

Venous Thromboembolism Within Professional American Sport Leagues

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Background: Numerous reports have described players in professional American sports leagues who have been sidelined with a deep vein thrombosis (DVT) or a pulmonary embolism (PE), but little is known about the clinical implications of these events in professional athletes.

Purpose: To conduct a retrospective review of injury reports from the National Hockey League (NHL), Major League Baseball (MLB), the National Basketball Association (NBA), and the National Football League (NFL) to take a closer look at the incidence of DVT/PE, current treatment approaches, and estimated time to return to play in professional athletes.

Study Design: Descriptive epidemiology study.

Methods: An online search of all team injury and media reports of DVT/PE in NHL, MLB, NBA, and NFL players available for public record was conducted by use of Google, PubMed, and SPORTDiscus. Searches were conducted using the professional team name combined with *blood clot*, *pulmonary embolism*, and *deep vein thrombosis*.

Results: A total of 55 venous thromboembolism (VTE) events were identified from 1999 through 2016 (NHL, $n = 22$; MLB, $n = 16$; NFL, $n = 12$; NBA, $n = 5$). Nineteen athletes were reported to have an upper extremity DVT, 15 had a lower extremity DVT, 15 had a PE, and 6 had DVT with PE. Six athletes sustained more than 1 VTE. The mean age at time of VTE was 29.3 years (range, 19-42 years). Mean (\pm SD) time lost from play was 6.7 ± 4.9 months (range, 3 days to career end). Seven athletes did not return to play. Players with upper extremity DVT had a faster return to play (mean \pm SD, 4.3 ± 2.7 months) than those with lower extremity DVT (5.9 ± 3.8 months), PE (10.8 ± 6.8 months), or DVT with PE (8.2 ± 2.6 months) ($F = 5.69$, $P = .002$). No significant difference was found regarding time of return to play between sports.

Conclusion: VTE in professional athletes led to an average of 6.7 months lost from play. The majority of athletes were able to return to play after a period of anticoagulation or surgery. Those with an upper extremity DVT returned to play faster than those with other types of VTE. Further study is needed to look into modifiable risk factors for these events and to establish treatment and return-to-play guidelines to ensure the safety of these athletes.

Keywords: venous thromboembolism (VTE); pulmonary embolism (PE); deep vein thrombosis (DVT); professional athlete; elite athlete; return to play

Venous thromboembolism (VTE) in professional athletes is a serious condition, often requiring long-term anticoagulation therapy with potential career- or life-threatening ramifications. VTE is an umbrella term encompassing upper

extremity deep vein thrombosis (DVT), lower extremity DVT, and pulmonary embolism (PE). Recently, numerous press reports have been published pertaining to VTEs in professional athletes, including members of the National Football League (NFL), National Hockey League (NHL), National Basketball Association (NBA), and Major League Baseball (MLB). However, the clinical implications and long-term sequelae of VTE in professional athletes remain uninvestigated.

The recent surge in case and press reports of VTEs in elite athletes warrants a thorough investigation into the causes, sequelae, and associated risk factors. Case reports have described VTE in seemingly healthy athletic populations including young competitive rowers, elite marathoners, mountaineers, and professional athletes.[§]

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Increased media attention has led to public awareness of VTE, particularly in athletes. Various potential risk factors associated with VTE in professional and elite athletes have been suggested, including increased travel to and from competitions, repetitive traumatic episodes resulting in vascular damage, and the high-force, repetitive nature of athletics.^{13,14,19,23,31,37,40}

Current research into VTEs in athletic populations explores the pathogenesis and causes of the disease. Eichner¹³ categorized and documented cases of travel-related VTE in elite athletes after flights. Parker et al³⁷ conducted similar work and found that the hemostatic balance of coagulation and fibrinolysis was altered after a marathon in a cohort of runners who underwent long flights prior to their race. Hull and Harris²¹ postulated that elite athletes are predisposed to develop Virchow's classic triad of hypercoagulability, vascular fluid stasis, and vascular damage with consequent VTE because of potentially thrombogenic risk factors associated with intense cardiovascular exercise. While VTEs represent serious medical conditions irrespective of profession, they can be career-ending in professional athletes. After experiencing a VTE, professional or elite athletes may need to alter their career plans by either retiring prematurely or missing prolonged periods of game or practice time due to the need for long-term anticoagulation therapy.

