Free-Floating Thrombus of the Carotid Artery Detected on Carotid Ultrasound in Patients with Cerebral Infarcts: A 10-Year Study

Dear Editor,

A free-floating carotid artery thrombus is rarely found in cerebral infarcts. Current understanding is limited with small case series.[1-5] The objective of this study is to characterise the clinical profile, treatment and outcome of this uncommon condition.

Materials and Methods

All patients admitted from 1999 to 2009 for cerebral infarcts were included. IRB approval 2009/867/A was obtained. Cases were retrospectively identified from carotid ultrasound reports showing free-floating thrombus. The latter is defined as "an elongated thrombus attached to the arterial wall with circumferential blood flow at its distal most aspect with cyclical motion relating to cardiac cycles."[1-3] Case notes and computerised medical records were reviewed.

Results

A total of 26,818 carotid ultrasound reports were screened and 25 patients were identified (mean age 59 ± 12.3 years; 18 were men). Risk factors: hyperlipidaemia (96%), hypertension (64%), ischaemic heart disease/atrial fibrillation (44%), diabetes (40%) and smoking (24%). The patients were divided into 3 groups based on clinical presentation: group 1 (transient ischaemic attack), group 2 (lacunar infarct) and group 3 (hemispheric syndrome). Thrombus aetiology: cardioembolism/atrial fibrillation (44%), carotid dissection (8%), 80% to 99% carotid stenosis (8%), history of malignancy (8%), antithrombin III deficiency (4%). Treatment: anticoagulation (76%), antiplatelets (24%). Thrombus resolution occurred in up to 58% of anticoagulated patients. Duration of anticoagulation: median 112 days (range, 10 to 180 days; mean 93 days). All patients treated with antiplatelets developed carotid occlusion. Long-term follow-up: recurrent stroke occurred in 2 patients and ischaemic heart disease in 6 patients.

Discussion

This rare condition was seen in 25 patients in our institution over a 10-year period with an incidence of 0.9% (25/26,818). The incidence from other studies using carotid ultrasound for diagnosis of thrombus ranged from 0.05% (1/2000)[1] to 0.9% (14/1528).[2]

The close association with atherosclerosis was evident by the high prevalence of risk factors, male preponderance and carotid plaques. All patients (except 1) had one or more risk factors. Severe carotid stenosis (80% to 99%) was found in 2 patients (2 and 11). In contrast, Buchan et al[4] found moderate carotid stenosis (50% to 90%) in 5 patients (17%), and severe stenosis (>90%) in 21 patients (70%). This reflects a higher incidence of extracranial carotid disease in Caucasians compared to Asians. Two patients (5 and 20) had carotid dissection.[6] Both did not give a history of trauma. Magnetic resonance imaging (MRI) brain for patient 5 showed an embolic distribution, suggesting the pathophysiology was artery-to-artery embolism. For patient 20, the watershed pattern of infarcts suggests that the mechanism was haemodynamic insufficiency. The association of malignancy with thrombus formation is well-known.[5,7] Patients 2 and 23 had a history of malignancy but no tumour recurrence at the time of stroke. Among patients without risk factors, there was a young man with low antithrombin III. This rare condition predisposes to thrombosis in patients with additional risk factors.[8]

Complete resolution of thrombus occurred in 58% of patients treated with anticoagulation. This compared with a thrombus resolution rate of 86% amongst anticoagulated patients in a systematic by Bhatti et al.[3] This highlights the need for vigilant international normalised ratio (INR) monitoring.[3-5,7,9] Surgical embolectomy[1-5,7] and new intervention techniques[10] should be considered when patients cannot be adequately anticoagulated or when they develop complications of anticoagulation.

This study is limited by a lack of angiographic or histological correlation as all patients received medical therapy and none were surgically treated. In our study, 8% (n = 2) had recurrent stroke and 24% (n = 6) developed ischaemic heart disease. However, it is difficult to interpret the relationship between carotid artery occlusion and subsequent strokes as the time interval between strokes could be quite long and the patients also had risk factors which predisposed them to stroke. With regard to ischaemic heart disease, the time interval is shorter (4 months to 6 years), suggesting an increased risk of cardiovascular events. This is not surprising as both stroke and heart disease are different

