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RESEARCH PAPER

PATTERNS OF CHRONIC ISCHAEMIA OF THE UPPER LIMBS IN THE CENTRAL PROVINCE OF SRI LANKA

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Abstract

Introduction: A 30-year audit and management experience of chronic upper limb ischaemia in the Central Province of Sri Lanka is detailed. No previous Sri Lankan documentation on this topic exists.

Materials and Methods: Data collected prospectively from 1974 to 2004 using a standard protocol was analysed.

Results: Patterns of chronic ischaemic disorders seen dominantly among the 290 patients included in the study were, Aortic Arch Syndrome caused by both atherosclerosis (n=11) and Takayasu's aorto – arteritis (n=7), Thoracic Outlet Syndrome (n=6), occlusion of major arteries of supply to the upper limb due to atherosclerosis (n=29), thromboangitis obliterans (n=188), giant cell arteritis (n=1), chronic arterial trauma (n= 2), chronic embolism (n= 3), haematological causes (n=4), Ergot induced vaso spasm (n=1), and vasculitides (n=38).Their management is detailed.

Discussion and conclusions: Knowing the Sri Lankan patterns will help local clinicians to expedite it's differential diagnosis.

Key Words: ischaemia, upper limb

Introduction

Chronic upper limb ischaemia (CULI) is less prevalent than chronic lower limb ischaemia. The better collateral circulation, the smaller muscle bulk and the less sustained demand may partly account for this difference. The lower prevalence of atherosclerosis in the arteries of the upper limb is a singular feature that also dominates the difference. The collateral source by a reversal of flow in the vertebral arteries (subclavian steal syndrome) remains an additional but risky resource in very proximal critical stenoses or occlusion of the subclavian artery. The reduction in collateral reserve caused by congenital anomalies of the ulnar, palmer and digital arteries[1] are unmasked by the earlier onset of ischaemia caused by small artery disease, in these patients[2].

The upper limb vasculature seems also to be more sensitive to triggers such as cold and emotion[3]and is followed by a vasospasm induced by sympathetic over activity [3]. Exposure to cold aggravates many aetiological mechanisms. They are changes associated with viscosity, the activity of the vascular endothelium, and local neural reflexes[3].Smoking too aggravates digital ischaemia[3,4].

Ischaemia of the finger or hand results in an excruciatingly painful state that invariably entails tissue loss, and often mandates intervention.

No studies of the aetiological patterns of this group of disorders have been documented in Sri Lanka. International patterns have been described[5, 6,7] and we are documenting the differences observed in the local scenario of these patterns to help prioritisation in clinical decision making.

Materials and Methods

All patients with CULI, who presented to the Vascular Unit of the Teaching Hospital Peradeniya, during the 30 year period (1974 -2004) were prospectively audited on protocols. The data were computerised on spreadsheets and analysed. Data on the management of these patients were also documented during this period. Where operations were performed, records were kept and their results and follow up documented.

Ethical clearance to peruse these patient records was given by the relevant committee of the Faculty of Medicine, Peradeniya.

Results

Two hundred and ninety patients were included in the analysis.

Causes of Chronic Ischaemia of the Upper Limb

Majority (n=218, 75.2%) of the patients had underlying chronic occlusive arterial diseases (COAD) while 13.1% of the patients presented with CULI due to vasculitis. The aetiological breakdown is shown in Table 1. Figure 1 highlights the clinical distribution of lesions. The commonest presentation among the study population was loss of radial pulse (n=98, %). Table 2 shows the clinical presentation leading to a diagnosis of chronic ischaemia of upper limb (CULI) in this period.

Aortic Arch Syndrome (n=18)

Patients with Aortic Arch Syndrome (AAS) (n=18) consisted of two groups based on their aetiology. Group1. Atherosclerotic (ASO), AAS (n = 11) and group 2. Takayasu's Aorto-arteritis AAS (n= 7).