To date, the media-reported rate of VTE, associated risk factors, typical treatment protocols, and medical outcomes within professional athletic leagues in the United States remain undetermined. The purpose of this study was to describe the incidence of VTE, current treatment approaches, and estimated time to return to play within professional athletic leagues (NFL, NHL, NBA, and MLB). This knowledge may elucidate risk differences in sport and subtype of VTE and allow assessment of treatment protocols and medical or return-to-play outcomes.

METHODS

A database of all VTEs among male professional athletes was compiled for the 4 major professional sport leagues in the United States (NFL, NHL, NBA, and MLB) via an online search for potential press releases or public information regarding VTE and professional athletes. Comprehensive Google, PubMed, and SPORTDiscus searches were performed with the name of each team and the terms *deep vein thrombosis*, *pulmonary embolism*, and *blood clot*. All relevant links were investigated in detail to identify all athletes who experienced a VTE. Only athletes with a confirmatory news report were included in the study.

Additional player information was compiled along with injury reports, press releases, and news articles pertaining to the player and the VTE incident. Player age at VTE was calculated based on date of incident and birthdate. Known risk factors, such as family history of VTE, previous history of VTE, and known clotting disorders, were recorded if they were available. Treatment protocols were determined based on news reports and press releases from team organizations. Time lost was calculated based on the difference

between the diagnosis of VTE and next professional game played. If the VTE was season ending, the date of the first game played of the next season was used. All career-ending and fatal VTEs were noted.

Descriptive statistics were performed on all athletes who were identified in the VTE search. Chi-square analysis was used to assess any differences in the percentage of athletes who returned to play based on sport as well as the percentage who returned based on location of VTE. Single-factor analysis of variance (ANOVA) was used to determine whether there was a difference in the average time it took for athletes to return to play based on sport and based on location of VTE. Potential risk factors for VTE were assessed by multivariable regression analysis. Microsoft Excel and R (R Foundation for Statistical Computing) were used for all statistical calculations.

RESULTS

A total of 55 VTEs were identified in online records from 1999 through 2016. Twenty-two events occurred in NHL players (Table 1), 16 in MLB players (Table 2), 12 in NFL players (Table 3), and 5 in NBA players⁶ (Table 4). Nineteen athletes were reported to have an upper extremity DVT, 15 had a lower extremity DVT, 15 had a PE, and 6 had a DVT with PE. MLB was found to have a statistically higher number of athletes sustaining an upper extremity DVT compared with other professional sports ($P < .001$). Six athletes (4 NHL, 1 MLB, 1 NBA) sustained more than 1 VTE. All MLB athletes who sustained a VTE were pitchers. For NHL players, 9 played offensive positions, 7 played defensive positions, and 2 were goaltenders. For NBA players, 3 were centers and 1 was a forward. For NFL players, 2 were defensive backs, 4 were defensive linemen, and 6 were offensive linemen.

The average age at the time of VTE was 29.3 years (range, 19-42 years). Average age per sport was NHL 28 years (range, 19-39 years), MLB 29 years (range, 23-42 years), NFL 30 years (range, 25-36 years), and NBA 30 years (range, 29-38 years). No significant difference in age was found between sports ($P = .640$), but the average age of players with upper extremity DVT (26 years) was significantly lower than that of players with other VTEs ($P = .002$). Numerous nationalities were represented among the sports (14 different countries); American was the predominant nationality represented in all sports except hockey, where Canadian was the predominant nationality (40.9%) ($P < .001$). No association was found between team geographic location (East Coast, West Coast, Midwest, Canada) and risk of VTE ($P = .368$).

Risk factors for VTE were assessed, including known clotting disorder, family history of clotting disorder, preceding injury or trauma, status postsurgery, and prior history of clot. Identifiable risk factors were found in 22 events, while 33 events had no specific risk factor noted. Ten events were associated with a known personal or family history of a clotting disorder, 5 entailed a prior history of clot with no confirmed diagnosis of clotting disorder, 4 occurred after recent injury or trauma, and 3 occurred after surgery. Having a potential variable that is known to predispose to

