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Incidence of Postthrombotic Syndrome in Patients Undergoing Primary Total Hip Arthroplasty for Osteoarthritis

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Abstract

Background Postthrombotic syndrome (PTS) is a chronic condition in the lower extremity that develops after deep vein thrombosis (DVT). The incidence of PTS after total hip arthroplasty (THA) is not well established.

Questions/purposes We (1) determined the incidence of PTS after DVT in patients undergoing primary THA for osteoarthritis; and (2) determined whether the incidence of PTS was greater in patients with DVT than without.

Methods We retrospectively reviewed records of all 1037 patients who underwent primary THA for osteoarthritis during a 4-year period. All patients underwent postoperative screening ultrasound. We identified 21 (2%) patients with a DVT by ultrasound of whom 14 had a minimum 1-year followup (mean, 3.4 years; range, 1.0–6.0 years). PTS was diagnosed if any two of the six clinical signs were documented.

Results Three of 14 patients with DVT had at least two signs consistent with PTS; two of these had a DVT proximal to the soleal arch. Three of 91 randomly matched patients undergoing THA without DVT had at least two signs of PTS. The incidence of developing PTS after THA appeared higher in patients with DVT than in patients without DVT.

Conclusions While we observed a difference between the incidence of PTS after THA in patients with and without DVT, that incidence was based on only three of 1037 patients with DVT after THA. PTS does not appear to be a major complication after DVT in patients undergoing THA.

Level of Evidence Level III, diagnostic study. See Guidelines for Authors for a complete description of levels of evidence.

Introduction

Postthrombotic syndrome (PTS) is a chronic condition in the lower extremity that develops after deep vein thrombosis (DVT). The incidence of PTS in the medical literature varies from 20% to 70%, making it the most common reported complication after lower extremity DVT [2, 5, 7, 8]. PTS is a syndrome generally consisting of, but not limited to, edema, skin induration, hyperpigmentation, venous ectasia, redness, pain with calf compression, and venous ulceration [6]. The diagnosis of PTS is made based on the development of the mentioned clinical manifestations in patients with a history of DVT.

Three clinical scales have been reported using various combinations of these clinical signs and imaging studies to diagnose and grade the severity of PTS [3, 4, 17]. The Ginsberg scale uses two criteria for the diagnosis of PTS including (1) pain and swelling present for more than 1 month in duration and occurring more than 6 months after acute DVT; and (2) objective evidence on venous Doppler of valvular incompetence [4]. If both criteria are

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present, the diagnosis of PTS is made. The Villalta scale uses a combination of five symptoms (pain, cramps, heaviness, pruritis, paresthesia) and six signs (edema, skin induration, hyperpigmentation, venous ectasia, redness, pain with calf compression). These signs and symptoms are then numerically graded to establish a score to determine a diagnosis of PTS [17]. The Brandjes scale uses separate scales to categorize patients as having no, mild to moderate, or severe PTS that include items on symptoms, signs, and differences in calf circumference. Points for the items are summed into a total score and cutoff values are used to classify the presence of mild-to-moderate PTS and severe PTS [3]. The clinical picture of PTS is nonspecific, because clinical conditions other than DVT may result in a similar set of symptoms and signs in the lower extremity, including superficial venous insufficiency, increased body mass index, and trauma [14]. Recently the Villalta scale emerged as the scale most suitable for defining the presence and severity of PTS after objectively diagnosed DVT [9]. PTS is believed to be a common and important complication of DVT with an estimated annual US cost of greater than $200 million in one report [1].

Using warfarin for postoperative DVT prophylaxis, the incidence of DVT after THA is reportedly between 9% and 26% [15]. Although much effort has been devoted to preventing DVT and pulmonary embolus, we identified only one study with more than 100 patients specifically examining the incidence of PTS in patients after THA, which was inconclusive as a result of the small sample size [12]. It is unclear if this is secondary to a lack of education regarding the syndrome of PTS or insufficient clinical monitoring for PTS or if PTS is not a major morbidity after DVT in patients undergoing THA.

We therefore (1) determined the incidence of PTS after DVT diagnosed on screening ultrasound in patients undergoing primary THA for osteoarthritis; and (2) determined whether the incidence of PTS was greater in patients with DVT than without.

Patients and Methods

We retrospectively reviewed the records of all 1037 patients who underwent primary THA for osteoarthritis from 2001 through 2004. None of the patients included in this study had been diagnosed with PTS before their THA. All patients were treated with a multimodal prophylaxis regimen consisting of a combination of mechanical (early mobilization, pressure stockings, and sequential compression devices) and pharmacologic thromboprophylaxis (warfarin at a weight- and effect-adjusted dose with a goal international normalized ratio of 2.0). All patients underwent bilateral screening lower extremity ultrasound on postoperative Day 3 according to our protocol [10]. If there was no DVT diagnosed on postoperative Day 3, a total of 4 weeks of warfarin was prescribed followed by an additional 2 weeks of aspirin therapy. If there was a DVT diagnosed on postoperative Day 3, 3 to 6 months of warfarin was prescribed at the discretion of the patient's primary care physician.

