Int J Clin Exp Med 2018;11(6):6351-6357
www.ijcem.com /ISSN:1940-5901/IJCEM0074545

Case Report

Long-term follow-up of seven patients with crutch-induced upper limb ischemia

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Received February 6, 2018; Accepted March 16 2018; Epub June 15, 2018; Published June 30, 2018

Abstract: Objective: Crutch-induced upper limb ischemia is a serious complication experienced by crutch users. Here, we report seven cases of crutch-induced upper limb ischemia after long-term observation, and describe our management experience in these patients. Materials and methods: We retrospectively reviewed the clinical and imaging data of 7 patients with crutch-induced upper limb ischemia, who were treated with surgery (1) or intravenous anti-coagulant and thrombolytic therapy (6) from Dec 2009 to Oct 2016. Results: All the patients were followed-up by postoperative phone call and in the outpatient clinic, including evaluation of relief of symptoms, arterial pulses, grip strength, and surveillance with Doppler ultrasound or computed tomography angiography (CTA). All the patients were followed-up monthly for 3 months after discharge, then every 6 months, and yearly thereafter. The ischemic symptoms were relieved to some extent in all 7 patients and no hemorrhagic complications related to anti-coagulation or anti-platelet therapy occurred during the follow-up period. Conclusions: The choice of therapy for crutch-induced upper limb ischemia should be based on the patient's specific situation. Conservative treatment for crutch-induced upper limb ischemia could be used to manage patients without signs of limb necrosis, without increasing the risk of amputation. Surgical treatment should be considered if there is aneurysm/pseudoaneurysm formation or if there is a high risk of amputation.

Keywords: Crutch-induced, upper limb, ischemia, follow-up, surgery, anticoagulant

Introduction

Acute or chronic upper limb ischemia is rare compared to lower limb ischemia [1]. The proportion of patients with upper limb ischemia is approximately 15-18% of all limb ischemia patients [2]. Cardiogenic embolism has been recognized as the main cause of upper limb ischemia. However, many other non-cardiac disorders, such as iatrogenic trauma, blunt trauma, thoracic outlet syndrome, and Raynaud's syndrome have also been identified as sources of upper limb ischemia. Long-term use of axillary crutches is a cause often easily ignored by the examiner. Prolonged use of axillary crutches may result in repetitive trauma, which could cause the degeneration and disruption of the tunica intima and tunica media of the axillary artery, resulting in the formation of an aneurysm or stenosis. Pathological examination of the axillary artery often shows fragmentation of the intima and adventitia related to a perivascular fibrous reaction [3]. Formation of aneurysms or stenosis and the disruption of the tunica intima may result in axillobrachial thromboembolic disease. Therapeutic methods include conservative treatment, surgical operations, and interventional therapy.

In this study, we report seven cases of patients with crutch-induced upper limb ischemia after a long term follow-up observation. Six of the patients received conservative treatment and one underwent surgical operation. We describe our management experiences of these patients.

Materials and methods

This retrospective study was approved by the Ethics Committee of our hospital, and all the patients in the study signed the consent form. We retrospectively reviewed the clinical and

Table 1. Clinical presentation of seven patients with crutch-induced upper limb ischemia

Variable	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7
Gender	Male	Male	Male	Male	Female	Male	Male
Age (years)	52	50	45	53	32	46	59
Weight (kg)	75	58	63	67	50	64	69
Causes of using crutch	Poliomyelitis	Poliomyelitis	Poliomyelitis	Lower limb necrosis	Poliomyelitis	Poliomyelitis	Lower limb amputation
Duration of using crutch (years)	46	37	40	50	22	40	45
Way of using crutch	Bilateral	Right	Right	Right	Right	Right	Right
Symptoms and Duration	Forearm pain, numb, and weakness, 7 days	Forearm swelling and pain, 5 days	Forearm pain, 20 days	Forearm pain and numb, 2 months	Forearm chills and weakness, 1 year	Forearm numb, 8 days	Forearm pain and numb, 1 month
Physical examination	CTD, LE, Swelling tenderness	CTD	CTD, HED	CTD, DGOF	CTD, FC	CTD	CTD
Absence of pulse	AA, BA, RA, UA	AA, BA, RA, UA	BA, RA, UA	BA, RA, UA	RA, UA	AA, BA, RA, UA	BA, RA, UA
Grip strength	0	3	2	1	3	3	3
D-Dimer	1.55 μg/mL	2.26 μg/mL	1.43 μg/mL	1.09 μg/mL	0.49 μg/mL	1.13 μg/mL	0.42 μg/mL
Results of CTA	Thrombosis of AA	Stenosis and thrombosis of AA	Occlusion of BA	Occlusion of AA	Occlusion of RA	Stenosis and occlusion of AA	Stenosis and occlusion of BA