TABLE 1
Details of Venous Thromboembolic Events in NHL Players^a

Type of VTE	Treatment	Risk Factors Identified	Time Missed From Play, mo	Year of Event	NHL Team
UE DVT	Surgery for clot/first rib/neck muscle removal; anticoagulation therapy (2 mo)	Noted after upper body workout	4.67	2012	Boston Bruins
	1 wk of IV anticoagulant for clot dissolution; 3 mo of oral anticoagulants	Brother (NHL player) diagnosed with TOS as well	2.97	2013	Calgary Flames (AHL)
	Surgery for first rib removal		5.87	2012	Dallas Stars (AHL)
	Surgery for first rib removal		3.07	1999	Los Angeles Kings (AHL)
	Surgery for first rib/neck muscle removal	Brother (NHL player) diagnosed with TOS as well	2.63	2016	New York Islanders
	Surgery for first rib removal, anticoagulation therapy (2 mo)		2.03	2015	Tampa Bay Lightning
	Surgery for first rib removal, anticoagulation therapy (2 mo)		5.73	2016	Tampa Bay Lightning
LE DVT	Oral anticoagulants (9 mo)	Event after blocked shot	9.50	2012	Calgary Flames
	Anticoagulation therapy (3 mo)	Pelvic venous scarring from childhood trauma	3.50	2006 ^b	Nashville Predators
	IVC filter placement; Lovenox injections indefinitely; wears extra padding on torso and compression stockings while traveling; undergoes monthly US of legs and abdomen; times injections based on practice/game schedule	Hereditary clotting disorder; history of prior PE 2 years before; occurred after revision ACL reconstruction	10.20	2008 ^b	Nashville Predators
	Anticoagulation therapy (3 mo)		4.60	2012	New Jersey Devils
	Anticoagulation therapy (continued for 3.5 years)	Hereditary clotting disorder	CE	1999	New York Islanders
	Anticoagulation therapy		8.33	2002	Toronto Maple Leafs
	Anticoagulation therapy (3 mo); no contact for 3 mo; compression stockings on flights		3.47	2013	Winnipeg Jets
	Surgery for clot dissolution, anticoagulation therapy (4 mo)	Previous history of pelvic thrombophlebitis	CE	2013 ^b	Pittsburgh Penguins
	Anticoagulation therapy	Protein C deficiency	0.33	2008 ^b	Philadelphia Flyers
	PE	Lovenox injections indefinitely	Hereditary clotting disorder, prior history of DVT	8.63	2011
	Lovenox injections	Hereditary clotting disorder; occurred after ACL reconstruction surgery	5.13	2006	New York Rangers
DVT and PE	Oral anticoagulants (unknown duration)	Protein C deficiency; event after a blocked shot	CE	2015	Buffalo Sabres
	Lovenox injections and then Xarelto indefinitely (did not take on game day)	Protein C deficiency; prior history of lower extremity DVT	6.87	2014 ^b	Philadelphia Flyers
	Lovenox injections indefinitely	Prior history DVT/PE	9.07 ^c	2014 ^b	Pittsburgh Penguins
	Lovenox injections and then oral anticoagulant (6 mo)	Occurred 1 wk after ACL reconstruction	5.03	2014 ^b	Pittsburgh Penguins

^aACL, anterior cruciate ligament; AHL, American Hockey League; CE, career ending; DVT, deep vein thrombosis; IV, intravenous; IVC, inferior vena cava; LE, lower extremity; NHL, National Hockey League; PE, pulmonary embolism; TOS, thoracic outlet syndrome; UE, upper extremity; US, ultrasonography; VTE, venous thromboembolism.

^bAthlete with recurrent VTE.

^cRetired 3 months after return due to continued blood clot concerns.

coagulopathy was found to be a risk factor for VTE in athletes ($P = .033$). All potential risk factors analyzed are noted in Tables 1 through 4.