The less specific diagnostic requirement (any two of six signs) was chosen to increase the sensitivity and decrease the false-negative rate. Patients with at least two of six signs were considered to have PTS. There are no gold standard tests for the diagnosis of PTS, and multiple criteria and diagnostic scales have been used in the medical literature [3, 4, 9, 17]. Although recently the Villalta scale has emerged as the scale most suitable for defining the presence and severity of PTS after objectively diagnosed DVT, it could not be used in a retrospective manner for this study [9]. Our diagnostic criteria are based on clinical signs present within the Villalta scale and are consistent with those used by McAndrew et al. in determining the incidence of PTS after TKA [13].

Each patient, regardless of presence or absence of DVT, was followed in the office at 6 weeks, 3 months, 6 months, 1 year, 3 years, and every 2 years thereafter. Other interim followup appointments were made with patients at their request for concerns or if a complication necessitated more frequent followup. Neither DVT nor PTS was, specifically, considered a complication requiring more frequent followup in this population. The patients' demographic, diagnostic, surgical, and complication information was entered into our prospective Institutional Review Board-approved joint replacement registry database.

We identified 21 patients who had a DVT after primary THA for osteoarthritis diagnosed on screening ultrasound on postoperative Day 3. Of these patients, 14 had at least 1 year of postoperative followup (mean, 4.8 years; range, 1.0–6.0 years) and medical records available for review. Attempts were made to optimize patient followup, including use of hospital records, radiographs from outside institutions, Internet directories, and the Social Security Death Index. This followup time was chosen to exclude patients who present with leg swelling in the immediate postoperative period. One hundred patients who had no DVT identified on screening ultrasound after primary THA for osteoarthritis were chosen at random from our database; from a list of patients with negative DVT studies listed in temporal order according to the day of their screening ultrasound, groups of 4–5 sequential patients were chosen and these small groups were picked every 50–60 patients. Ninety-one patients had at least 1 year of postoperative followup. We reviewed the outpatient medical records of these patients, including orthopaedic followup notes and

the notes of medical and surgical specialists, for documentation of the presence of six established signs of PTS: presence or absence of edema, venous ectasia, hyperpigmentation, varicose veins, venous ulceration, and pain with calf compression.

The two groups were similar in regard to age at surgery (mean 69.3 positive DVT group, mean 67.6 negative DVT group [df = 103] = −0.64, p = 0.52), gender ([df = 1] = 0.10, p = 0.75), and use of chronic anticoagulation prior to surgery ([df = 1] = 0.06, p = 0.80). Use of chronic anticoagulation before surgery was secondary to cardiac arrhythmias in all patients. There were no patients with a previous diagnosis of PTS, DVT, pulmonary embolus, or coagulopathy included in the study. Logistic regression was used to test the rates of PTS in patients with and without DVT with at least 1-year followup [16].

Results

The overall rate of DVT was 2% (21 of 1037). No patients were diagnosed with pulmonary embolism. The DVT was located in the operative limb in 19 patients and was contralateral in two. Seven of 21 DVTs were located distal to the soleal arch and 14 were located proximal to the soleal arch. Of the 14 patients with DVT and at least 1-year followup, three had at least two signs of PTS. The most common sign was edema (three) followed by erythema (two) with skin induration, hyperpigmentation, and pain with calf compression present in one patient each (Table 1). The three patients who had signs of PTS had DVTs in the operative extremity. Two of the three patients with PTS had a DVT located proximal to the soleal arch. Of the 91 without DVT and at least 1-year followup, three had at least two signs of PTS. The incidence of PTS was higher (p = 0.02; odds ratio = 8.0; 95% confidence interval = 1.4–45.1) in patients with DVT than in patients without DVT by screening ultrasound after THA for osteoarthritis.

Table 1. Patients with and without DVT with signs of PTS

Sign	DVT	No DVT
Edema	3	3
Induration	1	0
Hyperpigmentation	1	1
Venous ectasia	0	2
Erythema	2	0
Pain with calf compression	1	0

DVT = deep venous thrombosis; PTS = postthrombotic syndrome.