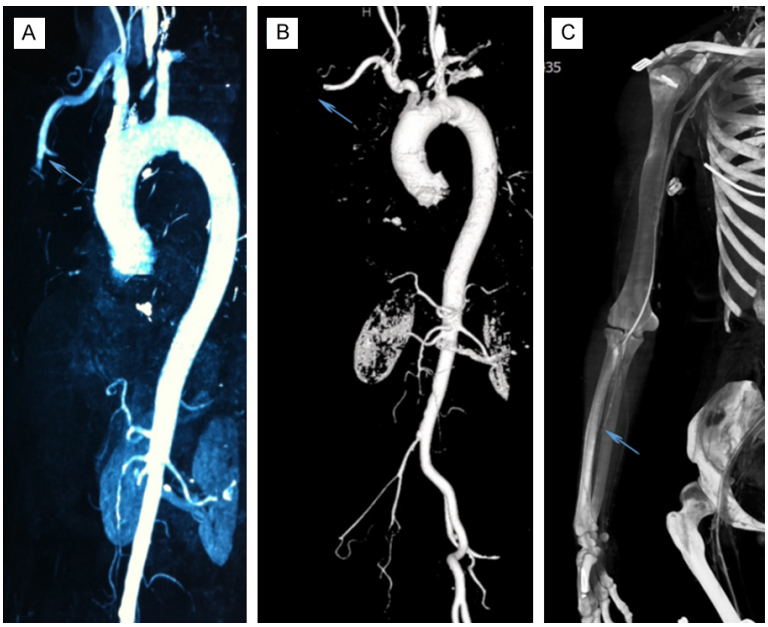

Figure 1. Preoperative CTA results of patient 1, 4, and 5. A-C: CTA showed thrombosis and occlusion of axillary artery (arrow) in patient 1 and patient 4 respectively, and occlusion of radial artery in patient 5.

imaging data of 7 patients with crutch-induced upper limb ischemia from the Vascular Surgery Department of the Second Xiangya Hospital from December 2009 to October 2016. All the patients were in accordance with the following inclusion criteria: 1. presented with acute or chronic upper limb ischemia; 2. use of axillary crutches as walking aid for at least 1 year; 3. no previous history of thromboembolic disease; 4. no evidence of cardiogenic emboli based on echocardiogram (echo) or ultrasonic cardiogram, nor of emboli caused by other diseases; 5. detection of axillobrachial artery vasculopathy by computed tomography angiography (CTA) or Doppler ultrasound.

There were 7 patients, including 1 female and 6 male patients, with a history of axillary crutch use for 22 up to 50 years. All the patients exhibited typical ischemic symptoms, including decreased cutaneous temperature, pain, pallor, weakness, paresthesia, and low pulse rate. The time from onset of ischemic symptoms to admission varied from 5 days to a year. Three of them presented as acute upper limb ischemia, while 4 patients presented as subacute or chronic ischemia. The clinical presentation of these patients is summarized in **Table 1**. No previous history of deep venous thrombosis was found in any of the patients. Of the 7 cases, 3 representative cases with well-documented data are described in detail in the following section.