Group 1.Aortic arch syndrome atherosclerotic (ASO).Were considered to be of ASO origin, in 11 patients, because of the presence of ASO risk factors, hypertension, diabetes, smoking, evidence of systemic atherosclerotic syndromes, a normal ESR and compatible features on aortography (Figure: 2).

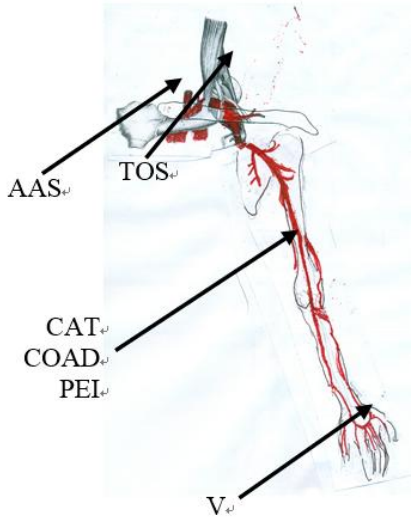

Figure 1: Common sources and sites of occlusion

Table 1: Diagnostic patterns of chronic upper limb ischaemia (N=290)

Diagnosis	n (%)
Chronic Occlusive Arterial Disease (COAD) (atherosclerotic – 29 thrombangitis obliterans – 188 Gaint cell arteritis – 1)	218 (75.2%)
Aortic Arch Syndrome (atherosclerotic – 11,) Takayasu's aorto – arteritis – 7)	18 (6.2%)
Vasculitides (V)	38 (13.1%)
Thoracic Outlet Syndrome (TOS)	06 (2.1%)
Haematological	04 (1.4%)
Post Embolic Ischaemia (PEI)	03 (1.0%)
Chronic arterial trauma (CAT)	02 (0.7%)
Ergot Induced Ischaemia (EII)	01 (0.3%)
Hypothenar-Hammer-Syndrome (HHS)	00
Cold induced/ cold immunoglobulins	00
Total	290

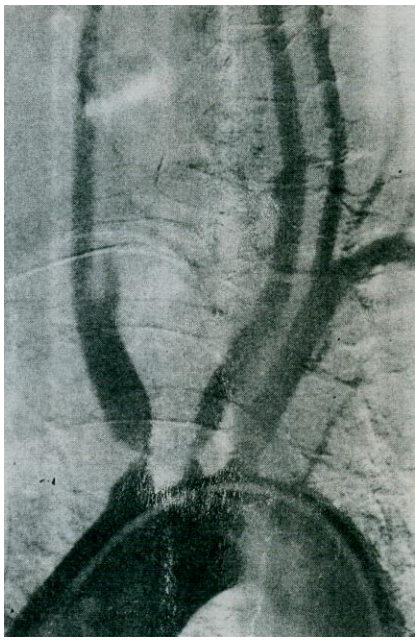

Figure 2: Atherosclerotic AAS

All eleven patients were referred to the clinic because of diminished or absent brachial pulse and all patients showed a stenosis or occlusion of the proximal subclavian artery on arch aortography. On angiography, 6 patients had lesions on left subclavian artery, 5 on right subclavian artery, 4 on innominate artery and 2 each on right and left carotid arteries.

Table 3, highlights their clinical details. They were older (mean 47.6 yrs) with a male dominance, presented 1-7 years after symptoms were apparent. A high incidence of neurological symptoms (n = 6) and of associated systemic ASO disease was noted. Clinical features associated with ischaemia were noted in the areas supplied by the cerebral (n=6), aorto-iliac (n=5), cardiac (n=3), renal (n=2), femoro-popliteal (n=2) and tibial (n=1) arteries.

The core blood pressure could not be properly assessed in all patients, but in those when it could be, 4 patients were hypertensive (ie > 160/90 mm of Hg). Two patients were diabetic and 6 of the 8 males were smokers. A non smoking male, was a diabetic. Eight others had other systemic systems involved, 2 patients with congestive cardiac failure and another 2 with chronic renal failure. They were not suitable candidates for major surgical reconstruction. Only patients 1, 5 and 11 (see Table 3) had clinical features of CULI, others were incidentally detected during clinical examination**.** and no attempt at reconstruction was made.