The majority of VTEs were treated with pharmacologic anticoagulation (oral or injectable) ($n = 31$, 56%), surgery

($n = 8$, 15%), or a combination of the two ($n = 16$, 29%). One athlete was treated with a calcium channel blocker in addition to an anticoagulant. Data on length of treatment with anticoagulation could be found for 18 VTEs: 3 were treated with pharmacologic anticoagulation for 2 months (all were

TABLE 2
Details of Venous Thromboembolic Events in MLB Players^a

Type of VTE	Treatment	Risk Factors Identified	Time Missed From Play, mo	Year of Event	MLB Team		
UE DVT	Surgical removal of first rib	Family history	7.10	2011	Tampa Bay Rays		
	Anticoagulation therapy, calcium channel blockers		0.10	2012	Toronto Blue Jays		
	Surgical removal of first rib, anticoagulation therapy		5.20	2015	Atlanta Braves		
	Surgical repair, anticoagulation therapy		7.53	2015	Detroit Tigers		
	Surgical repair, anticoagulation therapy		TOS previously with surgical removal of rib in 2001	2.83	2007	Detroit Tigers	
	Surgical removal (angioplasty), anticoagulation therapy		12.20	2008	Detroit Tigers		
	Surgical removal of first rib, anticoagulation therapy (6 mo)		5.83	2013	Minnesota Twins		
	Surgical removal of rib		3.50	1999	Minnesota Twins		
	Surgical removal of clot, vein graft		3.73	2006 ^b	Pittsburgh Pirates		
	Surgical removal of clot, anticoagulation therapy		1.60	2013	Los Angeles Angels		
	Surgical removal of clots, anticoagulation therapy		Previous DVT	3.67	2008 ^b	Colorado Rockies	
	LE DVT		Anticoagulation therapy	1 wk post ACL, third-degree sprain	10.87	2012	New York Yankees
	PE		Anticoagulation therapy		CE	2011	Boston Red Sox
Anticoagulation therapy (3-6 mo)		Regular air travel	29.20	2006	Chicago Cubs		
Anticoagulation therapy			7.63	2002	Texas Rangers		
DVT and PE	Surgical removal of first rib, anticoagulation therapy		11.90	2004	Colorado Rockies		

^aACL, anterior cruciate ligament; CE, career ending; DVT, deep vein thrombosis; LE, lower extremity; MLB, Major League Baseball; PE, pulmonary embolism; TOS, thoracic outlet syndrome; UE, upper extremity; VTE, venous thromboembolism.

^bAthlete with recurrent VTE.

TABLE 3
Details of Venous Thromboembolic Events in NFL Players^a

Type of VTE	Treatment	Risk Factors Identified	Time Missed From Play, mo	Year of Event	NFL Team
UE DVT	Surgical removal of clot		2.00	2001	Detroit Lions
LE DVT	Surgical removal of clot	Family history of blood clots	8.13	2015	New York Giants
	Surgical removal of clot		5.90	2012	New York Giants
	Surgical removal of clot, anticoagulation therapy; career ending; irreversible damage to tibialis anterior		CE	2015	Minnesota Vikings
PE	Anticoagulation therapy	Recent large weight loss (lost 70 lb recently, down from 410 lb)	CE	2010	Washington Redskins
	Anticoagulation therapy		10.37	1999	Seattle Seahawks
	Anticoagulation therapy	Major trauma	Death	2000	Kansas City Chiefs
	Anticoagulation therapy (6 mo)		13.13	2004	Baltimore Ravens
	Anticoagulation therapy (6 mo)	Previous PE/clotting issues	CE	2012	Cleveland Browns
	Anticoagulation therapy	Fell on ball on chest; air travel 12 h after	13.23	2000	Atlanta Falcons
	Anticoagulation therapy	Renal disease; potential medication side effect	10.07	2006	Carolina Panthers
	Anticoagulation therapy (Lovenox injections for 1 wk followed by 6 mo of oral anticoagulants)	Impact trauma to chest	9.13	2011	New York Giants

^aCE, career ending; DVT, deep vein thrombosis; LE, lower extremity; NFL, National Football League; PE, pulmonary embolism; UE, upper extremity; VTE, venous thromboembolism.

TABLE 4
Details of Venous Thromboembolic Events in NBA Players^a

Type of VTE	Treatment	Risk Factors Identified	Time Missed From Play, mo	Year of Event	NBA Team
LE DVT	Anticoagulation therapy (Coumadin 1 wk, heparin daily injections after, aspirin remainder of season)	Previous incidences (heart episode [1996], thrombophlebitis [1989])	0.43	2001	Houston Rockets
PE	Anticoagulation therapy	Previous DVT/PE	CE	2016 ^b	Miami Heat
	Anticoagulation therapy		3.00	2015	Brooklyn Nets
DVT and PE	Surgical removal, anticoagulation therapy (3 mo)	Postsurgical complication	9.37	2013	Cleveland Cavaliers
	Anticoagulation therapy (Xarelto 3 mo), followed by continual aspirin therapy		8.30	2015 ^b	Miami Heat

^aCE, career ending; DVT, deep vein thrombosis; LE, lower extremity; NBA, National Basketball Association; PE, pulmonary embolism; VTE, venous thromboembolism.