Patient 1 was a 52-year-old man who suffered sudden onset of pain, numbness, and weakness of the forearm for 7 days. He had a long history of poliomyelitis and had to use bilateral

axillary crutches at the age of 6 years old. Upon examination, his right hand was obviously cold and pale, and the capillary return was delayed. He had poor grip strength. Forearm swelling and local ecchymosis were detected as well. Sensation was decreased over the fingers. Right axillary, brachial, ulnar, and radial pulses were not observed. Chest radiography showed no scoliosis and no evidence of a bony cervical rib. Electrocardiography (ECG) revealed unremarkable findings. D-Dimer was higher than the normal level. CTA showed axillary artery thrombosis or occlusion (**Figure 1A**). Considering the severity and persistence of symptoms in the patient, surgery of the axillary artery was performed.

Patient 4 was a 53-year-old man who had a history of right forearm pain and numbness for 2 months. One month later, the pain and numbness progressed. Physical examination showed dry gangrene of the distal ring finger, small finger, and thumb. The CTA showed axillary artery occlusion (**Figure 1B**). We suggested axillary artery exploration because of the severity of finger necrosis, but the patient refused. Interestingly, after a 14-day anti-coagulation therapy in combination with vasodilators, the ischemic symptoms were alleviated and there was no further progression of the necrosis in the fingers during the hospitalization.

Patient 5 was a 32-year-old woman who suffered hand chills and weakness for a year. The symptoms would worsen after a long walk with crutches or during cold weather. On examination, the fingers on the right hand were cyanotic but had no necrosis. The ulnar pulse was palpable while the radial pulse was absent. Doppler ultrasound and CTA showed radial artery thrombosis (**Figure 1C**). Her heart sounds were normal, without murmurs. ECG revealed normal sinus rhythm. D-Dimmer, protein C, and protein S were normal. After anti-coagulation and vasodilator treatment, she had an obvious improvement in hand chills and grip strength.

Treatment

In this series, all the patients received anticoagulant therapy with low molecular weight heparin (5,000 IU q12h) and vasodilator treatment with alprostadil (20 μg BID) intravenously. The duration of therapy ranged from 7 to 13 days during hospitalization. Short medical histories and high levels of D-Dimer were observed in patients 1, 2, and 3, which indicated the possibility of acute axillary artery thrombosis. Therefore, intravenous thrombolytic therapy (Urokinase 200,000 IU BID) was administered in these 3 patients to dissolve the occluding thrombus and improve the ischemic symptoms. While the ischemic symptoms of patient 1 diminished after a day of intravenous thrombolytic therapy, consideration of the severity of the symptoms and the high risk for limb necrosis prompted surgical exploration of the axillary artery in the patient. Intraoperative findings included a 2 cm intimal dissection and thrombosis in the axillary artery. The intima of the distal axillary artery was fractured and the thinned-out wall of artery protruded outward like a micro-pseudoaneurysm. Fresh clots were retrieved from the proximal and distal axillary artery with a 3 F Fogarty balloon catheter, after which pulsatile blood flow was noted from the proximal side and retrograde flow detected from the distal side. We performed pseudoaneurysm resection and intimal fixation as planned, and injected 200,000 IU urokinase to the distal side of the brachial artery from the Fogarty catheter. The radial pulse was soon palpable and the patient's hand became warm postoperatively.

For prevention of recurrence of thrombosis after operation, warfarin was given in patients 1, 2, and 6 (2.5 mg PO qday) after discharge to maintain an international normalized ratio (INR) between 2.0 and 3.0 for 3 months, while the other 4 patients were treated with clopidogrel (75 mg/day). In addition, all 7 patients took Beraprost Sodium Tablets (40 μg PO TID) for 3 months. Four of 7 patients took mecobalamine (0.5 mg PO qday) to relieve numbness. All 7 patients were likewise advised to stop using axillary crutches and choose elbow-supporting crutches or wheelchairs, and to perform rehabilitation exercises (hand-clenching movements) simultaneously.