Table 2: Clinical patterns of presentation of chronic upper limb ischaemia

Presenting clinical feature	N (%)
Only Pulse loss (Brachial)	49 (16.9%)
Only Pulse loss (Radial)	98 (33.8%)
Pulse loss + Forearm Claudication	2 (0.7%)
Pulp atrophy + Spindling of finger tip	50 (17.2%)
Secondary Raynaud's Phenomenon	20 (6.9%)
Ulceration ± Pulse loss	6 (2.1%)
Gangrene ± Pulse loss	18 (6.2%)
Ex digital Amputation – Healed	44 (15.2%)
Ex digital Amputation – Not healed	15 (5.2%)
Forearm Amputation	2 (0.7%)

Table 3: Presentation of patients with atherosclerotic aortic arch syndrome (n=11)

Patient	Sex	Age of onset	Clinical features (duration)	Duration	Other Features
1	F	38	Left finger ischaemia	NA	CCF and diabetes
2	M	41	Aphasia, upper motor neuronal facial weakness, transient ischeamic attacks, amblyopia	1 yr	-
3	M	41	Faintness, dizziness and headache	NA	Chronic renal failure
4	M	41	Bilateral thigh claudication	NA	Hypertensive
5	M	44	Bilateral finger ischaemia	7 yrs.	Hypertensive, Congestive cardiac failure
6	F	46	Dizziness, subclavian steal syndrome:	3 yrs.	Aortic calcification
7	M	48	Dizziness, blurring of vision, fainting, right hemiparesis	1 yr	-
8	M	55	-	NA	Chronic renal failure
9	M	54	Transient ischaemic attack – visual	NA	
10	M	NA	Left hemiparesis, right hemiparesis	4 yrs	Hypertensive
11	F	68	Left finger ischaemic	4 yrs	Rheumatoid arthritis, diabetes

NA – Not available

2 Group, Takayasu's Aorto- arteritis

AAS was diagnosed based on the age (mean 23.43 yrs), an elevated erythrocyte sedimentation rate (ESR), angiographic features, (**Figure:3).** negative, Rheumatoid factor, Anti-Nuclear Factor (ANF), and, the absence of evidence for ASO systemic involvement and risk factors. Histological features detected on biopsies taken at surgical interventions confirmed the diagnosis in two patients.

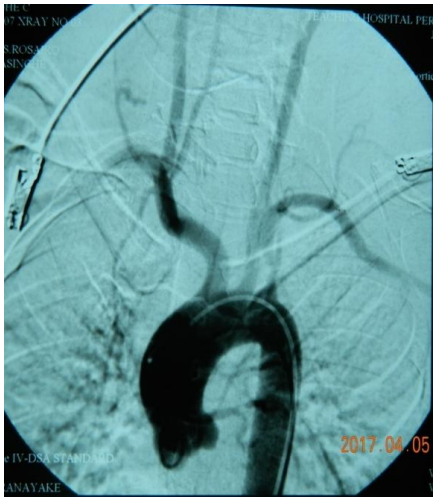

Figure 3: Aortogramme features of Takayasu's Aorto- arteritis

This group consisted of 7 patients 4 of whom were males with a mean age of 23.43 yrs, Table 4 highlights the clinical details of this group, 2 patients presented with neurological symptoms. Duration of symptoms ranged from 5 months to 8 years. Six patients had upper limb ischaemia. On angiography, lesions were detected in left subclavian (n=5), right subclavian (n=3), innominate(n=2) and right carotid (n=1) arteries while no lesions were detected on the left carotid arteries (this data was computed from data, obtained from clinical data and from arch aortography as **only four** of the **seven** had successful arch aortography).

