Clinical Presentation of Pulmonary Embolus After Total Joint Arthroplasty: Do Size and Location of Embolus Matter?

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Abstract

Pulmonary embolism (PE) is a potentially fatal complication of total joint arthroplasty. Therefore, it is essential to have reliable means for diagnosis and evaluation of severity.

In the study reported here, we evaluated the reliability of common clinical signs and symptoms in the diagnosis of PE. In addition, we used correlation analysis to assess for a correlation between clinical presentation and size and location of the embolus within the pulmonary vasculature. Included in this study were 13,133 patients who underwent total joint arthroplasty between 2000 and 2005. PE was diagnosed in 144 patients (1.1%). Shortness of breath (31.9%) and hypotension (30.6%) were the most frequent symptom and sign. Oxygen desaturation was the only indication for investigation of PE in 10% of patients. A pulse-oximetry reading of less than 90% was present in 63% of patients, and 92% of patients presented with an increased alveolar-arterial gradient.

Overall, clinical signs and symptoms as well as severity of hypoxia did not correlate with size and location of PE. Patients with PE demonstrated a significant decrease in arterial oxygen content; an abnormal alveolar-arterial gradient was the most consistent finding in these patients.

Common clinical signs and symptoms, as well as changes in vital signs, have a low sensitivity for diagnosis.

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Patients who undergo major orthopedic surgical procedures in general, and total joint arthroplasty (TJA) in particular, are at higher risk for developing thromboembolic complications.[1-4] Although prophylactic anticoagulation has been effective in reducing the incidence of thromboembolism, severe consequences and even death secondary to pulmonary embolism (PE) are possible after TJA.[5,6]

There are many challenges in preventing thromboembolic problems after surgical procedures. One challenge is diagnosing thromboembolism and, in particular, PE. Diagnosis of PE is hindered by lack of specificity in clinical presentation. In addition, a wide spectrum of conditions, such as congestive heart failure, sleep apnea, and analgesia-induced hypoxia, can resemble PE in their presentation.

In the study reported here, we evaluated the reliability of common clinical signs and symptoms in the diagnosis of PE. We determined the rates of these signs and symptoms and examined the results of arterial blood gas analysis of patients diagnosed with PE after TJA. This study was also designed to test a null hypothesis—that severity of presentation of PE does not correlate with size and location of the embolus in the pulmonary vasculature.

Methods

Demographic Data

We reviewed prospectively collected data on patients who underwent TJA between 2000 and 2005. During that time, 14,890 TJAs were performed in 13,133 patients. Of these TJAs, 6,950 (5,695 patients) were knee arthroplasties, and 7,940 (7,438 patients) were hip arthroplasties. We identified 144 patients (70% women, 30% men) with a confirmed diagnosis of PE after TJA. Mean age was 69.6 years (range, 36.2-90.0 years). Mean weight and height were 90.1 kg (range, 41.7-167.6 kg) and 166.6 cm (range, 63.5-221.0 cm), respectively, corresponding to a mean body mass index of 31.9 kg/m^2 (range, 15.8-54.0 kg/m^2).

Arthroplasty Data

At our institution, patients who undergo hip or knee arthroplasty are given a medical workup and a preoperative medical optimization by an internist. Hypotensive

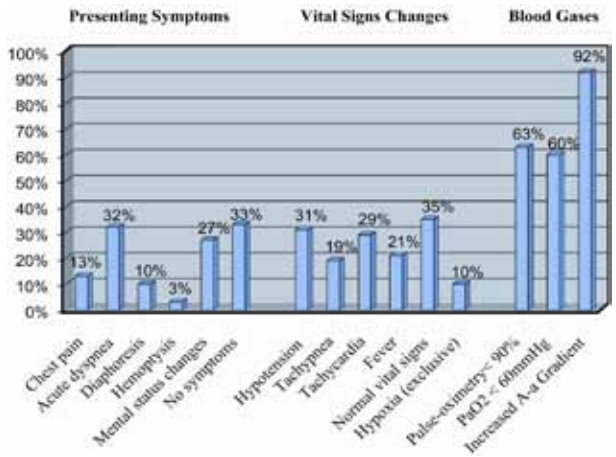

Figure. Clinical presentation of pulmonary embolism after total joint arthroplasty. Abbreviations: PaO_2, mean partial pressure of oxygen in arterial blood; A-a, alveolar-arterial.

regional anesthesia is used except when contraindicated. Hip arthroplasty is performed with the patient in the supine position using the anterolateral approach, and knee arthroplasty is performed under tourniquet using medial parapatellar arthrotomy. Uncemented prostheses are used for hip arthroplasty and cemented fixation for knee arthroplasty.