Follow-up

Follow-up was performed by postoperative phone call and outpatient clinic visits, with evaluation of relief of symptoms, arterial pulse, and grip strength, and surveillance by Doppler ultrasound or CTA. All the patients were followed-up at monthly intervals for 3 months after discharge.

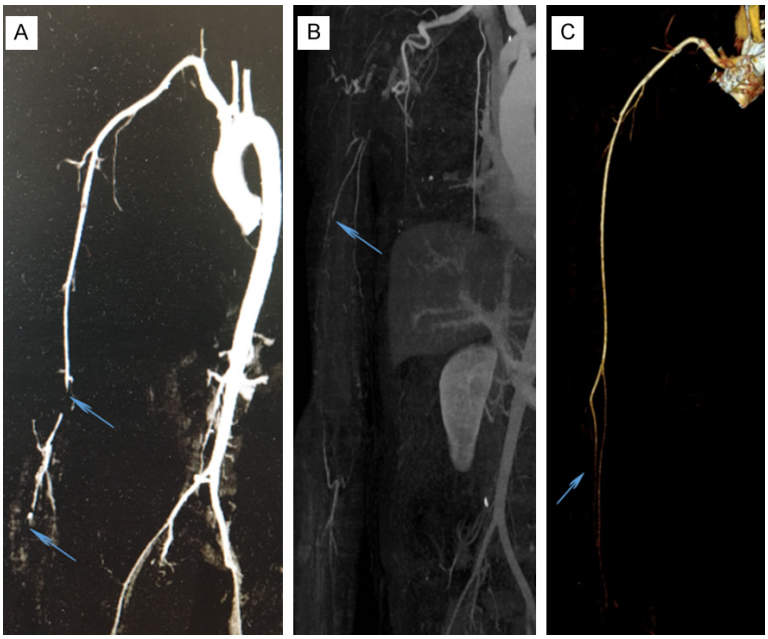

Figure 2. Follow-up CTA results of patient 1, 4, and 5. A: The 48th month postoperative CTA showed that multiple arterial thrombosis (arrow). B: The 12th month CTA revealed an establishment of collateral circulation in patient 4. C: The CTA results of 24th month demonstrated a poor filling in distal side of radial artery (patient 5).

Results

No patients were lost to follow-up and the follow-up duration ranged from 13 months to 84 months. The ischemic symptoms were relieved to some extent in all patients and no hemorrhagic complications related to anticoagulation or antiplatelet therapy occurred during the follow-up period. Cutaneous color of all the patients returned in 3-6 months. Cutaneous temperature in 6 patients (except patient 4) were restored in 12 months. Five patients had hand chills during cold weather. Rest pain resolved in all patients, but patient 5 still had hand tingling after a long walk on axillary crutches. All patients had normal sensory function without numbness of the hand or fingers. Grip strength increased to a large degree. The ulnar pulse was palpable in patient 5. Follow-up CTA showed that thrombosis of multiple arteries occurred in patient 1 (**Figure 2A**), while establishment of collateral circulation was detected in the other patients (**Figure 2B**). Patient 5 had poor perfusion to the area supplied by radial artery distally, but had normal perfusion on the ulnar artery (**Figure 2C**). Although all the patients were advised to stop using axillary crutches, only patient 3 and patient 7 started using a wheelchair and bilateral elbow-supporting crutches as walking aid, respectively. There was no recurrence of upper limb ischemia in these patients except in patient 1, who kept using axillary crutches after discharge. The details of the results of the follow-up are summarized in **Table 2**.

Discussion

Upper limb ischemia is an uncommon disease compared to lower limb ischemia. Most cases of upper limb ischemia are caused by cardiogenic emboli. Rare causes of upper limb emboli include proximal subclavian artery aneurysms and thoracic outlet syndrome [4]. The percentage of upper limb emboli is less than 20% of all emboli, and of these, 70% are cardiac while the remaining 30% are subclavian in origin [5]. Cardiac emboli often occur after myocardial infarction, originating from ventricular aneurysms and atrial fibrillation. These emboli tend to lodge at the brachial artery bifurcation, and smaller emboli may lodge more distally [1, 6]. Cardiogenic factors should be considered as the most likely etiology in patients with upper limb ischemia. Ultrasonic cardiogram could be helpful in identifying the existence of cardiac emboli. Crutch-induced arterial injury should be suspected in a patient with a long history of using a crutch and presenting with acute or chronic upper limb ischemia. Long-term use of axillary crutches could result in cumulative pressure, loading to the axillary and brachial arteries, and resulting in a high risk of developing axillary and/or brachial artery stenosis, occlusion, aneurysms, and secondary thromboembolic episodes in the upper limb.