^bAthlete with recurrent VTE.

upper extremity DVTs in association with surgery for first rib removal), 1 was treated for 4 months, 6 were treated for 3 months, 6 were treated for 6 months, 1 was treated for 9 months, and 1 was treated for 3.5 years (average, 4.3 months of treatment, excluding the athlete treated for 3.5 years). Four NHL players who had recurrent VTEs were treated with anticoagulants indefinitely (3 received daily Lovenox [Sanofi Aventis] injections, 1 received Lovenox and then Xarelto [Janssen Pharmaceuticals] daily), and 1 NBA player with a recurrent VTE was placed on Xarelto for 3 months and then daily aspirin indefinitely. Twenty-four athletes underwent some form of surgery for treatment of their VTE. The most common surgical procedure performed was surgical clot removal with or without first rib removal for upper extremity DVT associated with thoracic outlet syndrome, which was performed in all 19 athletes with upper extremity DVTs. Four other athletes underwent surgery for lower extremity DVT and 1 athlete underwent surgery for PE, which included surgical clot removal in 4 athletes and inferior vena cava filter placement in 1.

The average time lost from play was 6.7 ± 4.9 months (range, 3 days to career end). Chi-square analysis of the percentage of athletes who returned to play revealed no significant difference between sports (MLB 93.8%, NBA 80.0%, NFL 66.7%, and NHL 86.4%; $\chi^2 = 3.89$, $df = 3$, $P = .274$). The overall return-to-play rate among all sports was 85.4%. No significant difference was found in time to return to play between the different sports. NHL players returned at 5.3 ± 2.8 months compared with MLB (7.5 ± 7.0 months), NBA (5.3 ± 4.7 months), and NFL (9.0 ± 3.7 months) ($F = 1.36$, $P = .268$). Location of the VTE did not have a statistically significant impact on the percentage of athletes who returned to play ($\chi^2 = 6.05$, $df = 3$, $P = .110$), but there were significant differences in time to return to play based on DVT location. Players with upper extremity DVT had the fastest return to play (mean \pm SD, 4.3 ± 2.7 months) compared with lower extremity DVT (5.9 ± 3.8 months), PE (10.8 ± 6.8 months), and DVT with PE (8.2 ± 2.6 months) ($F = 5.69$, $P = .002$). Seven players never returned to play after the initial event (6 retired and 1 died from a PE weeks after sustaining significant injuries in a

car accident), and 1 retired due to concerns after sustaining a recurrent DVT/PE. One additional player has not yet retired but has still not played since sustaining a DVT.

DISCUSSION

VTEs are estimated to occur at a rate of 1 to 2 events per 1000 people in the general population.^{2,19} Virchow's triad of venous stasis, endothelial injury, and a hypercoagulable state is a long-recognized combination of risk factors for DVT.^{19,34} Numerous conditions are associated with increased risk of thrombosis, including both hereditary clotting disorders and acquired conditions such as immobilization, tissue trauma, pregnancy, smoking, advanced age, obesity, oral contraceptive use, surgery, prolonged air travel, and malignancy.^{2,10,19} Professional athletes are predisposed to a number of these acquired conditions associated with an increased risk of thrombosis, including tissue trauma from contact during play, hemoconcentration from dehydration, circulatory stasis secondary to bradycardia (high cardiopulmonary fitness), and long periods of immobilization from either travel or injury.^{19,21} While obesity is not a factor for most professional athletes, we found that 10 of the 12 NFL athletes who sustained a VTE were linemen, with an average body mass index of 38.2 (range, 33.9-44.8). Prior studies have shown an increased association between obesity and VTE, suggesting that this relationship may warrant closer investigation.^{12,26,41}

