CLINICORADIOLOGICAL CHARACTERISTICS AND OUTCOME IN PATIENTS WITH CERVICOCRANIAL ARTERY DISSECTIONS(CCAD)

PROJECT REPORT

Dr.Sreekumar.J

RESIDENT IN NEUROLOGY 2001-2003

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES & TECHNOLOGY
THIRUVANANTHAPURAM 695011.

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES & TECHNOLOGY
THIRUVANANTHAPURAM 695011.

PROJECT REPORT

TITLE OF THE PROJECT: **CLINICORADIOLOGICAL CHARACTERISTICS AND OUTCOME IN PATIENTS WITH SPONTANEOUS CERVICO CRANIAL ARTERY DISSECTIONS(CCAD)**

NAME Dr.Sreekumar.J

PROGRAMME DM,Neurology

MONTH & YEAR OF SUBMISSION November,2003

CERTIFICATE

I , Dr. Sreekumar. J, hereby declare that I have actually carried out the project titled Clinicoradiological Characteristics and Outcome in patients with Spontaneous Cervicocranial Artery Dissections (CCAD)

Signature

Name: DR.SREEKUMAR.J

Thiruvananthapuram
Date: 17/11/2003

Forwarded.

He has carried out the above mentioned Project in the Department of neurology, SCTIMST, Thiruvananthapuram.

Signature

Prof.K.Radhakrishnan
Head of the Department
Department of Neurology
SCTIMST, Thiruvananthapuram.

Acknowledgement

At the outset ,I express my heartful gratitude towards Dr .Sylaja P N, Assistant Professor of Neurology, SCTIMST for her constant review, proper guidance and keen interest and enthusiasm in the ongoing work.

I would like to thank Dr.KesavadasP, Assistant Professor of Neuroradiology for his valuable suggestions and patience in reviewing the images of all the patients.

I would also like to thank Dr.Gupta A K,HOD, Neuroradiology, for his kind co-operation for the study .

I would also like to thank Prof.Dr.K.Radhakrishnan ,HOD,Neurology for his guidance and encouragement.

Last, but not the least, I am thankful to all the patients who participated in this study, without whom the study would have been impossible.

Contents

INTRODUCTION

Spontaneous internal carotid artery dissection (ICAD) was first described by Jentzer in 1954 But it was not until late 1970's, when Fisher et al and Mokri et al described dissections of carotid and vertebral arteries as dectected by modern diagnostic techniques, that dissections began to be routinely diagnosed before death.Community based studies in US and France showed annual incidence of spontaneous dissections ranged from 2.5-3 per 100,000 population . Eventhough spontaneous cervicocranial artery dissections (CCAD) account for only 1-2% of all ischemic strokes ,they are an increasingly recognized cause of stroke in young adults and account for 10-25% of such cases · resulting in significant morbidity in the productive age group.

CCAD often develops in otherwise healthy individuals without known risk factors for stroke and frequently develops spontaneously without relevant trauma and its pathogens is largely unknown.Occasionally mild mechanical stress as sudden head movement as in chiropractic manuevers, infection, coughing and sport activities has been described

Other comorbid risk factors described are FMD, EDS, Migraine, Hyperhomocystinemia etc. Intracranial dissections are rare compared to Extracranial dissections, which has a benign prognosis with early diagnosis and intervention. Recurrence rate in CCAD was relatively low at 1 % /year except within 1 month of initial dissection and is 6 times higher in patients with a family history of dissections in any vessels .

In contrast to the vast literature in the west devoted to clinicoradiological characteristics and outcome, very little Indian data are available on the same.Hence this study is designed to look into the clinicoradiological characteristics and outcome of patients with CCAD at a tertiary referral centre for neurology ,in south India. We also looked into comorbid risk factors.

AIMS AND OBJECTIVES

1.To analyze the clinical and angiographic characteristics of CCAD

2.To study the course and prognosis of patients with CCAD

3.To analyze the associated risk factors in patients with CCAD.

Review of literature

Regarding the incidence of cervico cranial artery dissections (CCAD) only a few publications are available in literature, just because it is an under diagnosed condition.It was not until late 1970's when Fisher et al and Mokri et al described dissection of carotid and vertebral arteries as detected by modern diagnostic approach, that dissections began to be routinely diagnosed before death.