To the best of our knowledge, the first case of crutch-induced vascular injury was reported by Ryle in 1922 [7]. Since then, at least 47 cases have been reported from more than 10 countries [7-11]. Crutch-induced vascular injury is often represented by sudden or chronic ischemia of the upper limb, with symptoms of upper

Table 2. Follow-up results of seven patients with crutch-induced upper limb ischemia

Variable	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7
Follow up duration (months)	53	84	15	13	26	39	24
Therapeutic schedule	TSA	TSB	TSC	TSC	TSC	TSB	TSC
Cutaneous color	Normal	Normal	Normal	Normal	Normal	Normal	Normal
Cutaneous temperature	Normal	Normal	Normal	Slightly low	Normal	Normal	Normal
Hand chills	Yes	Yes	No	Yes	Yes	Yes	No
Rest pain	No	No	No	No	No	No	No
Numbness of hand or fingers	No	No	No	No	No	No	No
Grip strength	3	5	5	4	4	4	5
Pulse of arteries	AA, BA	Non	AA	AA	AA, BA, UA	Non	AA
Walking aid	BAC	RAC	Wheelchair	RAC	RAC	RAC	BEC

limb pain, pallor, weakness, paresthesia, and low pulse rate, and finger or hand gangrene occasionally seen in these patients [6, 12]. Certainly, severe ischemia will result in limb necrosis, requiring amputation [13]. The most common cause of crutch use is poliomyelitis, however other causes include lower limb amputation, severe infection [14], osteomyelitis, and rheumatoid arthritis [15]. Most of the patients with crutch-induced arterial injury have used crutches for decades. The longest duration for using axillary crutches was 67 years [16], although there was also a patient who had been using crutches for only 2 months [17] . As we know, the integrity of the endothelium of the blood vessels is imperative for normal flow. Intimal damage is an activation of thrombogenesis. However, intimal damage is not sufficient to produce thrombotic occlusion of the vessel without sluggishness of flow. Therefore, if intimal damage has occurred, and the blood vessel has been persistently compressed by external pressure, it will result in thrombosis or occlusion of the artery. It is not clear why the ischemic symptoms occur after decades of using crutches. However, the intimal dissection in patient 1 clearly indicates a traumatic pathogenesis. The repetitive blunt trauma also causes disruption and degeneration of the tunica intima and tunica media, which could lead to the formation of an aneurysm [18, 19]. Among the reported cases, at least 22 cases were identified with axillobrachial aneurysms, and there was no reported case of rupture [7, 9, 11, 20]. The percentage of axillobrachial artery aneurysm formation among all reported cases is as high as 46.8%, while the ratio is 27.3% in Asian countries, and 52.8% in non-Asian countries (most of them are Western countries). In our series, no typical axillobrachial artery aneurysm was detected by CTA in all patients. It was only through intraoperative exploration that slight aneurysmal dilation was found in patient 1. We believe that the formation of axillary artery aneurysms may be related to age, arteriosclerosis, and duration of crutch use. Body weight may likewise be another factor, as Westerners tend to be heavier compared to Asians.

It is not difficult to diagnose crutch-induced upper limb ischemia. Acute or chronic upper limb ischemia with a long-term history of axillary crutch use, without any history of deep venous thrombosis, and exclusion of other significant cardiovascular risk factors, support the diagnosis of crutch-induced upper limb ischemia. CTA and Doppler ultrasound are the most useful tools for diagnosing such vasculopathy.