Strenuous exercise itself may predispose some athletes to hypercoagulability.^{15,19,20,30} Several studies have explored the relationship between exercise and the blood coagulation cascade, finding a blood level increase in prothrombotic markers and factor VIII as well as simultaneous activation of the fibrinolytic system and increased platelet aggregation.^{15,19,20,30} Particularly for those athletes with hereditary clotting disorders, studies have shown enhanced clotting response to exercise in people with protein C deficiency.⁴⁷ De Caterina et al⁹ described 4 cases of DVT after vigorous exercise in athletes who were carriers of protein C deficiency. Parker et al³⁷ specifically looked at the effect of

air travel on exercise-induced coagulatory and fibrinolytic activation in Boston marathon runners and found that the combination of air travel longer than 4 hours and endurance running stimulated a prothrombotic state, inducing an increase in procoagulatory factors relative to fibrinolytic factors immediately after exercise.³⁷ In our study, although identifiable acquired major risk factors, including known clotting disorder, family history of clotting disorder, preceding injury or trauma, status postsurgery, and prior history of clot, were noted in 22 cases of VTE, 33 had no identifiable major risk factor, suggesting a more subtle predisposition to coagulopathic events possibly related to the effects of strenuous exercise or travel.

Frequent long-distance travel is a unique phenomenon for professional athletes; games are played nationwide as well as in other countries including Canada (NHL), China (NBA), Europe (NFL), and Australia (MLB), and there are plans by MLB to increase the number of international games played.⁴² Professional athletes often travel immediately after games in the prothrombotic postexercise state, as described by Parker et al.³⁷ Studies have also shown an increased risk of VTE with long-distance travel without other acquired risk factors. Ferrari et al¹⁷ reported a 4-fold increased risk of VTE after 4 hours of travel (airplane, train, or car), and Philbrick et al³⁸ showed an increased risk after 6 hours of air travel. Interestingly, in our study, we found no association between team geographic location (East Coast, West Coast, Midwest, Canada) and risk of VTE ($P = .368$), suggesting that geographic location of teams that potentially have to travel farther for most play is not a major factor. However, most team schedules are organized now so that away game series are grouped by geographic location to reduce the amount of time spent traveling for teams. Additionally, high altitude is known to be a risk factor for developing VTE with increased levels of hypoxia and hemoconcentration.¹³ This is particularly meaningful for those athletes who train in places such as Denver, Colorado, where athletic stadiums are all located at elevations over 5000 feet. While our study is insufficiently powered to make any conclusions, we noted that 2 MLB players from the Colorado Rockies sustained a VTE. Heightened awareness of the signs and symptoms of VTE for athletes who compete and train at elevation is important.

While we did not find a significant correlation with geographic location, the professional sport with the most VTEs was the NHL ($n = 22$); of the 30 NHL teams, 7 are located in Canada. It is possible that our numbers are too low to detect a significant difference based on team location, but it is important to point out this finding because NHL players likely spend more time traveling for games than do players in other professional sports. Unique also to the NHL is that athletes practice and play on an ice rink, where the average temperature of the surface is below freezing. Numerous studies have detected a seasonal relationship with the pathogenesis of DVT,^{4,5,18,25} with DVTs occurring at a more frequent rate in the winter months.⁸ While the association between cold weather and DVT is not well understood, it is postulated to be related to prothrombotic changes induced by cold temperature³⁵ or vasoconstriction¹⁸ or a link

between increased upper respiratory infections in winter months and their relation to hypercoagulability.^{32,44}

Upper extremity DVT/thoracic outlet syndrome, also known as effort thrombosis of the subclavian vein or Paget-Schroetter syndrome, was found to have unique characteristics in our study compared with lower extremity DVT/PE.³³ Players who sustained an upper extremity DVT were significantly younger (average age, 26 years) and more likely to return to play than those who sustained other forms of VTE. All but 1 of the 19 upper extremity DVTs occurred in NHL ($n = 7$) and MLB athletes ($n = 11$), with all MLB athletes being pitchers. The repetitive overhead motion of pitching is a previously described risk factor for developing thoracic outlet syndrome, but not specifically DVT.^{11,24,33}