Table 1. Classification into 3 Groups According to Clinical Presentation

Case	Age/Sex/Race	Risk Factors	Concurrent Illness	Stroke Syndrome	CT/MRI Brain	Thrombus Site (Stenosis /Dissection)	Reason(s) Not Anticoagulated /Problems with Anticoagulation	Treatment (Duration)	Follow-up Ultrasound (Result)	Follow-up (Adverse Events, if Any)
Group 1										
1	47/ M/Ch	Hypertension Hyperlipidaemia IHD	Nil	TIA	Normal	Left ICA	N.A.	Anticoagulation (4 months) followed by antiplatelets	4 months (clot resolved)	2 years
Group 2										
2	70/F/Ch	Hypertension Hyperlipidaemia Diabetes mellitus	Colon carcinoma operated 2 years ago, post-op chemo and radiotherapy	Left sensory stroke	Right corona radiata infarct	Right CCA (80% to 99%) RICA stenosis	Gross haematuria and abdominal wall haematomas	Anticoagulation (1 month) followed by antiplatelets	6 months (right ICA occluded)	5 years
3	52/M/Ch	Hyperlipidaemia	Nil	Left ataxic hemiparesis	Right MCA/ ACA water shed infarct	Right ICA	N.A.	Anticoagulation (4 months) followed by antiplatelets	4 months (clot resolved)	2 years
4	62/M/Ind	Hyperlipidaemia IHD CABG (EF 25%) Diabetes mellitus	Nil	Left pure motor stroke	Right corona radiata infarct	Right ICA	N.A.	Anticoagulation (2 months) followed by antiplatelets	2 months (clot resolved)	5 months
5	52/M/Ch	Hypertension Hyperlipidaemia Diabetes mellitus	Nil	Right sensorimotor stroke	Small left frontal parietal and occipital infarcts	Left Bifurcation (distal left CCA dissection)	N.A.	Anticoagulation (4 months) followed by antiplatelets	2 weeks (clot resolved)	1 year
6	66/M/Ch	Hypertension Hyperlipidaemia IHD Diabetes mellitus Smoker	Nil	Left sensorimotor stroke	Right MCA / ACA water shed infarct	Right ICA	Non-compliant	Anticoagulation (6 months) followed by antiplatelets	6 months (right ICA occluded)	9 years (acute myocardial infarct 7 months later)
7	60/M/Ind	Hypertension Hyperlipidaemia IHD Diabetes mellitus Smoker	Nil	Left brachial facial stroke	Right MCA water shed infarct	Right ICA	N.A.	Anticoagulation (6 months) followed by antiplatelets	9 months (right ICA occluded)	2 years (acute myocardial infarct 2 years later)

Table 1. Classification into 3 Groups According to Clinical Presentation (Con't)

Case	Age/Sex/ Race	Risk Factors	Concurrent Illness	Stroke Syndrome	CT/MRI Brain	Thrombus Site (Stenosis /Dissection)	Reason(s) Not Anticoagulated /Problems with Anticoagulation	Treatment (Duration)	Follow-up Ultrasound (Result)	Follow-up (Adverse Events, if Any)
Group 3										
8	45/M/Ch	Hyperlipidaemia Smoker	Nil	Right hemispheric stroke	Right MCA infarct	Right ICA	BGIT	Anticoagulation (2 weeks) followed by antiplatelets	7 days (clot resolved)	4 years
9	49/F/Ch	Hypertension Hyperlipidaemia	Patent foramen ovale with left to right shunt	Right hemispheric stroke	Right MCA / PCA water shed infarct	Right ICA	N.A.	Anticoagulation (3 years) for PFO followed by antiplatelets	3 days (clot resolved)	3 years
10	75/M/Ch	Hypertension Hyperlipidaemia AF	Nil	Left hemispheric stroke	Left MCA infarct	Left CCA	N.A.	Anticoagulation (lifelong)	3 weeks (clot resolved)	5 years
11	76/F/Ch	Hyperlipidaemia IHD Diabetes mellitus	Nil	Right hemispheric stroke	Right MCA infarct	Right CCA (80% to 99% right ICA stenosis)	N.A.	Anticoagulation (4 months) followed by antiplatelets	4 months (clot resolved)	3 years
12	52/M/Ch	Hyperlipidaemia	Nil	Left hemispheric stroke	Left MCA water shed infarct	Left ICA	Hemoptysis and thigh haematoma	Anticoagulation (10 days) followed by antiplatelet	3 ½ months (clot resolved)	4 years
13	76/M/Ch	Hypertension Hyperlipidaemia Smoker	Nil	Right hemispheric stroke	Right MCA / PCA water shed infarct	Right ICA	N.A.	Anticoagulation (4 months) followed by antiplatelets	4 months (clot resolved)	1 year
14	68/M/Ch	Hypertension Hyperlipidaemia Diabetes mellitus AF	Nil	Left hemispheric stroke	Left MCA infarct	Left CCA	N.A.	Anticoagulation (13 days) followed by antiplatelets	4 days (clot resolved)	13 days (died from sepsis)
15	69/F/Ch	Hypertension Hyperlipidaemia	Nil	Left hemispheric stroke	Left MCA infarct	Left ICA	N.A.	Anticoagulation (3½ months) followed by antiplatelets	3 months (left ICA occluded)	8 years (right corona radiata infarct 8 years later)
16	56/M/Ch	Hypertension Hyperlipidaemia IHD Smoker	Nil	Right hemispheric stroke	Right MCA and left MCA and PCA infarcts	Right ICA	Pangastritis	Antiplatelets	7 years (right ICA occluded)	7 years (left fronto parietal infarct 7 years later)

