Positional compression of the axillary artery causing upper extremity thrombosis and embolism in the elite overhead throwing athlete

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Objectives: **To describe the spectrum of axillary artery pathology seen in high-performance overhead athletes and the outcomes of current treatment.**

Methods: **A retrospective review of patients that had undergone management of axillary artery lesions in a specialized center for thoracic outlet syndrome (TOS). Treatment outcomes were assessed with respect to arterial pathology and operative management.**

Results: **Nine male athletes were referred for arterial insufficiency in the dominant arm between January 2000 and August 2010, representing 1.6% of 572 patients treated for TOS (19% of 47 patients treated for arterial TOS). Seven were elite baseball pitchers (six professional, one collegiate), and two were professional baseball coaches with practice pitching responsibilities, with a mean age of 30.9 ± 2.9 years. Presenting symptoms included arm fatigue (five), finger numbness (four), cold hypersensitivity/Raynaud's (two), rest pain (one), and cutaneous fingertip embolism (one). Three patients underwent transcatheter thrombolysis prior to referral, including one with angioplasty and stenting. At angiography and surgical exploration 2.5 ± 0.8 weeks after symptom presentation (range, 1-8 weeks), six patients had occlusion of the distal axillary artery opposite the humeral head either at rest (three) or with arm elevation (three), one had axillary artery dissection with positional occlusion, and two had thrombosis of circumflex humeral artery aneurysms. Five patients had embolic arterial occlusions distal to the elbow. Treatment included segmental axillary artery repair with saphenous vein (n = 7; five interposition bypass grafts and two patch angioplasties), ligation/excision of circumflex humeral artery aneurysms (n = 2), and distal artery thrombectomy/thrombolysis (n = 2). Mean postoperative hospital stay was 3.8 ± 0.5 days, and the time until resumption of unrestricted overhead throwing was 10.8 ± 2.7 weeks. At a median follow-up of 15 months (range, 3-123 months), primary-assisted patency was 89%, and secondary patency was 100%. All nine patients had continued careers in professional baseball, although one retired during long-term follow-up.**

Conclusions: **Repetitive positional compression of the axillary artery can cause a spectrum of pathology in the overhead athlete, including focal intimal hyperplasia, aneurysm formation, segmental dissection, and branch vessel aneurysms. Prompt recognition of these rare lesions is crucial given their propensity toward thrombosis and distal embolism, with positional arteriography necessary for diagnosis. Full functional recovery can usually be anticipated within several months of surgical treatment, consisting of mobilization and segmental reconstruction of the diseased axillary artery or ligation/excision of branch aneurysms, as well as concomitant management of distal thromboembolism. (J Vasc Surg 2011;53:1329-40.)**

Arterial thoracic outlet syndrome (TOS) is a rare condition most frequently observed in relatively young, active, and otherwise healthy individuals.[1-4] The most typical form of arterial TOS involves development of a subclavian artery aneurysm, caused by compression of the subclavian artery within the scalene triangle at the level of the first rib. This usually occurs in conjunction with a congenital cervical rib or first rib anomaly, with formation of mural thrombus and embolic occlusion of distal arteries in the arm and/or hand. Similar pathologic and clinical findings can develop from lesions in the distal axillary artery or its immediate branches, also caused by extrinsic compression, thereby representing a variant of arterial TOS (Fig 1, *A*). Compressive lesions of the axillary artery have been occasionally reported in overhead athletes since the early 1970s, particularly in baseball pitchers and volleyball players.[5-8] The rare nature of these lesions and their unique clinical setting requires different considerations from those applicable to other forms of vascular disease.

During the past decade, our group has developed a comprehensive multidisciplinary approach to the management of all forms of TOS, including standardized protocols for the treatment of subclavian and axillary artery lesions caused by extrinsic compression. Based on this experience, the purpose of this study was to describe

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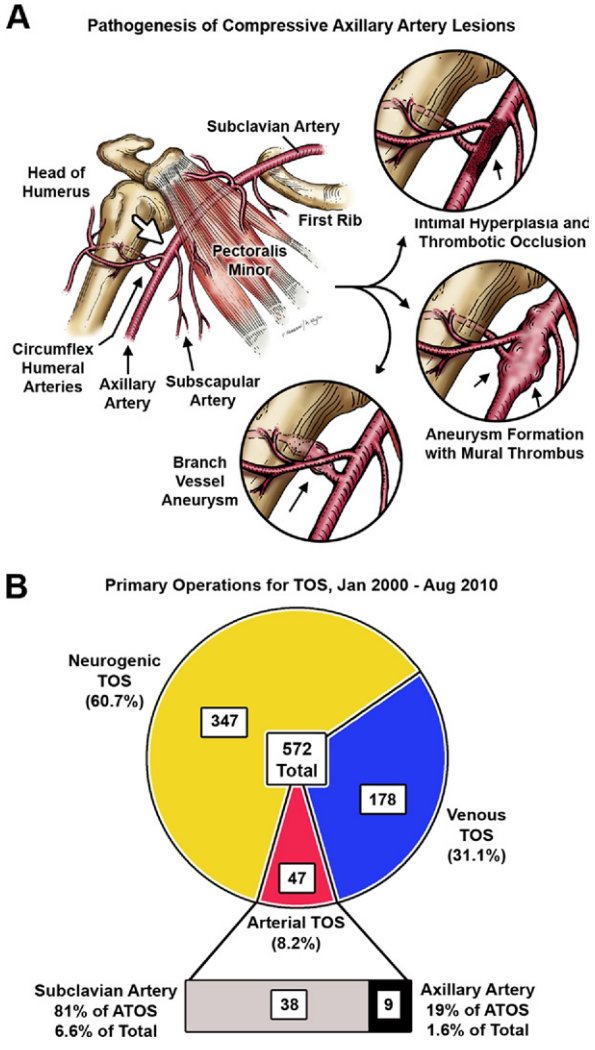

Fig 1. Pathogenesis of axillary artery lesions and patient population. **A**, Compression of the third portion of the axillary artery by anterior displacement of the humeral head during the overhead throwing motion. The axillary artery is relatively fixed in position at this site by the overlying fascia, branch vessel origins, and the pectoralis minor muscle. Repetitive compression can lead to intimal hyperplasia with stenosis, or aneurysm formation, with or without thrombotic occlusion. Similar lesions may also arise in the adjacent axillary artery branches (subscapular and circumflex humeral arteries). (Drawing by Ms. Vicki Friedman, MedPIC, Washington University School of Medicine.) **B**, Distribution of 572 consecutive patients undergoing primary operative treatment for TOS at Washington University/Barnes-Jewish Hospital from January 2000 through August 2010. The subset of patients with arterial TOS involving the subclavian and axillary arteries is indicated.

the spectrum of pathology and the outcomes of treatment for axillary artery lesions in high-performance overhead throwing athletes, with particular emphasis on the methods of surgical treatment and the subsequent return to athletic activity.