None of them were hypertensive (ie < 160/90 mmHg) and none suffered from diabetes mellitus. All 4 males smoked. Associated ischaemia of the other vascular beds such as the heart and the kidneys were not present. However 3 patients had associated aortoiliac and another tibial occlusions. The ESR was elevated in all. The Rheumatoid Factor, ANF, and Lupus Erythematosus (LE) Cells were negative. Only two patients warranted subclavian to

Table 4: Presentation of patients with aortic arch syndrome due to Takayasu's Aorto-arteritis (n=7)

Initials of patients	Sex	Age of onset	Clinical features	Duration (months)
1	F	15	Right forearm and hand claudication	24
2	M	19	Right hemiparesis	48
3	F	27	Bilateral forearm claudication and subclavian steal syndrome	64.
4	M	24	Left forearm wasting	5.
5	M	23	Left forearm wasting and claudication, and fingertip ulceration	6
6	M	30	Left finger ulceration	-
7	F	26	-	-

subclavian prosthetic bypass and were completely relieved of their symptoms. Another patient managed conservatively, was followed up for 15 years and eventually regained her absent radial pulses and was completely asymptomatic by the age of 30 years.

Thoracic outlet Syndrome n=15

Thoracic outlet syndrome due to cervical rib (Gruber 1-1V) caused 15 patients[16] to have their subclavian artery compressed (Figure 5). leading to post stenotic dilatation of the subclavian artery (n = 7), and aneurysm formation (n= 2) (Figure 4) , leading to chronic embolism of thrombus from an aneurysm and ischaemia of the distal upper limb (n = 6)(Gruber III).

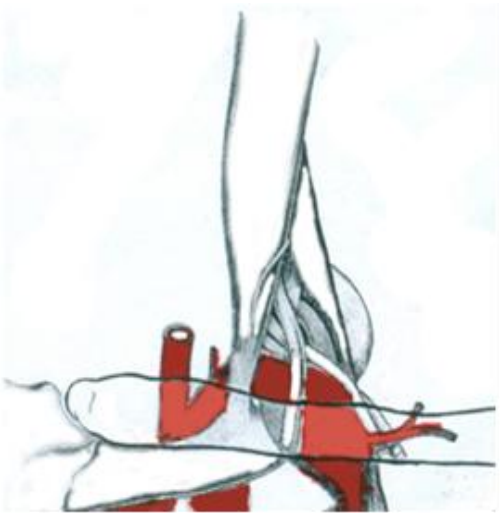

Figure 4: Right Cervical rib with subclavian artery compressed with post stenotic aneurism

Chronic Arterial Trauma n=2

Case 1 A 29-year-old male sustained a cut by a glass pane of his right brachial vessels. The cut injury included the entire neurovascular bundle. The artery was unsuccessfully reconstructed at a local hospital. The patient presented one month later with a Volkmann ischaemic contracture and the contractures of the muscles of the hand. He had gangrene of the tips of his three radial fingers. The artery was reconstructed with an interposition vein graft and the median and ulnar nerves repaired. The limb, his dominant, though vascularised was irretrievably damaged and the final functional result gained was poor.

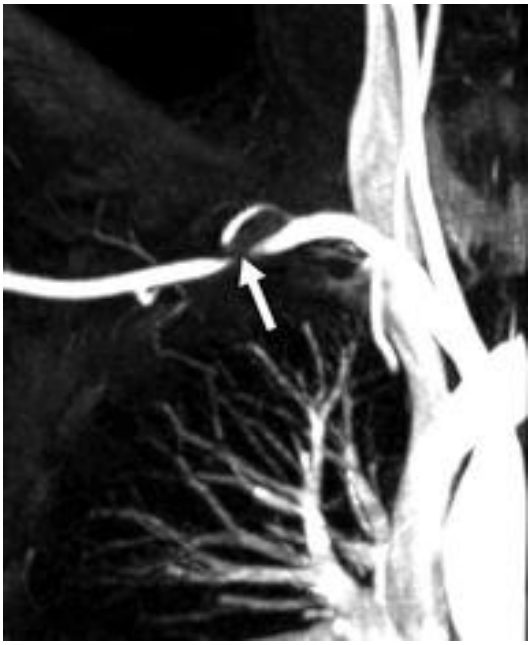

Figure 5: Digital subtraction angiogram showing stenosis caused by Left cervical rib