Treatment for crutch-induced upper limb ischemia concentrated on the prevention of limb loss or dysfunction. Treatment methods include open surgery, interventional therapy, and conservative therapy. Surgical treatment includes arterial exploration [21] (thrombectomy + arterial reconstruction), sympathetic block [22], and sympathetic ganglionectomy [23]. Sympathetic block and sympathetic ganglionectomy were more commonly performed in the past. It helps in relieving symptoms, but it is not effective in removing the etiology. Arterial exploration is suggested upon the discovery of an axillary artery aneurysm or pseudoaneurysm, even if asymptomatic, and is also recommended for patients with severe ischemia at high risk of limb necrosis. Axillary or brachial

artery exploration will help determine the exact cause of limb ischemia, and adopt appropriate treatment strategies according to the intraoperative findings. Through axillary artery exploration, we observed thrombosis, intimal fracture, and dissection in patient 1. The fractured intima could result in recurrence of thrombosis. Therefore, we performed thrombectomy with Fogarty balloon catheter and intimal fixation simultaneously. Song Jinhua [24] reported 6 cases of crutch-induced axillary artery occlusion who underwent catheter thrombolysis treatment and angioplasty. There was a patient who was readmitted due to recurrence after 3 months, and treated with stent implantation. However, the outcomes of this patient were not stated. In our opinion, angioplasty or stent implantation is not recommended for patients with crutch-induced axillobrachial injury, due to the pathological features of the disease. Endovascular procedures may aggravate the intimal damage, and easily cause recrudescence. Catheter-directed thrombolysis could effectively dissolve the acute thrombus. However, it still cannot remove the lesion, and is associated with a high risk of hemorrhagic complications compared to intravenous thrombolytic therapy. For patients with thrombosis or occlusion but without any severe ischemic symptoms, intravenous anticoagulant and thrombolytic therapy may be a safe and less invasive alternative.

Long-term anti-coagulant or anti-platelet therapy and relief of compression or mechanical injury to the axillary artery were crucial to these patients. The anti-coagulant and anti-platelet were used to prevent the occurrence of thrombosis in the patients. In our series, all the patients received vasodilator therapy combined with anti-coagulant or anti-platelet therapy for 3 months. It is clear that ischemic symptoms improve after conservative treatment during follow-up observation. Although arterial pulses could not recovered and sensorimotor function was not affected. Based on our experience, we recommend anti-coagulant therapy for acute ischemia (<2 weeks), and anti-platelet therapy for subacute or chronic ischemia (>2 weeks) after discharge. Almost every expert recommends that patients with crutch-induced vascular injury should change the way they use crutches, however, this is often not feasible, because the patients rely too much on crutches. Although all patients were advised to use non-axillary crutches, there were only 2 patients who shifted to using bilateral elbow-supporting crutches or a wheelchair. During follow-up observation, these 2 patients had more powerful grip strengths compared to others, which may be related to the release of the axillary artery compression upon use of wheelchair or elbow-supporting crutches. One patient who kept using axillary crutches after discharge was readmitted due to multiple arterial thr-omboses.

Performing rehabilitation exercises, like hand-clenching movements, may promote collateral circulation, improve grip strength, and relieve the symptoms. Left scoliosis, resulting from long-term use of lateral crutches, was also detected in 2 patients through chest radiography. We believe that lateral crutches may aggravate compression of the axillary artery and result in a worse injury, while bilateral crutches could distribute the burden of body weight and reduce axillary artery injury. More research is needed to prove this theory.

In summary, the choice of therapy for crutch-induced upper limb ischemia patients should be based on the patient's specific situation. For severe crutch-induced upper limb ischemia, surgical operation is imperative. For those patients without severe upper limb ischemia, conservative treatment (long-term anti-coagulant or anti-platelet therapy) can significantly improve the ischemic symptoms, while not increasing the risk of amputation. In addition, it is worth noting that eliminating axillary pressure is the critical step in all the therapeutic strategies.

Disclosure of conflict of interest

None.

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