METHODS

Patients with arterial TOS were identified through a clinical database of patients treated for all forms of TOS on the vascular surgery service at Washington University School of Medicine and Barnes-Jewish Hospital (St. Louis, Mo). Patients with lesions of the axillary artery or its immediate branches were identified for review, as defined by arteriographic imaging prior to or at the time of referral and by subsequent surgical exploration. Detailed information regarding each patient was summarized from office notes, hospital charts, imaging studies, and records from treating physicians, physical therapists, and athletic trainers. Descriptive group data are presented as the mean ± standard error and/or the median and range of values. The study was approved by the Human Research Protection Office at Washington University in St. Louis.

RESULTS

Patient population. A total of 572 patients underwent primary surgical treatment for all forms of TOS between January 2000 and August 2010, including 47 patients with arterial TOS (8.2% of the total). Nine patients had primary lesions of the axillary artery or its immediate branches, representing 19% of the patients with arterial TOS and 1.6% of all patients treated for TOS during the period reviewed (Fig 1, *B*). Each of these individuals was a male overhead throwing athlete involved in competitive baseball, seven as a starting or relief pitcher (six professional, one collegiate) and two as a professional coach with daily batting practice pitching responsibilities (Table I). The mean age at the time of treatment was 30.9 ± 2.9 years (median, 31.0 years; range, 20-49 years).

Clinical presentation, diagnosis, and initial management. None of the patients had a previous thromboembolic disorder, hypercoagulable condition, vascular disease, arterial catheterization, or history of a cervical rib or first rib anomaly, and none had experienced upper extremity trauma, shoulder dislocation, or prolonged or recent use of crutches. All were actively participating in competitive baseball at the time of the onset of symptoms, and in each case, the dominant throwing arm was affected. Presenting symptoms that prompted diagnostic evaluation included pronounced arm fatigue while pitching (five patients), finger numbness (four patients), Raynaud's syndrome and cold hypersensitivity in the hand and/or fingers (two patients), rest pain in the hand (one patient), and localized fingertip discoloration consistent with cutaneous digital embolism (two patients; Table I). One of the patients had a history of intermittent finger numbness and Raynaud's syndrome for 2 years with no definitive diagnosis but had only recently developed substantial arm fatigue with throwing. None of the patients had digital ulcerations or nonhealing wounds, palpable bony abnormalities or pulsatile masses in the neck, or clinical findings suggestive of neurogenic TOS. In each case, a clinical diagnosis of upper extremity arterial insufficiency was initially suspected by the examining sports medicine orthopedic surgeon or team

Table I. Characteristics of patients with axillary artery lesions

#	*Age/Gender Side*	*Occupation*	*Presenting symptoms*	*Initial diagnosis and treatment*	*Symptom duration*
1	49 M Right	Professional baseball coach	Cold hand and arm fatigue	Arteriogram Thrombolysis Angioplasty/stent Anticoagulation	8 weeks
2	20 M Left	National Collegiate Athletic Association baseball starting pitcher	Digital emboli	Arteriogram Clopidogrel	1 week
3	29 M Right	Professional baseball starting pitcher	Finger numbness and arm fatigue	Arteriogram Anticoagulation	2 years (fingers) 2 weeks (arm)
4	32 M Right	Professional baseball relief pitcher	Arm fatigue	MR angiogram Anticoagulation	4 weeks
5	33 M Right	Professional baseball relief pitcher	Rest pain hand	Arteriogram Thrombolysis Anticoagulation	1 week
6	24 M Right	Professional baseball starting pitcher	Cold hand	CT angiogram Arteriogram Anticoagulation	1 week
7	37 M Right	Professional baseball coach	Finger numbness and arm fatigue	Arteriogram Anticoagulation	2 days
8	31 M Right	Professional baseball relief pitcher	Finger numbness	Arteriogram Anticoagulation	2 weeks
9	23 M Right	Professional baseball starting pitcher	Arm fatigue Finger discoloration	CT arteriogram Anticoagulation	3 weeks

CT, Computed tomography; *MR,* magnetic resonance.
Side affected, all represent the dominant side.
Symptom duration, time interval between the onset of symptoms and surgical treatment.

physician. Several patients had noninvasive vascular laboratory studies, including brachial-brachial indexes and forearm/digital waveforms that confirmed the clinical impression of arterial insufficiency.

Six patients underwent transfemoral upper extremity arteriography as the initial diagnostic study prior to referral, and three underwent magnetic resonance (MR) or computed tomography (CT) arteriograms (Table I). In four patients, there was complete occlusion of the distal axillary artery (Figs 2 and 3). In two patients, there was only mild distal axillary artery stenosis at rest, with positional compression and occlusion during arm elevation (Fig 4), and in another, there was a patent but narrowed axillary artery with segmental dissection and positional occlusion (Fig 5). Two patients had a normal-appearing axillary artery at rest and in arm elevation, with occlusion of the posterior circumflex humeral artery adjacent to the distal axillary artery (Fig 6).

Three of the four patients with axillary artery occlusions initially underwent an attempt at transcatheter intra-arterial thrombolysis prior to referral, with restoration of a patent axillary artery in two and inability to cross the axillary artery with a guidewire in the third. One of these individuals was also treated with balloon angioplasty and stent placement in the distal axillary artery. This patient was treated with anticoagulation and instructed to discontinue all overhead arm activities for an indefinite period of time, which obviated a livelihood in professional baseball. Although asymptomatic 2 months later, he was subsequently offered more definitive surgical treatment. Following the initial diagnostic studies and interventions, eight of the nine patients were maintained on therapeutic anticoagulation with subcutaneous low-molecular-weight heparin and/or warfarin, and one was treated solely with clopidogrel. The mean interval from the onset of symptoms until referral to our center for surgical treatment was 2.5 ± 0.8 weeks (median, 2.0 weeks; range, 1-8 weeks; Table I).

