheter-directed therapies (thrombolytics or mechanical thrombectomy), systemic thrombolytics, or surgery. "

HEMODYNAMICS

The "Cardiac and Hemodynamic effects refer to the size and location of emboli and the presence or absence of underlying cardiopulmonary disease (CPE). In contrast to clot loading, acute PE is

classified according to the hemodynamic effect, with special attention to the effects of the physiology of the right ventricle (RV). Non-massive PE patients are those who are normotensive with normal RV function. Massive PE involves hemodynamic instability due to RV failure, and patients with submaxive PE may be clinically normotensive, but have evidence of RV dysfunction by echocardiography or CT imaging. ”

NATURAL HISTORY

The prognosis for PE depends on “the degree of obstruction and the hemodynamic effects of PE. People with massive PE may have an imminent risk of death with an estimated mortality of 25 to 65%, people with submassive PE have a mortality of 3 to 15%, 58 while people with low-risk PE and normal heart function they have a mortality <1% with anticoagulation. The risk of recurrent VTE is estimated at 20-25% after 5 years in unselected cohorts and above 25% in those without a clear provocative cause. Relapse also increased in subjects with associated congenital or acquired risk factors. There are possible long-term consequences of PE regarding functional decline, especially for those with recurrent PE.”

2. REVIEW OF GEO EPIDEMIOLOGY OF PULMONARY EMBOLISM

The “incidence of PE is likely to increase due to over diagnosis, and although mortality is decreasing, PE remains a common and life-threatening form of VTE. Inherited and acquired risk factors (especially surgery and cancer) increase the likelihood of VTE and PE. ”

Based on current evidence, “CT-PA is considered the gold standard for diagnosing PE [13, 14]. Chest contrast enhanced CT has replaced catheter angiography due to its less invasive nature and precision and has been shown to be greater than or equal to angiography [14]. The reported sensitivity for diagnosing PE with CT-PA varies from 45 to 100% and the specificity from 78 to 100% [14]. CT-PA has some limitations in the detection of isolated sub-segmental PE [14], but the introduction of the multi-detector CT technique currently allows the evaluation of the pulmonary vessels up to the branches of the sixth order, which significantly increases the rate of PE detection (sensitivity: 83%, specificity: 96%) [15, 16]. ”

Many authors have “attempted to correlate specific risk factors with the development of PE. A strong correlation was identified for the number and extent of surgery, the previous history of VTE and the length of the hospitalization period [17]. The next highly reported risk factors for VTE are cardiovascular disease [18] and obesity [10, 12]. More than half of our study cohort belongs to the high risk category with more than two risk factors present as defined by the NICE guidelines [19]. ”

Although “79.3% of study population was on TP treatment, patients developed PE. Researcher were unable to identify additional specific factors related to the development of VTE, but we observed that in cohort 62.4% of patients were over 60 years old and 22.4% were over 80 years old. Several studies have reported age as an independent risk factor for VTE [1,]. In cohort, many of these elderly patients also had pathologies of the lower extremities (83.5%). A synergistic effect of these two parameters could be hypothesized to reduce mobility and lead to an increased risk of developing PE. In a recent crossover case study, reduced mobility was reported to be a significant trigger for VTE hospitalization. The risk of hospitalization for VTE was 4.2 times higher in the period when reduced mobility occurred [20]. ”

In 13 cases, “TP treatment was not prescribed even if the patients had risk factors for VTE. Eight of these 13 cases reported foot and ankle injuries, were managed non-operatively, and were followed up at outpatient fracture clinics. The reason for this can be attributed to the lack of clarity of national and local guidelines on the treatment of PE in the outpatient setting, particularly with the injury schemes that are considered less debilitating. Shibuya et al. [21] stated that the routine use of TP treatment in foot and ankle injuries is unwarranted in contrast to our findings, which support the view that even minor foot injuries cannot be overlooked and risk assessment it must be done individually. This led to the expansion of routine risk assessment of patients treated at our outpatient facilities, and regular audit cycles were implemented to ensure consistent compliance. ”

Concomitant "DVT was identified in one third of our study cohort. Knudson et al. [22] analysed the National Trauma Data Bank of the American College of Surgeons and found 522 cases of PE from 450,375 trauma patients (0.11%). Only in 16% of these cases was a concomitant DVT diagnosed. In a prospective cohort study [23] of 397 patients with clinical suspicion of PE, 149 were PE positive and less than a third had concomitant DVT. Onions et al. [24] performed a trauma registry analysis of 10,141 trauma admissions and found 30 cases of PE, of which only 5 (16.7%) had coexisting DVT. Furthermore, in a retrospective review of the medical records of 247 trauma patients who underwent TPA / CTV over a three-year period, Velmahos et al. [25] recognized positive PE results in 46 patients (19%) and of these, only 7 (15%) also had DVT. Assuming that CTPA / CTV was considered the most accurate method of diagnosing VTE, the same authors [25] studied this lack of association between PE and DVT. They said it was unlikely that a DVT diagnosis could have been significantly overlooked due to insufficient sensitivity of the diagnostic tool. Therefore, they hypothesized that clots could form de novo within the pulmonary circulation as a consequence of changes within the pulmonary vascular endothelium and in the rheological properties of blood induced by a posttraumatic hyper adrenergic and hyper inflammatory state. However, there is still no definitive evidence on the etiological relationship between DVT and PE, and it is desirable to carry out further studies to understand this phenomenon. "