In a study from Rochester, Minnesota, Schievnik et al reported annual incidence of internal carotid artery dissection(ICAD) to be 2.6 per 100,000 population ,making it an uncommon condition,but not rare.The annual incidence of vertebral artery dissections can be estimated at 1 per 100,000 to 1.5 per 100,000.But CCAD account for about 1-2% of all Ischemic strokes.These dissections are potentially serious and account for around 20% of stroke in young and 5% in middle & elderly patients.

Pathophysiology

In most cases no specific etiology can be found in dissections ie:they are spontaneous. Main pathophysiology that happens in CCAD is an intimal tear, which allows blood under arterial pressure to enter the wall of the artery or a primary intramural hemorrhage of vasa vasorum that ruptures in to the true lumen to form an intramural hematoma,the so called false lumen.

Depending on the plane of cleavage of dissection,it results in stenosis of arterial lumen or aneurysmal dilatation of the artery.There is subsequent extension of the dissection across varying distance along the length of the artery, typically in the direction of blood flow..The dissection may occur closer to the intima WHICH IS termed subintimal dissections or closer to the adventitia,designated as subadventitial dissections. When subadventitial dissection occurs ,it causes dilation of the artery and are often referred to as dissecting aneurysm or pseudoaneurysms(not true "pseudo" since their walls composed of blood vessel elements).When subintimal dissection occurs,the intimal lining is forced away from the media of the artery as circulating blood invades the area.This effectively narrows the arterial lumen.Occasionally a double lumen is formed when the subintimal hemorrhage ruptures in to arterial lumen distally resulting in double lumen.On follow p the dissected artery often recanalises and aneurysms that develop may show regression.

This changes in the vessel wall lead on to cerebral ischemic symptoms either by hemodynamic changes or distal embolisation from a dislodged fragment of thrombus.

Pathogenesis

Regarding the pathogenesis of dissections, both genetic and environmental factors play a role.Patients with spontaneous dissections of CCAD are thought to have an underlying structural defect of the arterial wall,although the exact type of arteriopathy remains elusive in most cases.Foremost among the heritable disorders associated with an increased risk of dissections of CCAD is Ehlers-Danlos syndrome type IV.Others include Marfan's syndrome,autosomal dominant polycystic kidney disease and osteogenesis imperfecta typeI. These well characterized heritable connective tissue disorders account for only 1-5 % of patients with spontaneous CCAD.

Angiographic changes of fibromuscular dysplasia are found in about 15 % of patients with CCAD and cystic medial necrosis is a common finding at postmortem examination.Ultrastructural abnormalities of dermal connective tissue componenets have been detected in 2/3 of patients with CCAD.Extensive studies however has failed to identify any mutations in the gene for type III procollagen(COL3A1),the gene for type V procollagen (COL5A1) or the gene for tropoelastin(ELN)&staining for antibody against fibrillin-1 has been negative for these patients with CCAD.

Tzourio et al has demonstrated strong association between migraine and cervical artery dissection and postulate an underlying arterial wall disease could be a predisposing condition for both.Other concurrent pathological conditions associated with CCAD are hypertension in 26-36% patients ,ICA redundancies,kinks,coils or loops in 30-62% in various series .

The environmental factors as a precipitating event is frequently elicited in patients with CCAD .Colourful terms are often given to such occurrences like "bottoms-up dissection" and "beuty-parlor stroke" etc.Beatty in 1977 first described the association of ICAD with chiropractic manipulation.Since then association between chiropractic manipulative therapy(CMT) and dissections,especially for VADdissection is available in literature.

CMT does not cause dissections in elderly patients because of the redundancy or coils of the vessels in this group result only in

straightening of the vessel rather than stretching and intimal tear.The possibility of an infectious trigger is supported by the finding of a seasonal variation in the incidence of spontaneous dissection of CCAD with a peak incidence in the fall.

Clinical manifestations

With the advent of noninvasive imaging,dissection is being diagnosed in many patients who present with subtle manifestations of the disease.Most of the patients have at least two symptoms,although symptoms may occur in isolation and some dissections remain asymptomatic.Localised warning signs are common and provide an opportunity to recognize and treat patients before cerebral ischemia occurs.