Surgical treatment. All patients were treated with a surgical protocol that began with transfemoral upper extremity arteriography, which was performed with the affected arm at rest and in the overhead elevated position (abduction and external rotation). In several instances, intravascular ultrasound was also employed to assess pathologic characteristics of the arterial wall, such as intimal thickening or aneurysmal dilatation.

Surgical exploration was performed through an upper medial arm incision (Fig 2, *B*). The distal (third) portion of the axillary artery was exposed and circumferentially mobilized from its surrounding fascia, with gentle retraction of the axillary vein and brachial plexus nerves. The axillary artery branch vessels (subscapular and circumflex humeral arteries) were also mobilized and circumferentially controlled. The location of the axillary artery lesion was identified by direct palpation and reference to the previous arteriograms, with proximal and distal control obtained where the vessel appeared normal. In seven patients, the axillary artery pathology was confined to the third portion of the vessel or its branches (lateral to the pectoralis minor muscle). In two patients, the lesion extended more proximally, such that a transverse infraclavicular incision was used to control the proximal axillary artery. In no patient was division of the pectoralis minor muscle necessary for vascular exposure.

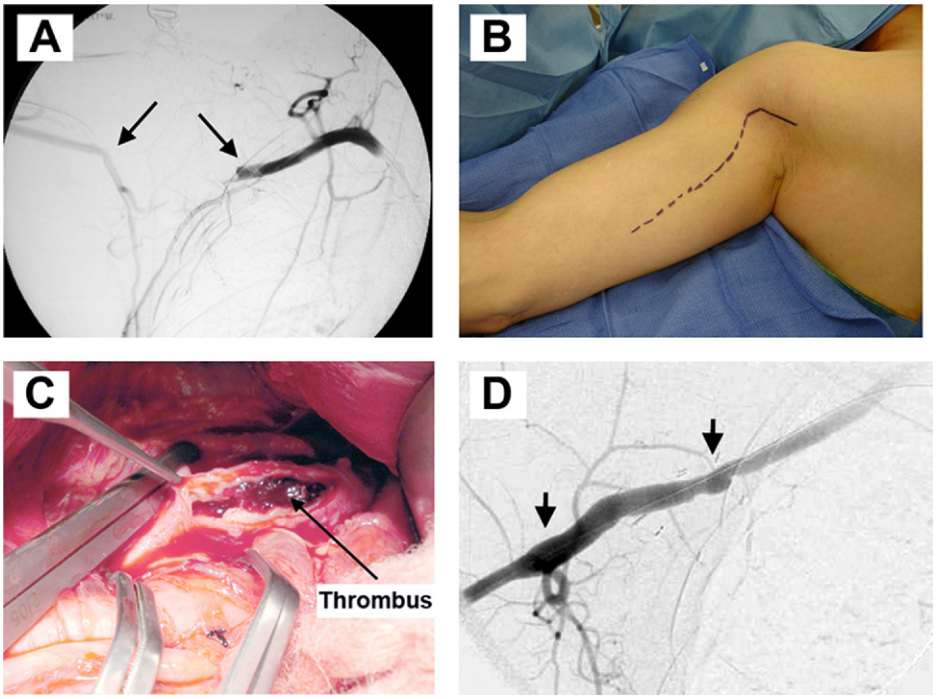

Fig 2. Axillary artery thrombosis. **A-D**, Initial arteriogram demonstrating occlusion of the right axillary artery in a 29-year-old baseball pitcher **(A)**. Surgical exploration through an upper arm incision **(B)**, revealing acute thrombosis of the distal axillary artery **(C)**, superimposed on an area of intimal thickening caused by focal intimal hyperplasia. The excised arterial segment was replaced by an interposition vein graft, illustrated on a completion arteriogram **(D)**.

Surgical pathology, summarized in Table II, included two patients with thrombotic occlusion of the axillary artery opposite the head of the humerus. Four patients had a patent axillary artery at rest with positional occlusion in arm elevation (two of which had undergone successful thrombolysis for axillary artery thrombosis before referral), and one patient had an axillary artery dissection with positional occlusion. The remaining two patients had thrombosis of axillary branch vessel aneurysms (circumflex humeral artery). There was angiographic evidence of thromboembolism distal to the elbow in five of the patients. A classification and staging system was developed to better summarize the characteristics of these axillary artery lesions, based on a combination of arteriographic findings and surgical pathology (Table III).

Direct repair of the diseased axillary artery was performed in all seven patients with lesions confined to this site (Table II). In five of these individuals, the affected segment of the axillary artery was excised, and the artery was reconstructed with a reversed saphenous vein interposition bypass graft. These bypass grafts were placed with widely beveled end-to-end anastomoses, after measuring the appropriate length of the graft while the arm was placed in overhead extension to avoid subsequent tension, torsion, or compression by the humeral head during the pitching motion. In addition, the distal anastomoses were created to incorporate at least one of the axillary artery branch vessel origins (subscapular and/or circumflex humeral arteries). In two patients, an axillary artery thrombectomy was performed followed by resection of a focal web-like intimal lesion, and reconstruction was performed with a saphenous vein patch rather than a bypass graft. The two patients found to have lesions confined to the axillary artery branch vessels underwent simple ligation and excision of the circumflex humeral artery aneurysm, without axillary arteriotomy or direct reconstruction.

All patients underwent intraoperative completion arteriography immediately following treatment of the axillary artery or branch vessel lesion. Selective injections were taken with the arm at rest and in overhead elevation, and the distal circulation was assessed from the brachial artery to the hand. No patient required revision of the axillary artery reconstruction. Two patients had brachial artery exploration and thrombectomy for distal thromboembolism, along with intra-arterial thrombolysis and vasodilator infusion into the distal vessels of the forearm. At the completion of each operative procedure, a closed-suction drain was placed into the axillary space for 24 to 48 hours, and therapeutic anticoagulation was resumed (intravenous heparin followed by conversion to warfarin) along with antiplatelet therapy (clopidogrel).