Perioperative Care

Our institution's postoperative protocol for joint arthroplasty patients includes administration of warfarin chemoprophylaxis with a goal international normalized ratio (INR) of 2.0. Warfarin is started on the evening of surgery and continued for 6 weeks. This strategy has been demonstrated to be effective for thromboembolic prophylaxis.[7] Patients also receive 1000 IU of intravenous heparin at time of hip dislocation or before tourniquet inflation for knee arthroplasty. The protocol also includes early mobilization and the use of continuous passive motion devices.

Clinical Evaluation

At our institution, patients are monitored closely during the postoperative period. Nursing surveillance with recording of pulse oximetry after TJA has become common practice. Hence, any drop in oxygen saturation tends to trigger medical consultations and possible investigations for PE. Symptoms, such as chest pain, shortness of breath, diaphoresis, and mental status change, are also considered serious events and are investigated. Abnormal clinical findings, such as tachypnea, tachycardia, hemoptysis, cough, and fever, which are known to be associated with PE,[8-10] also result in further evaluation with an electrocardiogram, chest radiograph, cardiac enzymes, arterial blood gas, blood chemistry, and electrolytes. When there is high suspicion for PE, an internist is consulted, and a further workup, including use of multidetector computed tomography (MDCT), is performed.

Radiographic Evaluation

During this study, simple CT was used in 86% of cases for the diagnosis of PE. This radiographic modality allows confirmation and localization of PE within main, lobar, segmental, and subsegmental branches of the pulmonary vasculature. Only a few subjects were diagnosed with high-probability ventilation perfusion scan. During the last 3 years of the study, MDCT became the preferred diagnostic imaging for investigation of PE at our institution. With its improved image resolution and collimation, MDCT can localize a PE in smaller and more peripheral vessels.

Assessment of Severity of Obstruction

An adaptation of the pulmonary vasculature obstruction index of Qanadli and colleagues[11] uses CT information to assess obstruction severity. The index divides the pulmonary arterial tree into 10 segments per lung: 3 in the upper lobes, 2 in the middle lobe or lingula, and 5 in the lower lobe. An embolus in a segmental artery is considered totally occluded and is assigned 2 points. Proximally located emboli are assigned points for every distal segmental artery that they block. Subsegmental emboli that are detected are considered partially occluding and are assigned 1 point. The maximum CT obstruction index is 40. In this study, we tallied the obstruction score for each case, and the percentage of total lung vasculature occluded was estimated. According to current physiologic models of PE, already outlined, PEs that occluded 25% or less of total lung vasculature were considered *small,* those that occluded 25% to 50% were considered *medium,* and those that occluded more than 50% were considered *large*.

PE location within the pulmonary vasculature is another estimation of severity. In this study, severity of clinical presentation by PE location was also analyzed. Location within the pulmonary vasculature was estimated on the basis of the observed findings in the imaging study. Each PE case was categorized *main-stem, lobar, segmental,* or *subsegmental* on the basis of PE location within the pulmonary tree.

"...many factors other than size and occlusiveness of PE appear to affect clinical presentation, and clinical presentation alone is inadequate for estimating PE severity."

Table I. Presenting Clinical Symptoms, Changes in Vital Signs, and Blood Gas Measurements by Size of Pulmonary Embolism

	Size of Pulmonary Embolism			
Symptoms	**Large (n = 28)**	**Medium (n = 27)**	**Small (n = 85)**	***P***
Dyspnea	46%	42%	24%	0.06
Change in mental status	21%	26%	28%	0.48
Chest pain	15%	11%	14%	0.93
Diaphoresis	18%	11%	7%	0.1
Hemoptysis	4%	0%	3%	0.81
None	32%	30%	33%	>0.05
Changes in vital signs				
Hypotension	21%	15%	38%	0.56
Tachycardia	25%	26%	28%	0.94
Tachypnea	18%	22%	16%	0.08
Fever	18%	19%	22%	0.57
Hypoxia (exclusive)	7%	7%	11%	>0.05
Normal vital signs	50%	37%	31%	>0.05
Blood gases				
Desaturation (pulse oximetry <90%)	59%	62%	67%	0.45
Hypoxemia (PaO_2 <60 mm Hg)	76%	75%	62%	0.19
Increased alveolar-arterial gradient	100%	94%	88%	0.06

Abbreviation: PaO_2, mean partial pressure of oxygen in arterial blood.

Clinical presentation and exhibited signs were evaluated and compared with the obstruction index to determine a possible correlation between obstruction severity and clinical presentation.