Case 2.A 26-year-old male had his left elbow region wounded by a shot gun injury. He was managed in the local hospital conservatively despite the coldness of his hand. He presented 37 days later with an arterio-venous fistula arising due to a false aneurysm rupturing into a vein. This was infected and there was diminished movement of the ipsilateral fingers. The fistula was taken down and reconstructed and the brachial and radial pulses returned. Residual stiffness of the fingers remained despite physiotherapy.

Chronic Occlusive Arterial Disease of the Upper Limb (n=218)

Chronic Occlusive arterial disease of the upper limb (COADUL) causing ischaemia, was seen in 21**8** patients. Hundred and ninety three (88.9%) of the patients with COADUL developed symptoms below the

age of 49 years, while 96 developed symptoms before 35 years (Table 5).

Males dominated, (M/F:20**7**/11).The distribution of females in the age groups in years <35/35-49/>50 were 4**;**4;3 respectively. Smoking either beedi or cigarettes and less commonly cigars, was indulged by all but one male. Females did not smoke. The percentage heavy smokers (>20/ day) was found in 62/61/62 in the age groups <35, /35-49/>49 respectively. Mild smoking (<10/day) was very infrequent.
The percentage prevalence of systemic involvement, ie ischaemic heart disease (IHD), and strokes(CVA) were in the age groups in years, <35/35-49/>49; 1%/10%/16% for IHD and 0%/7%/12% for CVAs respectively.

The percentage prevalence of primary risk factors for atherosclerosis obliterans, diabetes, and hypertension other than smoking and hyperlipdaemia in the age groups in years <35, /35-49/, >49 were for diabetes 0%/2%/8% and for hypertension 2%/11%/ 12% respectively.

Forty three (43/193, 22%) of the patients below the age of 49 years had superficial thrombophlebitis. and only 1 patient of the 25 patients over 50 years had thrombophlebitis.
Further biopsy findings of specimens of digits (n= 43), and dissected distal forearm arteries of amputated limbs showed a thrombus within a artery which on light microscopy did not show any changes of ASO.

Level of pulse loss and the duration of ischaemia at presentation, is highlighted in Table 6. This does not reveal an overt proximal ascent with time.

Angiographic data on a few patients did not reveal any significant run off. Most patients were subject to short courses of vasodilator drugs namely nefidepine and guanethidine, failing which the patients were mostly relieved of their rest pain by cervical sympathectomies and amputation of their gangrenous digits.

Post Embolic Ischaemia (n=3)
Six patients presented secondary to embolism. In 3 patients with thoracic outlet syndrome, embolism followed post stenotic aneurysmal dilatation as has been previously described.[16]. Three other patients presented late with what seemed clinical features of embolism.

Case 1: A 47-years-old female had developed an acute on chronic onset cold left forearm and hand at the beginning of this study. The left hand was cold and blue. The pulses were only felt up to the axillary artery. No lesion was detected in the heart, aortic arch or thoracic outlet though an axillary artery embolism was suspected. The limb was managed conservatively as was the practice then, recovered but developed mild forearm claudication on effort.

Case 2: A 67-year-old male with angina developed an acute coldness leading to gangrenous ulceration of the right little finger tip one month previously which he then self traumatised. No pulse loss was detected. A primary site as a source could not be detected. Athero-embolism from an aortic source was suspected .A cervical sympathectomy was effective in relief.

Case 3: A 55-year-old female was admitted with burning pain in both hands and Raynaud phenomenon of 3 month duration. She had a history of a thyroidectomy for thyrotoxicosis at 19, on admission both radial pulses were felt but were irregularly irregular, serum thyroxine was 14.1ug/dl and recurrent thyrotoxicosis with atrial fibrillation was diagnosed. The Raynauds of both hands was either unrelated or post embolic from a cardiac source. She was placed on carbimazole and an oral anti coagulant.