Postoperative recovery, return to activity, and secondary procedures. The mean postoperative hospital stay was 3.8 ± 0.5 days (median, 3.0 days; range, 2-7 days), with

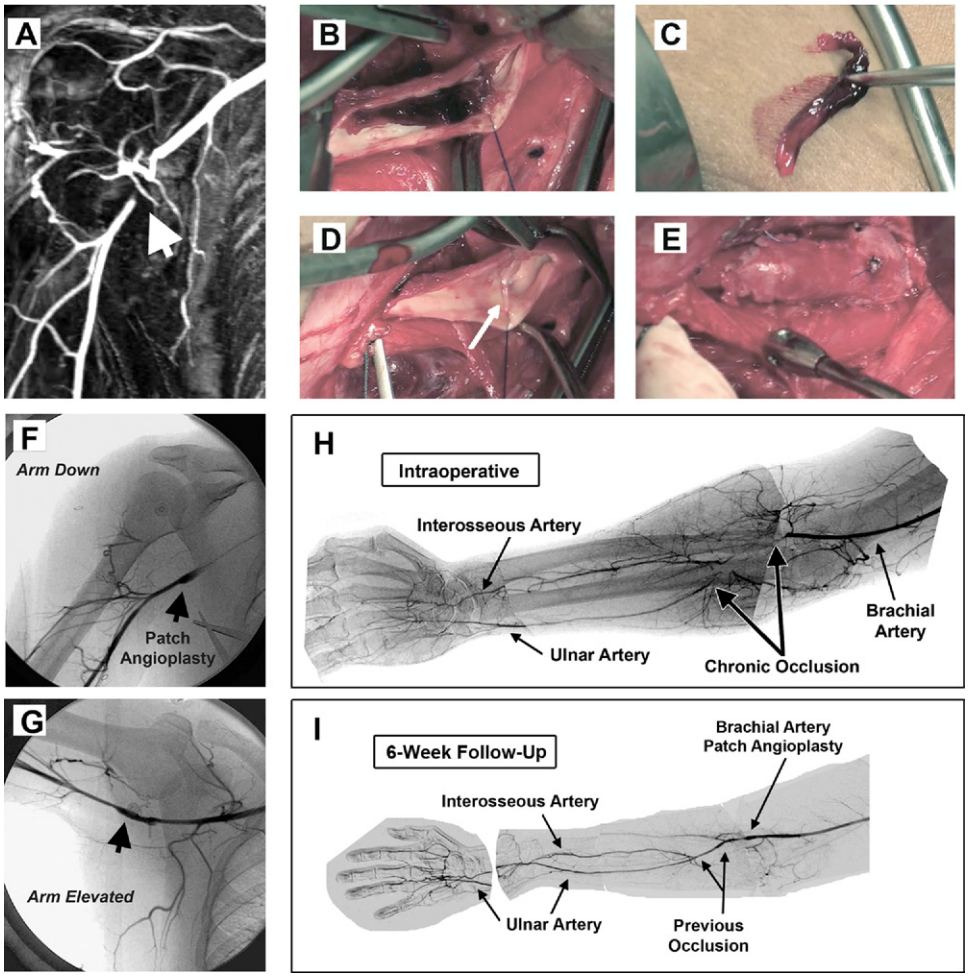

Fig 3. Axillary artery thrombosis and distal embolism. Magnetic resonance arteriogram in a 24-year-old baseball pitcher, demonstrating segmental occlusion of the distal right axillary artery **(A)**. Operative exploration revealed acute thrombosis **(B)**. The thrombus was removed **(C)**, revealing a smooth luminal surface with a focal intimal web-like stenosis **(D)**. This intimal lesion was resected, allowing repair with a saphenous vein patch angioplasty **(E)**. Completion arteriography demonstrated a widely patent axillary artery repair with the arm at rest **(F)**, and in elevation **(G)**. Concomitant embolic occlusion of the distal brachial and ulnar arteries **(H)**, treated by direct thrombectomy, intra-arterial thrombolysis, and vasodilator infusion. A follow-up arteriogram 6 weeks after surgery showed patent brachial and ulnar arteries **(I)**.

each patient having an uncomplicated recovery. All patients were maintained on postoperative anticoagulation and antiplatelet therapy for approximately 6 weeks. Physical therapy was also continued for 6 weeks, with an initial focus on restoration and maintenance of upper extremity range of motion, while restricting vigorous overhead activity and upper-body weight lifting. This was allowed to progress to upper-body strengthening and throwing beginning 6 weeks after surgery, with an emphasis on maintaining proper posture and shoulder mechanics during overhead motion.

Planned secondary imaging procedures were typically performed 4 to 6 weeks after surgical treatment, to evaluate the axillary artery reconstruction and distal circulation before the return to more vigorous upper extremity exercise. These studies included MR angiography in two patients and selective (transfemoral) arteriography in five (two patients treated early in this series did not have follow-up radiographic imaging but were assessed by clinical examination and vascular laboratory studies). In each case, the axillary artery was shown to be patent with the arm at rest and in elevated positions, with a palpable radial and/or ulnar pulse and satisfactory arterial flow in the distal arm, hand, and fingers.

Anticoagulation was discontinued 6 weeks after surgery, at which time patients were permitted more vigorous athletic rehabilitation overseen by a team physician and trainer. The mean time to a resumption of unrestricted overhead throwing (including regular baseball pitching) was 10.8 ± 2.7 weeks (median, 8.0 weeks; range, 6-32 weeks). All nine patients were initially able to resume their