The predilection for upper extremity VTE in MLB pitchers may be due to biomechanical demands and altered blood flow mechanics associated with the throwing motion of pitchers. Bast et al¹ demonstrated that peak blood flow in the throwing arm of pitchers occurred at 40 pitches and steadily declined as pitch count increased thereafter. Further, the decrease in blood flow is dependent on the position of the arm during throwing (abducted, horizontally abducted, and externally rotated), with reduced blood flow when the arm is in the throwing position compared with at rest by the side.²⁸ When compared with MLB positional players, a significant difference in blood flow was noted between the throwing arms of pitchers, but only for provocative position.²⁸ Laudner et al²⁹ measured a decrease in blood flow during the provocative pitching position after 1 competitive season of pitching. As pitchers approach fatigue in a simulated game, the biomechanical properties of the pitching motion are only minimally altered, negating the idea that shoulder and elbow torque increase as muscle fatigue approaches.¹⁶ Additionally, thoracic outlet syndrome has rarely been reported in hockey players. Our study suggests that this event may be more common in elite hockey players than previously described. One could hypothesize that this is associated with the repetitive motion of stick handling or trauma due to frequent upper body checks into the boards.

The literature has little information concerning acute and long-term management of athletes who have sustained a VTE or guidelines on return to play after VTE. Much of the literature consists of case reports¹¹ on diagnosis and management. We found that the majority of VTEs in professional athletes were treated with anticoagulants (oral or injectable) ($n = 31$, 56%), surgery ($n = 8$, 15%), or a combination of the two ($n = 16$, 29%). Of those players treated with anticoagulants for a first-time DVT ($n = 47$), the average length of treatment (of those with treatment data; $n = 18$) was 4.3 months. The 5 athletes who sustained a recurrent VTE and were treated with anticoagulants were placed on anticoagulation indefinitely after the recurrent event. In our study, treatment of upper extremity DVT was consistent with that reported in the literature; surgical clot removal with or without first rib removal was performed in all 19 athletes with upper extremity DVTs.³³

¹¹References 7, 11, 13, 14, 19, 22, 24, 27, 36, 39, 43, 45, 46, 48.

The American College of Chest Physicians recommends a minimum of 3 months of anticoagulation for the treatment of DVT in a patient with modifiable risk factors.¹⁹ For a patient without significant acquired risk factors, the American College of Chest Physicians recommends at least 3 months of anticoagulation and a subsequent risk-benefit evaluation to determine the need for prolonged treatment.¹⁹ While anticoagulation guidelines for DVT have been established for the general population, these do not necessarily apply to elite athletes because they have unique requirements for their sports, including frequent travel, vigorous training regimens, and risk of significant contact. No evidence-based guidelines exist regarding when it is safe for these athletes to return to play and whether it is safe to play while receiving a form of anticoagulation.

Depenbrock¹⁰ attempted to create return-to-training guidelines for athletes who had sustained a VTE, suggesting that nonweightbearing activities can be resumed at 4 weeks after the event, nonimpact exercises such as cycling at 5 weeks, and impact loading exercises such as running at 6 weeks. These guidelines were based on animal models of the natural history of thrombosis, which show that by weeks 4 to 6, dissolution of the clot and recanalization of the vessel have occurred. Depenbrock's¹⁰ guidelines recommend return to contact sports only after completion of anticoagulation therapy.

Berkowitz and Moll³ studied individualized, intermittent anticoagulation management for athletes who sustained a VTE, suggesting use of short-acting direct oral anticoagulants (such as dabigatran) and personalized measurements of the plasma half-life of the drug in the athlete's system to allow athletes to participate in contact sports while receiving treatment for VTE. Compression stockings are a noninvasive method that is frequently used to combat circulatory stasis. Zaleski et al⁴⁹ explored the effect of marathon running and use of compression stockings on hemostasis in a population of 20 runners; the investigators found activation of hemostatic factors (both procoagulatory and fibrinolytic) to be lower in the compression stocking group than in controls. Zaleski et al⁴⁹ concluded that compression stockings do not adversely affect normal clotting system activation with exercise but may lower overall activation, which potentially could be beneficial for athletes traveling long distances or with a known clotting history.

We looked at specific treatment regimens of some of the players in our study population who returned to play after sustaining a recurrent VTE; some of the interventions included wearing compression stockings while traveling and timed administration of anticoagulation based on practice and game schedules in 2 NHL players. One player was reported to undergo monthly ultrasonography of the lower extremities and abdomen for DVT screening. Overall, however, we found little consistency in treatment methods, duration of treatment, and time lost from play among participants with VTE in our study population.