Table 5: Chronic occlusive arterial diseases of the upper limb

	Age group (AO)		
	<35 years	35-49 years	>49 years
Occlusive disease			
Thrombobangitis obliterans (n=188)	94	85	9
Atherosclerosis obliterans (n=29)	2	11	16
Giant cell arteritis (n=1)	0	1	0
Demographic features			
Males	92	94	22
Females	4	3	3
Risk factors			
Smoking	92	94	21
Hypertension	2	11	3
Diabetes	0	2	2
Systemic involvement			
Ischaemic heart diseases	0	7	3
Cerebro vascular accidents	1	7	4

Table 6: Proximal most pulse occlusion in patients with chronic occlusive arterial diseases

Duration in years at presentation	Brachial occlusion N=55 (n,%)	Radial occlusion N=123 (n,%)	Digital occlusion N=39 (n,%)
0-5	27 (49.1%)	69 (56.1%)	20 (51.3%)
>5-10	10 (18.2%)	25 (20.3%)	9 (23.1%)
>10-15	9 (16.4%)	20 (16.3%)	5 (12.2%)
>15	9 (16.4%)	9 (7.3%)	5 (12.2%)

*Level of pulse in the case of Giant Cell Arteritis is unavailable.

Table 7: Patterns of vasculitis diagnosed in the study period (N=38)

Type of vasculitis	**n (%)**
Scleroderma	4 (10.5%)
Thrombo angiitis Oblitrrans	4 (10.5%)
Secondary Raynauds with Rheumatoid Disease.	3 (7.9%)
Mixed Connective Tissue Disorder	2 (5.3%)
Arteriosclerosis	1 (2.6%)
Systemic Lupus Erytematosis	1 (2.6%)
Poly arteritis nodosa	1 (2.6%)
Myeloma	1 (2.6%)
Carcinoma of the colon	1 (2.6%)
Atherosclerosis	1 (2.6%)
Vascultis , undiagnosed	19 (50%)

Drug induced Ischaemia n=1 (Ergot induced Vasospasm)

Case report: A-26-year old female professional, presented with a cold right upper limb. The coldness affected the digits, palm and forearm, 2 digits were cyanosed. The radial pulse was not felt and the brachial pulse was weak. The capillary return on the nail bed was slow. There was no wasting of the forearm muscles but tenderness of the forearm tissues and pain on stretching her right fingers was present. She had discomfort whilst using the right upper limb and the entire episode was less than a week's duration. She did not have rest pain. The other limbs were normal. Angiography showed the right arm and forearm vessels were of very narrow but of uniform calibre.
She has been on ergot tablets for migraine for approximately three months. On stopping the ergot tablets she made a complete recovery in 5 days without intervention. The dose of ergot had unfortunately not been recorded.

Vasculitis (n= 38)
Many patients presented with Primary Raynaud's Disease, but were not included. They were mostly young women with painless discolouration of fingers .classically showing sequential pallor, cyanosis and crimson colour on reperfusion as time elapsed (Raynauds phenomenon) (Table 7).

Scleroderma was diagnosed on the basis of patients who had a previous medical diagnosis based on microstomia, dysphagia, high ESR and on cutaneous biopsy.
Those with secondary Raynauds had painful finger/s with Raynauds phenomenon, ie similar colour changes. Common presentations often included a spindled (due to pulp atrophy) finger or fingers of one or both hands. Minor ulceration, coldness, rest pain, pre gangrene and occasionally gangrene were features. 3 of these patients had a positive rheumatoid factor on serology.

Two patients with malignancy came with secondary Raynaud's of a single digit and were thought to result from a gammopathy causing elevated viscosity. . The Raynaud's phenomenon responded to the resection of the colonic cancer and the management of myeloma.

Management included a careful study of the protean causes of vasculitis and treatment of the disease. Procedures to improve the vascularity included cervical sympathectomy often done via the neck, if a local anaesthetic to the stellate ganglion was helpful to relieve the pain.