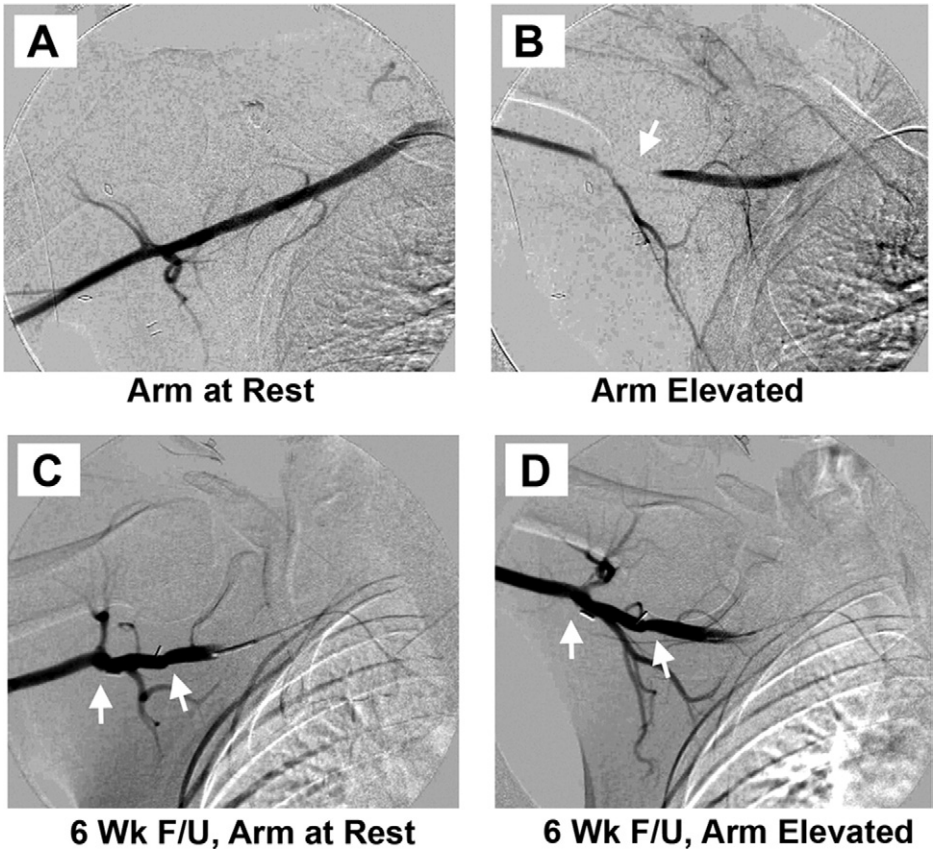

Fig 4. Positional compression of the axillary artery. **A-D**, Initial arteriogram in a 33-year-old baseball pitcher demonstrating a patent right axillary artery with the arm at rest **(A)** but compression and axillary artery occlusion with the arm in an elevated position **(B)**. Repeat arteriography 6 weeks after axillary artery repair with an interposition vein graft, demonstrating a widely patent vessel with the arm at rest **(C)** and when elevated **(D)**.

careers in collegiate or professional baseball, returning to a level of performance at least equivalent to that achieved previously.

Two of the nine patients had unplanned secondary operative procedures during follow-up. One of these individuals (patient #3) originally underwent axillary artery thrombectomy and an interposition saphenous vein bypass graft, at which time he had also been noted to have significant digital embolism. He returned to pitching in professional baseball but 2 years later, developed recurrent symptoms of finger numbness and cold hypersensitivity. He was found to have extension of distal thrombosis in the digital arteries and hand, along with mild restenosis at the proximal anastomosis of the previous bypass graft. He was successfully treated with transbrachial radial artery thrombectomy, intraarterial thrombolysis, and vasodilator infusion, as well as balloon angioplasty of the proximal anasotosis of the saphenous vein graft. He again had a successful return to professional baseball pitching, but after another 2 years had persistent symptoms attributable to digital vasospasm. As these symptoms had become refractory to conservative measures (ie, heat, vasodilators, topical nitrates), he underwent elective thoracoscopic cervical sympathectomy followed by a successful return to overhead activity.

The second patient initially had repair of the axillary artery with an extended saphenous vein bypass graft (patient #4). He developed bypass graft occlusion 1 month after the initial procedure despite satisfactory anticoagulation, related to proximal vein graft compression at the level of the first rib. This patient underwent supraclavicular thoracic outlet decompression with first rib resection, followed by subclavian-to-axillary artery reconstruction with an externally-supported polytetrafluoroethylene bypass graft. He returned to play baseball but 1 year later developed thrombotic occlusion of the polytetrafluoroethylene graft after a recent increase in pitching activity. This was successfully treated by catheter-directed thrombolysis, and he was thereafter treated with prolonged low-level anticoagulation. While able to return to pitching, he subsequently retired from professional baseball and remained asymptomatic with full use of the arm as a baseball instructor.

The median duration of postoperative follow-up was 15 months (range, 3-123 months). All patients were free of