Severe "Acute Respiratory Syndrome (SARS) has become a worldwide epidemic with a mortality of 9.2%. This new emerging infectious disease in humans is dominated by severe lower respiratory tract disease and is etiologically linked to a new coronavirus (SARS-CoV). It affects the respiratory system which causes the pulmonary embolism-artery blocks which results clot, oxygen deterioration in circulation it to artery heart brain etc. Pulmonary pathology and clinical correlates were studied in seven patients who died of SARS in whom there was a strong epidemiological link. Investigations include a review of clinical characteristics, morphological evaluation, histochemical and immunohistochemical staining, ultra structural study, and virological investigations in post mortem tissue. Positive coronavirus viral culture was detected in the majority of the premortem nasopharyngeal aspirate samples (five out of six) and post mortem lung tissue (two out of seven). Viral particles, consistent with the coronavirus, could be detected in lung pneumocytes in most patients. These characteristics suggest that pneumocytes are probably the main target of infection. The pathological characteristics were dominated by diffuse alveolar damage, with the presence of multinucleated pneumocytes. The proliferation of fibro granulation tissue in the small airways and air spaces (bronchiolitis obliterans that organizes pneumonia-like lesions) in sub pleural areas has also been observed in some patients [26]. "

After "the SARS epidemic, the World Health Organization reported that the disease generally attacked the lungs in three stages: viral replication, immune hyperactivity, and lung destruction. The COVID-19 epidemic killed more than 1,800 people, surpassing the number of deaths from SARS in a matter of weeks. While the death rate for COVID-19 appears to be a fifth of SARS, the new coronavirus has spread faster. The first 72,314 patients were diagnosed until February 11. The report shows that COVID-19 killed 2.3 percent of patients, which means it is currently 23 times more deadly than seasonal flu. Serious illness and death have been reported in all age groups, with the exception of children under the age of nine."

The "mortality was low and consistent with other news in the literature. The low mortality rate observed in the subgroup of trauma patients could be attributed to the high percentage of less severe traumatic injuries (frequency of patients with multiple injuries: 11.9%) and to the good compliance rate in the implementation of our TP treatment protocols. "

3. CONCLUSION

Pulmonary embolism " (PE) is caused by emboli, which originated from venous thrombi, which travel and occlude the pulmonary arteries. PE is the most dangerous form of venous thromboembolism, and undiagnosed or untreated PE can be fatal. Acute PE is associated with right ventricular dysfunction, which can cause arrhythmia, hemodynamic collapse, and shock. Furthermore, people who survive PE

can develop post-PE syndrome, which is characterized by chronic thrombotic debris in the pulmonary arteries, persistent right ventricular dysfunction, decreased quality of life, and / or chronic functional limitations. In recent years, several important improvements have been made in the diagnostic and therapeutic treatment of acute PE, such as the introduction of a simplified diagnostic algorithm for suspicious PE and phase III studies that demonstrate the value of direct oral anticoagulants in the Acute and extensive treatment of thromboembolism venous. "

It has been reported in "the international literature, while the mortality rate was considerably lower. Local protocols, according to NICE guidelines, appear to be effective in preventing VTE and reducing mortality in trauma and orthopedic patients. However, despite widespread administration of mechanical and chemical TP treatments, patients may still develop PE. It seems that the possibility of developing PE is not only related to certain risk factors related to the patient, but also with the consequence of all aspects of the post-traumatic or post-surgical disease process. Overall, the type of treatment, the type and duration of drug administration, the duration of immobilization, and the individual response of each patient appear to contribute to the development of this rare but terrifying complication. "

The "disease represents a new emerging infectious disease in humans dominated by a serious lower respiratory tract disease. SARS is etiologically linked to a new coronavirus (SARS-CoV). Most patients show chest X-ray abnormalities (CXR) at presentation, with irregular airspace disease. Covid 19-Coronavirus affect the respiratory system which causes the pulmonary embolism-artery blocks which results clot, oxygen deterioration in circulation it to artery heart brain etc."

Despite "continuous improvement in medical knowledge and treatment modalities, the incidence of VTE and its related complications has remained fairly static for the past three decades. Despite the implementation of prevention protocols, the clinical manifestation of PE is not clear or specific. PE can be expressed silently and the clinical team may lose it. For this reason, PE may be underdiagnosed. "

In addition to "the immediate morbidity and mortality caused by PE, post-PE functional limitation syndrome in association with cardiac dysfunction and continuous gas exchange has received more attention in recent years, and research is on-going to determine who is in increased risk. for these consequences especially in the submissive population with the question of the benefits of early intervention to reduce the burden of the clot. "

Future "research should point to new treatment options (e.g., fibrinolysis enhancers) and improved methods to predict long-term complications and define optimal parameters of anticoagulant therapy in individual patients and to gain a better understanding of post-PE syndrome."

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