Statistical Analysis

Descriptive statistics were generated for continuous and categorical variables. Continuous variables were reported as means and SDs, and categorical variables as proportions. Parametric and nonparametric analysis was performed for continuous (*T,* Wilcoxon) and categorical (χ^2 test, Fisher exact test) variables, as appropriate, to compare them across PE size and PE location categories. Correlation analysis was performed to determine association between variables. All analysis, performed with SAS software (version 9.1), was 2-tailed at an α level of .05.

Results

During the study, 144 (1.1%) of 13,133 patients developed PE. Overall, PE incidence was higher (P<.0001) after total knee arthroplasty (1.81%, 103/5695) than after hip arthroplasty (0.55%, 41/7438).

Clinical Presentation

Presenting clinical symptoms for PE were shortness of breath (31.9%, n = 46), change in mental status (27.1%, n = 39), chest pain (12.5%, n = 18), diaphoresis (9.7%, n = 14), and hemoptysis (2.8%, n = 4). Clinical symptoms were not present in 32.6% (n = 47) of patients (Figure).

Changes in vital signs included hypotension (systolic pressure, <90 mm Hg) in 30.6% (n = 44) of patients, tachycardia (heart rate, >100 bpm) in 28.5% (n = 41), fever (body temperature, >100.4°F) in 20.8% (n = 30), and tachypnea (respiratory rate, >20 breaths per minute) in 18.8% (n = 27). At presentation, 35.4% (n = 51) of patients had normal vital signs. Almost 10% (n = 14) of patients had no symptoms or abnormal changes in vital signs; the only indication of PE was oxygen desaturation on pulse oximetry.

Arterial oxygen desaturation and confirmed hypoxemia were the most common findings, with 63% (n = 91) of patients in this cohort having pulse oximetry below 90%. Arterial blood gas analysis was performed in 70% of the study population (101/144 patients with PE). Hypoxemia was confirmed in 60% of patients (n = 61) with mean partial pressure of oxygen in arterial blood (PaO_2) less than 60 mm Hg. Ninety-two percent (n = 93) of patients had an increased alveolar-arterial (A-a) gradient. Mean pulse oximetry for all patients in this cohort was 85.7% (range, 55%-100%). Mean arterial O_2 saturation was 88.2% (range, 62%-100%). Mean PaO_2 was 59.0 mm Hg (range, 35-112 mm Hg).

Size and Location of Pulmonary Embolism

The embolus was characterized as *large* in 19.4% (n = 28), *medium* in 18.8% (n = 27), and *small* in 59.0% (n = 85). There was no correlation between PE size and clinical presentation. The occurrence of clinical signs, symptoms, and blood gas measurements in the clinical presentation of PE grouped by size is detailed in Table I.

Embolism severity determined by location included 14.6% (n = 21) *main-stem,* 21.5% (n = 31) *lobar,* 54.2% (n = 78) *segmental,* and 8.3% (n = 12) *subsegmental*. Tachypnea was the only clinical finding that correlated

Table II. Presenting Clinical Symptoms, Changes in Vital Signs, and Blood Gas Measurements by Location of Pulmonary Embolism

Symptoms	Main-stem (n = 21)	Lobar (n = 31)	Segmental (n = 78)	Subsegmental (n = 12)	*P*
Dyspnea	38%	52%	29%	18%	0.06
Change in mental status	19%	29%	26%	13%	0.51
Chest pain	14%	13%	14%	8%	0.81
Diaphoresis	10%	19%	8%	0%	0.2
Hemoptysis	5%	3%	3%	0%	0.44
None	43%	23%	31%	50%	>0.05
Changes in vital signs					
Hypotension	24%	29%	30%	50%	0.22
Tachycardia	29%	23%	35%	17%	0.82
Tachypnea	24%	16%	18%	8%	0.03
Fever	14%	29%	22%	0%	0.53
Hypoxia (exclusive)	10%	9%	9%	17%	>0.05
Normal vital signs	43%	38%	30%	42%	>0.05
Blood gases					
Desaturation (pulse oximetry <90%)	60%	60%	66%	75%	0.33
Hypoxemia (PaO_2 <60 mm Hg)	81%	57%	66%	73%	0.72
Increased alveolar-arterial gradient	100%	92%	89%	91%	0.23

Abbreviations: PaO_2, mean partial pressure of oxygen in arterial blood.

with PE location (P = .03). There was no correlation between PE location and other presenting signs or symptoms. The occurrence of clinical signs, symptoms, and blood gas measurements in the clinical presentation of PE grouped by location is detailed in Table II.