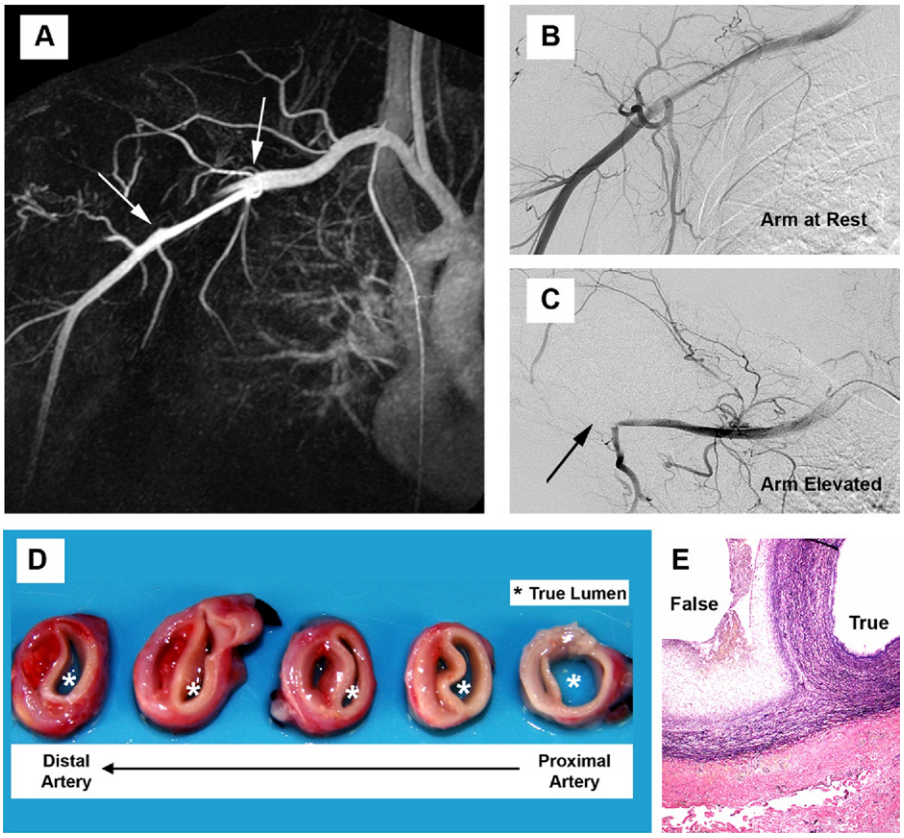

Fig 5. Axillary artery dissection. Magnetic resonance arteriogram in a 32-year-old baseball pitcher, demonstrating dissection of the right axillary artery **(A)**. Operative arteriography confirming axillary artery dissection with partial restriction in flow with the arm at rest **(B)**, and positional occlusion at the level of the distal axillary artery with the arm in elevation **(C)**. Surgical pathology **(D)** and histologic section **(E)** demonstrated extensive axillary artery dissection.

symptoms with unrestricted use of the upper extremity at the time of last follow-up. Eight of the nine patients (89%) had continued careers in professional baseball, and one had retired from the sport. With respect to the long-term status of the arterial reconstructions, the primary-assisted patency rate was 89% (8/9), and the secondary patency rate was 100%.

DISCUSSION

The overhead throwing position involves 90 to 120 degrees of shoulder abduction, excessive external arm rotation, and full wrist pronation.[9] During this maneuver, the third portion of the axillary artery is potentially subject to compression by the head of the humerus distal to the pectoralis minor muscle, where the artery is relatively fixed in position by its surrounding fascia and branch vessel origins (the subscapular and circumflex humeral arteries; Fig 1, *A*). Anterior translation of the glenohumeral joint has been demonstrated by ultrasound with the arm in the throwing position, and Stapleton et al demonstrated a correlation between this anterior translation and the degree of axillary artery compression as detected by duplex ultrasound, as well as more distal flow-mediated vasodilation.[10] Cadaveric and angiographic studies have also documented that abduction and external rotation of the arm can cause compression of the axillary artery by the humeral head, acting as a fulcrum.[11,12] Moreover, tethering of the third portion of the axillary artery can result in stretch injury to its branch vessel origins during the extremes of arm abduction and external rotation, particularly where the posterior circumflex humeral artery passes into the quadrilateral space.[13,14] Although the insertion of the pectoralis minor tendon on the coracoid process can also compress the second portion of the axillary artery, potentially contributing to this phenomenon, this is probably of little significance.[15,16]

Repetitive positional compression of the distal axillary artery or its branches has been sporadically reported in baseball players and other competitive overhead athletes, with most cases described in conjunction with various upper extremity neurovascular conditions or rare arteriopathies (Table IV). Along with the present series, review of these reports indicates that there have been 46 cases described, including 28 (61%) with axillary artery pathology and 18 (39%) with lesions of the axillary artery branch

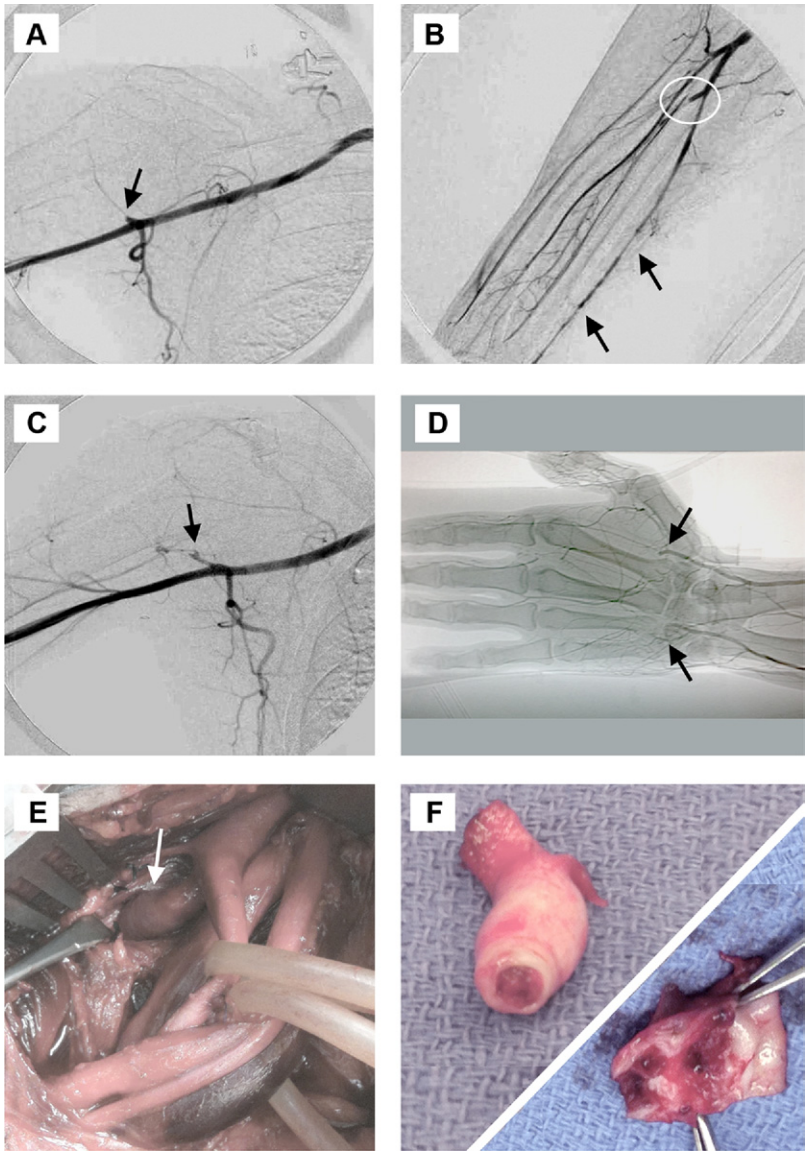

Fig 6. Axillary artery branch vessel aneurysm thrombosis. Initial arteriogram in a 37-year-old baseball coach, demonstrating occlusion of the right posterior circumflex humeral artery **(A)**, along with multiple emboli to the interosseus and ulnar arteries **(B)**. **C-F**, Initial arteriogram in a 31-year-old baseball pitcher demonstrating occlusion of the right posterior circumflex humeral artery **(C)**, with embolic occlusion of the radial and ulnar arteries in the hand **(D)**. Operative exploration demonstrated a branch vessel aneurysm **(E)**, which was ligated and excised **(F)**.