No patients were seen with Hypothenar Hammer Syndrome or cold induced immunoglobulins.

Discussion

The striking prevalence of CULI in Sri Lanka compared to most western international literature,[5,6,7] is due to the disorder Thromboangiitis Obliterans (TAO). . Collagen disorders and those caused by trauma ie vibratory white finger and hypothenar hammer syndrome are significantly uncommon in comparison to the west[5,6,7.]. The warm climate could be argued to be a factor.[3]The other causes of transient digital ischaemia such as Primary Raynauds disease, were not included in our study.

Recent studies[8] have shown some claudication handicaps if the traumatised vessel is simply tied off and every encouragement is given to reconstruct the artery. In the first case above with chronic trauma of a mainline artery, technically poor reconstruction and delay should have been avoided. Chronic arterial injury with the development of an arterio-venous fistula ,highlighted in case 2., illustrates the distal ischaemia that can result from shunting caused by fistulation as occurs sometimes, even in iatrogenic constructions for vascular access[9.]

Likewise upper limb embolism, formerly managed conservatively[10] is now thought to need a more active approach to avoid ischaemic symptoms like claudication, loss of delicacy of hand movements , and the loss of sensitivity of the digits especially if the dominant limb is involved [11].

The centrifugal force and the sheer effect on the aortic arch are thought to be responsible for the development of a ASO plaque at that site with spill over ASO into the arteries arising from the arch. The stenoses, the occlusions, the emboli arising from unstable plaques lead to cerebral ischaemia or to upper limb arterial compromise. It is seen in the cases we highlighted in this study. The forty year age group involved in our study is about a decade or two younger than western series[12] This leads to a possibility, as we have no histological data, to the possibility of us having included some patents with Takayasu's aorto arteritis into this group though the ESR was normal.. Appearances on aortography (n=11) showed all 11 to have subclavian stenoses or occlusion but only three presented with ischaemic features in their ipsilateral upper limbs, on presentation. They were referred to the vascular clinic with loss of brachial pulse and for their neurological features accompanying the carotid pulse loss or the presence of a carotid bruit. Our study also reflected the systemic nature of the ASO in these cases as has been shown to be common in the West.[12] Further, subclavian steal syndrome[13] was seen in two of our patients.

Those with upper limb ischaemia with aortoarteritis were probably selected for referral to the vascular clinic. A slightly greater prevalence of males in those presenting with CULI were seen compared to other studies.[14,15,16]. The natural recovery of a young patient (1, Table 4), is noteworthy.

A detailed study of cervical rib compression and its complications have been made earlier by the first author.[17] Management by early embolectomy would have been the way to avoid ischaemia in this group. [16] Reconstructions are risky once the distal arterial run off becomes filled with episodic bouts of embolisation.
Drug induced ischaemia, a classical case is described. Similar experiences, has been documented [18].

Vasculitides presenting with digital ischaemia posed a major problem especially in the dominant limb. It was usually painful and tissue loss was either present or was imminent. We probably saw most cases admitted in the Central Province. 38 cases over 20 years does not amount for much and highlights the relatively low prevalence at present .compared to the West [19]. The significant absence of Hypothenar hammer syndrome is a feature.

Among the patients with occlusive arterial diseases, 43.1% of the patients were below the age of 35, dominantly male, heavy smokers with a significant prevalence of thrombophlebitis with a no prevalence of diabetes and a low prevalence of hypertension and systemic occlusive arterial disease of the heart and brain. These patients under 35 years were afflicted almost exclusively by Thrombo angiitis obliterans (TAO).This was true of a significant proportion in those afflicted in the under 49 age group as well.

In conclusion the dominant disorder causing CULI in our study was due to the prevalence of TAO. Encouraging the patients to stop smoking and the use of cervical sympathectomy and digital amputations were the mainstays in our management. This data we hope will provide a scaffolding for clinicians to make a diagnosis.

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