Discussion

PE after TJA is a relatively common and serious complication with potentially devastating consequences.[9,12-14] The challenges of this perioperative complication are certainly of great concern to the orthopedic and medical community. Management of thromboembolic disease after major orthopedic surgery is hindered by controversial issues in the evidence for current prophylaxis, postoperative care, diagnosis, treatment, and outcome of this complication.

One of the challenges of PE relates to its being accurately diagnosed. Diagnosis is based on clinical suspicion for the condition and confirmation by imaging techniques. Although clinical symptoms and signs associated with PE have been described,[8,10,14-16] we believe the present study is the first to evaluate its clinical presentation after TJA, an exponentially growing population. The triad of pleuritic chest pain, dyspnea, and hemoptysis has been cited as the classical PE presentation,[16] but controversy surrounds the usefulness of these symptoms and signs in diagnosing PE.[16,17] The present study demonstrated that common symptoms are insensitive for detection of PE after TJA. Overall, a minority of patients with proven PE presented with the commonly attributed clinical signs and symptoms. For example, shortness of breath, the most common presenting symptom, was present in a third of the patients in this cohort.

This study also rejected another important finding—the common perception that PE size and location determine clinical presentation. We found no correlation between "severity" of occlusion, using the available model, and clinical presentation. Our study results suggest that PE severity cannot be estimated by clinical evaluation in patients who undergo TJA.

The question, then, is what strategy if any can be used to detect this undesirable and serious complication without overusing the resources and subjecting a large number of patients to cross-sectional imaging modalities. Although an algorithm that would allow cost-effective and accurate diagnosis of PE is desirable, implementation of such a strategy is hindered by the absence of any sensitive or specific clinical signs for this condition. One consistent finding of this study, however, is that almost all patients with PE presented with hypo-oxygenation exhibited by a drop in oxygen saturation or an increase in the A-a gradient. The latter is in agreement with previous studies demonstrating the same finding.[10,14,17]

Although hypoxemia cannot be claimed as a sensitive or specific sign for PE diagnosis, it can easily be determined using relatively simple and inexpensive tests. Obstruction of the pulmonary vasculature leads to increases in alveolar dead space, right-to-left shunting, and ventilation–perfusion mismatch. The A-a gradient was calculated to determine if hypoxemia was secondary to impaired gas exchange.[18] Previous studies have demonstrated a correlation between degree of pulmonary arterial occlusion and progression of severity in the physiologic manifestation of PE, as measured by mean pulmonary artery pressure (mPAP), PaO_2, and pulse oximetry.[14,19] Decreased PaO_2 is commonly the first and only sign in patients with small (<25%) occlusion and no preexisting cardiopulmonary disease.[14,19] Occlusions above 25% to 30% of the pulmonary vasculature have

been shown to increase mPAP and lead to pulmonary artery hypertension. Massive PE has been defined as more than 50% occlusion of the pulmonary vasculature or occlusion of 2 or more lobar arteries that can cause significant increase in vascular resistance. According to the current physiologic model, these large PEs can lead to increased pressures and result in subsequent right ventricular failure.[13,14,19]

Based on the findings of this study, we have implemented a strategy that includes administration of simple tests before subjecting patients to MDCT. These tests include evaluation of oxygenation using pulse oximetry and arterial blood gases as well as chest radiographs. The findings of the latter tests, as well as the overall suspicion for PE based on clinical acumen, determine the need for further investigations. This strategy has been implemented to limit resource use and patient inconvenience during the attempt to diagnose PE and prevent its undesirable consequences. All patients with a confirmed diagnosis of PE in this cohort were treated with anticoagulation continued for up to 6 months.

This study has a few limitations. First, though patients with PE after TJA were identified using prospectively collected data, retrospective chart review was performed to gather the data regarding the clinical presentation, and hence some unrecorded details may have gone undetected. Second, the best attempts were made to estimate PE severity by calculating the percentage of occlusiveness based on a pathophysiologic model and the described occlusion index. However, this model is static and lacks information regarding blood flow around a given embolus. In other words, all emboli were considered totally occlusive, which may not have been the case. Estimation of the occlusiveness of an embolus within a vessel may have provided a more accurate assessment of pulmonary vascular occlusion. Third, there was no possibility of evaluating the influence of preexisting medical conditions in general, or cardiopulmonary disease in particular, on PE severity.

Conclusions

This study demonstrated that analysis of clinical signs and symptoms is inadequate for PE detection and severity assessment. Evaluation of blood gases and determination of A-a gradient might be a reliable means of PE screening. Furthermore, many factors other than size and occlusiveness of PE appear to affect clinical presentation, and clinical presentation alone is inadequate for estimating PE severity.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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This paper will be judged for the Resident Writer's Award.