vessels, with the majority (70%) occurring in baseball players. It is evident that these lesions are typically caused by chronic vessel wall injury and frequently associated with intimal hyperplasia, stenosis, and thrombus formation, as well as distal embolization to smaller vessels. More abrupt and substantial axillary artery injury may result in acute thrombotic occlusion or dissection, and progressive medial degeneration can lead to aneurysm formation in the axillary artery or its branches. Although small in size, turbulent flow in these aneurysms readily produces mural thrombus that has a high propensity to embolize to the distal extremity, particularly during repetitive movement at the level of the shoulder joint.

It appears likely that this condition is under-recognized, in part because symptoms resembling arm claudication, digital numbness, and circulatory changes are not uncommon in high-performance overhead throwing athletes, and in this setting, such symptoms are often initially attributed to fatigue and musculoskeletal etiologies. Due to the rich collateral network surrounding the shoulder, acute axillary artery occlusion rarely presents with limb-threatening ischemia. Significant arm claudication, however, is a disabling

Table II. Surgical pathology and treatment in patients with axillary artery lesions

#	*Classification*	*Surgical pathology*	*Emboli*	*Operative procedure(s)*
1	Type II Stage 0	AxA patent + compression[a] In-stent restenosis Intimal hyperplasia	No	Segmental AxA (stent) excision Interposition SV graft
2	Type I Stage 0-D	AxA patent + compression Luminal surface ulceration	Yes	Segmental AxA excision Interposition SV graft
3	Type II Stage 3-D	AxA occlusion/thrombosis Intimal hyperplasia	Yes	Segmental AxA excision Interposition SV graft Completion arteriogram
4	Type II Stage 0	AxA dissection + compression Intimal hyperplasia	No	Segmental AxA excision Interposition SV graft Completion arteriogram
5	Type II Stage 0	AxA patent + compression[a] Intimal hyperplasia	No	Segmental AxA excision Interposition SV graft Completion arteriogram
6	Type II Stage 2-D	AxA occlusion/thrombosis Intimal hyperplasia (web)	Yes	AxA thrombectomy SV patch angioplasty BA and UA thrombectomy Arteriogram/thrombolysis
7	Type IV Stage 3-D	AxA patent, CHA aneurysm Occlusion/fresh thrombus	Yes	Aneurysm excision/ligation BA and UA thrombectomy Arteriogram/thrombolysis
8	Type IV Stage 2-D	AxA patent, CHA aneurysm Occlusion/fresh thrombus	Yes	Aneurysm excision/ligation Completion arteriogram
9	Type II Stage 0	AxA stenosis/thrombosis Intimal hyperplasia (web)	No	AxA thrombectomy SV patch angioplasty Completion arteriogram

AxA, Axillary artery; *BA,* brachial artery; *CHA,* circumflex humeral artery branch; *RA,* radial artery; *SV,* saphenous vein; *UA,* ulnar artery.
Type and stage, see Table III.
Emboli, clinically evident digital emboli or arteriographically evident distal arterial occlusions (brachial, radial, ulnar, palmar arch, or digital arteries).
[a]Patient had successful thrombolysis of an occluded axillary artery prior to referral.

Table III. Classification and staging of axillary artery lesions

Lesion type	*Arteriographic findings and surgical pathology*
Type I	Patent axillary artery with positional compression alone
Type II	Fixed axillary artery stenosis or occlusion ± positional compression
Type III	Axillary artery aneurysm formation ± positional compression
Type IV	Axillary artery branch vessel aneurysm or occlusion (circumflex humeral or subcapsular arteries)

	Lesion stage (Distal arteriographic findings)		
	Brachial artery across elbow	*Radial and ulnar arteries through forearm and wrist*	*Palmar arch through hand*
Stage 0	Patent	Both patent	Patent
Stage 1	Occluded	Both patent	Patent
	Patent	One occluded	Patent
Stage 2	Occluded	One occluded	Patent
	Patent	Both occluded	Patent
Stage 3	Occluded	Both patent	Interrupted
	Patent	One or both occluded	Interrupted
Stage 4	Occluded	One or both occluded	Interrupted
"D" modifier	Presence of digital artery emboli or occlusions		

symptom in the throwing athlete. Embolism to the end arteries in the hand and digits may also result in disabling numbness, pain, and occasional fingertip ulcerations. Cold temperature hypersensitivity is also a common symptom and can be misinterpreted as vasospasm in the presence of normal radial and ulnar pulses. Collegiate and professional athletes are usually first evaluated by their team physicians, who need to have a high index of suspicion when these symptoms arise acutely or when they are associated with pulse deficits, pallor, or differences in temperature. The variety of presenting symptoms noted in this current series reinforces the need for team physicians to remain vigilant in evaluating upper extremity complaints in baseball pitchers.

Noninvasive vascular laboratory testing may be useful in diagnosis, as brachial-brachial indexes may be abnormal with axillary artery occlusion, and digital waveforms may be dampened in the cases of thromboembolism. These studies can be deceptive, however, as normal results may be obtained in the resting position when there is only positional compression of the axillary artery. Plain films may reveal cervical ribs in cases of subclavian artery compression in the thoracic outlet, but are normal in cases of axillary artery compression. Angiography remains the most important and essential diagnostic study, whether performed by catheter-based techniques or contrast-enhanced MR or CT. Angiograms should be performed with the arm both at rest and in the overhead throwing position (90 to 120