Table 1. Classification into 3 Groups According to Clinical Presentation (Con't)

Case	Age/Sex/Race	Risk Factors	Concurrent Illness	Stroke Syndrome	CT/MRI Brain	Thrombus Site (Stenosis /Dissection)	Reason(s) Not Anticoagulated /Problems with Anticoagulation	Treatment (Duration)	Follow-up Ultrasound (Result)	Follow-up (adverse events, if Any)
17	34/M/Ch	Nil	AT III deficiency AVM surgery 10 yrs ago, scar epilepsy	Left hemispheric stroke	Large left MCA infarct, right frontal encephalomalacia	Left ICA	Large infarct with mass effect and mildline shift, poor premorbid status	Antiplatelets	1 week (clot persists)	2 years
18	45/F/Ch	Hypertension Hyperlipidaemia Diabetes mellitus	End stage renal failure, fibroids	Right hemispheric stroke	Right PCA / MCA water shed infarct	Right ICA	Menorrhagia Hb 5.1 g/dL	Antiplatelets	1 week (clot persists)	7 years (recurrent myocardial infarct at 3 years and 7 years post-stroke. Died)
19	71/F/Ch	Hypertension Hyperlipidaemia IHD AF	Nil	Left hemispheric stroke	Left MCA water shed infarct	Left ICA	N.A.	Anticoagulation (lifelong)	4 days (clot persists)	7 years (acute myocardial infarct 6 years later)
20	47/M/Ch	Hypertension Hyperlipidaemia	Nil	Left hemispheric stroke	Left ACA / MCA water shed infarct	Left ICA (left ICA dissection)	Non-compliant	Anticoagulation (6 months) followed by antiplatelets	7 months (left ICA occluded)	2 years (ischaemic heart disease 1 year later)
21	83/F/Ch	Hypertension Hyperlipidaemia IHD Diabetes mellitus	End stage renal failure	Right hemispheric stroke	Right MCA	Right ICA	Large right MCA infarct with mass effect and BGIT	Antiplatelets	5 days (clot persists)	4 months (died of acute myocardial infarct 4 months later)
22	57/M/Ch	Hyperlipidaemia Smoker	Nil	Right hemispheric stroke	Right MCA	Right bifurcation	Right MCA infarct with haemorrhagic conversion	Antiplatelets	3½ months (right ICA occluded)	2 weeks (defaulted followup after discharge)
23	56/M/Ch	Hypertension Hyperlipidaemia Diabetes mellitus	Nasopharyngeal carcinoma 12 years ago, treated with radiotherapy	Right hemispheric stroke	Right MCA watershed infarct	Right CCA	N.A.	Anticoagulation (3 months) followed by antiplatelets	3 months (right ICA occluded)	1 year 8 months
24	79/M/Ch	Hyperlipidaemia IHD CABG AF	Renal impairment, aortic valve replacement	Right hemispheric stroke	Right striato capsular infarct	Right ICA	Post-TPA haemorrhagic infarct	Antiplatelets	3 months (right ICA occluded)	6 months
25	52/M/Ch	Hyperlipidaemia	Nil	Right hemispheric stroke	Right MCA watershed infarct	Right ICA	Non-compliant	Anticoagulation (1 month) followed by antiplatelets	4 days (clot persists)	1 year

Ch: Chinese; Ind: Indian; IHD: Ischaemic Heart Disease; AF: Atrial Fibrillation; CABG: Coronary Artery Bypass Graft; EF: Ejection Fraction; ICA: Internal Carotid Artery; CCA: Common Carotid Artery; TIA: Transient Ischaemic Attack; ACA: Anterior Cerebral Artery; MCA: Middle Cerebral Artery; PCA: Posterior Cerebral Artery; PFO: Patent Foramen Ovale; AT III: Antithrombin III; AVM: Arteriovenous Malformation; BGIT: Bleeding Gastrointestinal tract; N.A.: Not Applicable

presentations of the spectrum of atherosclerosis. The rarity of this condition makes a prospective study difficult. Recurrent events underline the importance of secondary prevention to reduce atherothrombotic disease burden.

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