Future considerations need to be explored for establishing treatment guidelines and a timeline for safe return to play after clotting events in the elite athlete. We found that the average time of return to play was 6.7 months for all forms of VTE and 4.3 months for upper extremity DVT. We

found no difference between sports regarding the number of players affected and time to return to play. These are considerable time periods for a professional athlete to be out of competition, and they carry potentially significant monetary and mental effects for the player as well as the team. A better understanding of the pathogenesis of prothrombotic factors in athletes and the effects of anticoagulation treatment would allow for improvements in screening and treatment of VTEs in athletes.

Improved treatment guidelines could significantly reduce the time missed from competition; the current average time lost from play after a DVT is substantially greater than the 3-month minimum treatment period recommended by the American College of Chest Physicians. Treatment that involves tailored dosing of anticoagulation, based on pharmacokinetics of the drug in an individual patient, might allow earlier return to play, which could be particularly important for players with recurrent DVT or known history of a hereditary clotting disorder. However, further research is needed on this topic and the timing of return to contact play, keeping in mind the risk-benefit ratio for the athlete. Consideration should be given to screening athletes for thrombogenic risk factors during sports preparticipation physicals and preoperatively. Such risk factors include prior venous thrombosis and family history of hereditary clotting disorders; this is an important consideration given that 27.2% (15/55) of VTEs in this study were associated with either a personal or family history of previous clots or a known clotting disorder.¹⁹ The role of intermittent screening ultrasonography for those who have had a DVT or who are at risk of DVT is not yet determined. Finally, an improved database registry of VTE in athletes would help researchers monitor the frequency of these events and treatments used, in order to direct future guidelines.

The results of this study must be viewed within the context of the limitations. All of the data regarding incidence of VTE, treatment, and return to play are drawn from media reports found during internet searches and are not from actual patient records. Only 1 article was identified through the PubMed and SPORTDiscus search; that article identified 3 NBA players with PE who were already included from the Google search, and the remainder of athletes included were from reports from the Google search.⁶ This may have resulted in a selection bias, such that our study included high-profile professional athletes with access to high levels of medical care and intense media attention. Therefore, our study likely underestimated the true incidence of VTE given that an unknown number of athletes may have been missed.

Further, this study included only male athletes who were at the peak of physical fitness and at the height of their respective professional athletic leagues, potentially limiting the scope of applicability. Female athletes were not included in this analysis. Given women's additional unique risk factors for VTE, including oral contraceptive use and pregnancy, inclusion of female athletes would be of interest for future study. Additionally, full patient treatment data and risk factor data were not available to us, since we exclusively used publicly available data. For example, 8 patients

were listed as being treated with surgery alone, but information regarding anticoagulation treatment may not have been included in the media report. Also, we found specific data on length of anticoagulation treatment in only 18 of 55 athletes and data on specific risk factors in 22 of 55 athletes. While it is possible that a number of these athletes did not have identifiable risk factors, our data and results are subject to the limitations of our search method, our limited power to analyze risk factors, and the need for improved database registries in American professional sports leagues. Access to league injury databases would provide better insight into the true incidence and clinical impact of VTE in professional athletes. For return-to-play analysis, if the VTE was season ending, the first game start date of the next season was used as date of return, which may have overestimated the actual time missed from the event. Furthermore, any nonsignificant findings in this study may be a result of the small sample size, as VTE is a relatively rare event. We believe that despite these unavoidable shortcomings, this study adds value to the treatment of athletes, as this is the first analysis of the impact of VTE across all 4 major American professional sports. We hope that this study provides a foundation for future research on VTE management to help athletes stay healthy and get back to their sport.

CONCLUSION

Overall, VTE is uncommon in NHL, NBA, MLB, and NFL players; however, they are exposed to a number of acquired risk factors that may predispose them to clotting, including frequent travel, repetitive microtrauma, and hemoconcentration from dehydration. The impact of vigorous exercise on the coagulation cascade is not yet understood, and the combined effect of exercise and hereditary clotting disorder may compound these factors.¹⁹ While there is no consensus on how to manage these events, this research showed that the average time lost from play was 6.7 months and the majority of American professional sports players were able to return to play after a period of anticoagulation or surgery. Those with an upper extremity DVT returned to play faster than players with other types of VTE. Future study is needed regarding appropriate treatment as well as screening and prevention guidelines for the elite athlete who sustains a VTE.

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