Table IV. Axillary artery and branch vessel lesions in competitive athletes

Year	*Author (reference)*	*No. of patients (sport)*	*Types of lesions*
1989	McCarthy[21]	4 patients (three BB, one SB)	3 AxA positional compression 1 branch artery occlusion
1990	Nuber[6]	6 patients (BB)	5 AxA positional compression 1 branch artery occlusion
1990	Rohrer[11]	1 patient (BB)	1 AxA thrombosis
1993	Reekers[23]	1 patient (VB)	1 branch artery aneurysm
1995	Kee[24]	2 patients (BB)	2 branch artery aneurysms
1998	Todd[25]	2 patients (BB)	2 AxA aneurysms
1999	Schneider[7]	1 patient (BB)	1 AxA aneurysm
2000	Caiati[26]	1 patient (TN)	1 AxA dissection
2000	Ikezawa[27]	2 patients (one VB, 1 TN)	1 branch artery aneurysm 1 branch artery thrombosis
2001	Arko[18]	5 patients (NA)	1 AxA aneurysm 4 branch artery aneurysms
2001	Ishitobi[22]	4 patients (BB)	4 AxA thrombosis
2001	Vlychou[28]	1 patient (VB)	1 branch artery aneurysm
2006	McIntosh[29]	2 patients (VB)	2 branch artery aneurysms
2006	Simovitch[15]	1 patient (BB)	1 AxA positional compression
2006	Takach[30]	1 patient (BB)	1 AxA positional compression
2007	Baumgarten[19]	1 patient (BB)	1 branch artery aneurysm
2008	Seinturier[31]	1 patient (NA)	1 branch artery aneurysm
2009	Ligh[17]	1 patient (BB)	1 AxA positional compression
2010	Present report	9 patients (BB)	1 AxA positional compression
Totals		46 patients (32 BB, 70%) (1 SB, 2%) (5 VB, 11%) (2 TN, 4%)	12 AxA positional compression (26.1%) 10 AxA thrombosis (21.7%) 4 AxA aneurysms (8.7%) 2 AxA dissection/compression (4.3%) 3 branch artery occlusions (6.5%) 15 branch artery aneurysms (32.6%)

AxA, Axillary artery; *BB,* baseball; *NA,* not available; *SB,* softball; *TN,* tennis; *VB,* volleyball.

degrees shoulder abduction, full external rotation), with good visualization of the axillary artery, its branches, and the more distal vessels.

Successful nonoperative treatment with physical therapy has been described in one case of positional compression of the axillary artery.[17] However, even in the published literature, there are many examples of failed nonoperative management resulting in recurrent symptoms, progression of the disease process, and significant disability from thrombus propagation or distal embolization.[18-20] Thrombolytic therapy alone, without treatment to address the underlying lesion and its cause, can also be predicted to lead to recurrence.[11] One patient in our series had undergone thrombolysis, angioplasty, and stenting prior to his referral to our institution, with unacceptable restrictions on activity and in-stent restenosis only 8 weeks after the initial intervention. Given the likelihood that vigorous overhead arm activity will accelerate axillary artery restenosis, neither angioplasty or stent placement can be recommended in this patient population.

Surgical approaches provide optimal treatment for the entire spectrum of compressive lesions of the axillary artery and its branches. This initially involves operative exposure of the second and third part of the axillary artery to help mobilize the vessel from its surrounding fascia. Although some authors have described pectoralis minor tenotomy as the procedure of choice for decompressing the distal axillary artery,[15,21] we have not found this necessary or sufficient since the underlying pathology lies distal to the muscle tendon. As performed in most of our patients, the diseased arterial segment should be removed as a potential source of thromboembolism. Although extra-anatomic bypass has been described as an alternative approach in a small series of patients,[22] placement of such grafts does not remove or exclude the damaged axillary artery, particularly if constructed with end-to-side anastomoses. When resection of a portion of the axillary artery is necessary, it is ideally repaired with a reversed saphenous vein interposition bypass graft; however, when intimal resection is sufficient to restore a suitable axillary artery lumen, repair with a wide vein patch angioplasty may be a useful alternative. When a vein graft is used, it is measured to have enough redundancy to prevent recurrent compression, and the absence of compression with arm elevation is confirmed by completion arteriography. If the source of embolism is an axillary artery branch vessel, such as the subscapular or circumflex humeral arteries, the affected branch can be ligated and excised without the need for direct replacement. Despite the attraction of endovascular approaches to these lesions, this may lead to obstruction of crucial axillary artery branches or the potential for further embolism and cannot be recommended. Antecedent digital embolism can usually be managed with intraoperative transbrachial thrombectomy of the forearm vessels coupled with intraar-

terial thrombolytic therapy and vasodilator infusion. Anticoagulation is continued postoperatively and maintained for 6 weeks, after which antiplatelet therapy with clopidogrel or aspirin is continued. Physical therapy is started on the first postoperative day with passive and active range-of-motion exercises, and a transition to athletic rehabilitation over a period of 4 to 6 weeks.

Outcome after surgical repair is very favorable. This is illustrated by the observation that most of our patients resumed unrestricted physical activities within 2 to 3 months, and all were able to resume their baseball careers. Persistent finger sensory deficits as a result of chronic digital arterial occlusion may nonetheless continue to limit the tactile ability that is necessary for control of pitches and thereby the overall success of the professional baseball pitcher. Thus, delayed diagnosis can be a limiting factor in the recovery process, particularly if chronic digital embolism is present prior to surgical treatment.

CONCLUSIONS

Repetitive positional compression of the axillary artery is a rare but important problem in the elite overhead throwing athlete. It can cause a spectrum of pathology, including focal intimal hyperplasia, aneurysm formation, segmental dissection, and branch vessel aneurysms or occlusions. Prompt recognition of these lesions is crucial, given their propensity toward thrombosis and distal embolism. Positional arteriography is necessary for accurate diagnosis, and thrombolysis may be useful as initial management when axillary artery thrombosis is present, but balloon angioplasty and placement of axillary artery stents should be avoided. Anticoagulation should be instituted upon confirmation of the diagnosis, along with referral for surgical treatment as soon as feasible. Full functional recovery can be anticipated within several months following surgical treatment, consisting of mobilization and segmental reconstruction of the diseased axillary artery, or ligation and excision of branch artery aneurysms, as well as concomitant management of any distal thromboembolism.

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AUTHOR CONTRIBUTIONS

Conception and design: YD, VE, MD, JE, RW, GP, RT
Analysis and interpretation: YD, VE, RT
Data collection: YD, VE, RT
Writing the article: YD, VE, RT
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