Discussion

PTS is a recognized complication of DVT. In the medical literature, the incidence of PTS developing in patients with symptomatic DVT ranges from 20% to 70% [2, 5, 7, 8], and DVT develops in 9% to 26% of patients undergoing THA using modern chemical prophylaxis [15]. According to Nationwide Inpatient Sample data reported by Kurtz et al. [11], the total number of primary THAs in the United States was 202,500 in 2003 and projected to increase to more than 500,000 per year in 2030. If PTS develops at the same rate of incidence in patients with asymptomatic DVT diagnosed on screening studies after THA, the clinical and financial burden would be staggering. We undertook this study to determine the incidence of PTS in a well-defined population of patients undergoing THA to better delineate the magnitude of this syndrome. We found an overall incidence of PTS of 21% in patients with DVT after THA, which is greater than in patients without DVT after THA.

This study is limited by a number of considerations. First, knowing the true incidence of PTS after THA is hampered by the use of varying definitions of PTS [3, 4, 6, 9], inconsistent prophylactic regimens [9], and mixed populations used in previous studies [4, 12]. Specific inquiry regarding symptoms and observation of signs of a newly reported syndrome is likely to be limited in routine followup, and inherent bias is introduced when reviewing surgeons' followup evaluation notes in a retrospective manner. Second, the number and availability of notes varied widely among our population. Loss to followup was minimized using hospital records, radiographs from outside institutions, Internet directories, and the Social Security Death Index. On the other hand, we had a relatively large population followed closely with routine clinical followup. With large numbers, we were able to focus this study on a well-defined subset of patients undergoing arthroplasty to minimize potential confounding factors that may affect the rate of PTS such as previous diagnosis of PTS, DVT, pulmonary embolus, or coagulopathy. Third, the clinical picture of PTS is nonspecific, and conditions other than DVT may result in a comparable set of signs and symptoms affecting the lower extremities of patients with a previous DVT, including superficial venous insufficiency, old age, increased body mass index, and trauma [14]. As a result of the retrospective nature of this study, we recognize that confounding variables may not have been correctly documented in the medical record and lead to bias. However, our protocol includes routine screening for DVT with ultrasound of bilateral lower extremities in all patients undergoing THA, so the diagnosis is not solely dependent on identification by symptoms or signs; and this study includes a cohort with similar age, gender, and

comorbidities to compare with the study group to better define the effect that the presence of DVT has on development of PTS. Finally, we do not have longer-term followup of these patients and cannot comment on the severity of the PTS or the impact of those symptoms on the patient's daily lives.

Our data suggest a low incidence of PTS in patients with DVT after THA. In comparison to the work of Mant et al. [12], our reported rate of DVT after THA was much lower (2% compared with 13%). Their reported rate of PTS of 12 of 188 patients (6%) includes all patients after THA, including patients with and without DVT. However, using their data, PTS was diagnosed in four of 25 patients using only patients diagnosed with DVT. This is closer to our incidence of PTS after DVT of three of 21 patients. These authors made no direct comparisons to patients without DVT in their study. It is difficult to directly compare the two studies as a result of differences in DVT prophylaxis protocols, DVT detection techniques, sample size, and diagnostic criteria. Ginsberg et al. [4] studied a mixed population of patients with DVT after THA and TKA and found a rate of DVT of 35% overall and an incidence of PTS of 5.5% after DVT. However, their study population included both THA and TKA in patients who received a mix of prophylactic regimens, including placebo, once again making direct comparison difficult. McAndrew et al. [13] previously reported a rate of PTS of 6% in patients with DVT after TKA in a patient population using the same multimodal prophylactic regimen as the patients in this study. The rate of PTS of 21% in patients with DVT after THA may point to an anatomic etiology for the development of PTS, because more patients in this study had proximal clots compared with patients undergoing TKA reported previously.

Second, we found a significant difference in the incidence of PTS in patients after THA in patients with and without postoperative DVT diagnosed with screening ultrasound. There are no previous studies in the literature that we are aware of that have addressed this question. It is important to realize, however, that although the difference is statistically significant, we were only able to identify three patients of 1037 who met our criteria for the diagnosis of PTS.

Based on our data, we suggest the overall incidence of PTS after THA remains extremely low, but PTS appears to occur more frequently after DVT in patients undergoing THA. The clinical importance of patients developing PTS after THA remains unknown. Further studies examining the clinical importance of PTS and the anatomic contribution of DVT to the incidence of PTS in patients undergoing total joint arthroplasty is warranted. Multimodal prophylaxis for venous thromboembolic disease after THA appears to reduce the incidence of DVT after THA as demonstrated by the extremely low incidence of DVT in this study. We consider effective DVT prophylaxis to be an important factor to minimize the incidence of not only DVT, but also PTS. Given the general lack of awareness of the diagnosis of PTS within the orthopaedic community, and the potential for increased morbidity, further education of orthopaedic surgeons on the signs and symptoms of PTS is important.

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