Typical patient with ICAD presents with pain on one side of face,head,or neck accompanied by a partial Horner's syndrome and followed hours or days later by cerebral or retinal ischemia.This classic triad is found in only less than 1/3-1/5 of patients,but the presence of any two elements in this triad should suggest the diagnosis.

Local manifestations

Pain develops on one side of the neck in ¼ of patients and usually confined to upper anterolateral cervical region.Persistent isolated neck pain may mimic idiopathic carotidynia, if it is associated with local tenederness.Unilateral facial or orbital pain present in half of the patients and ipsilateral headache in 2/3 of the patients.Onset of headache is usually gradual ,but it may resemble thunderclap headache.

The headache most commonly described as a constant steady aching,but it may also be throbbing or steady and sharp.Median time to appearance of other Ischemic symptoms is around four days.

Typical patient with VAD present with pain in the back of neck or head followed by ischemia of the posterior circulation. The initial manifestations of VAD however are less distinct than those of ICAD and are usually interpreted as musculoskeletal in nature.The neck pain may be bilateral. Median interval from the neck pain to the appearance of other symptoms is 2 weeks..Unilateral pain and weakness of an arm as result of cervical root involvement at C5-C6 level resembling neuralgic amyotrophy is reported in literature .Spinal epidural hematomas are unusual manifestations of VAD.

Ischemic manifestations

Cerebral or retinal ischemic symptoms are reported in 50 to 95 percent of patients with spontaneous ICAD,although this frequency has decreased over the years as the condition is diagnosed in more patients with less obvious manifestations.As in carotid stenosis resulting from carotid atherosclerosis ,TIA's and transient monocular blindness usually precedes the ischemic stroke in ICAD..Only about 1/5 of patients have an ischemic stroke without any warning signs.Permanent blindness as a result of ischemic optic neuropathy or occlusion of retinal artery is rare. Other rare manifestations include pulsatile tinnitus in one fourth patients,trigeminal,oculomotor,facial and lower cranial nerve palsy in around 10 % of patients,.Facial diplegia is seen in bilateral dissection.

Ischemic symptoms occur in more than 90% patients in whom VAD is diagnosed and may involve brainstem,particularly lateral medulla (Wallenberg's syndrome),as well as the thalamus and the cerebral or cerebellar hemispheres.Isolated ischemia of the cervical spinal cord is an uncommon but increasingly recognized complication of VAD.TIA's are less frequent after VAD than ICAD.

Diagnosis

Conventional angiography has long been the gold standard since it can show the arterial lumen and allows extensive characterization of dissections of carotid and vertebral arteries. Pathognomonic features such as intimal flap or a double lumen are detected in <10% of dissected arteries.Other typical angiographic findings are irregular and tapered stenosis with string sign ,which starts about 2-3 cm distal to carotid bulb and extend usually upto petrous segment.

Occlusion by dissection has got a tapered flame-like appearance. Aneurysmal dilation involve mainly subcranial segment. In vertebral artery dissection both stenotic and aneurysmal lelsions are most common at the level of C1/C2 vertebra. Vertebral artery entres the skull through foramen magnum, which explains the high incidence of intracranial extension even up to 10%, in VA dissections.The angiographic appearance of vertebral artery stenosis and particularly occlusion caused by dissection is less specific than that seen in carotid dissection.

Magnetic resonance angiographic technique(MRA) is replacing conventional angiography because of its resolution approaching that of conventional angiography and can show the intramural hematoma.Intramural hematoma has a crescent shape adjacent to the vessel lumen and often spirals around the length of the artery.

Fat suppression techniques are important to differentiate small intramural hematomas from surrounding tissues. MRA is superior to angiography in cases of dissections without luminal abnormalities or in cases resulting in nonspecific occlusion. Auer et al has studied the usefulness MRA in VAD and showed that MRI improves the triage for selective angiography and discloses complementary information for the diagnosis of VAD and also helps in omission of invasive procedures. They report a sensitivity of 94 % and specificity of 30 % compared to 100 % sensitivity of angiography.

Ultrasonographic techniques are useful in the initial assessment of patients,who are thought to have dissection of carotid artery.Abnormal pattern of flow is identified in more than 90% patients. Specific abnormalities of dissection such as intimal flap or intramural Haematoma are found in less than 1/3 of patients.

Transcranial ultrasonography is valuable in following the heamodynamic changes in ICAD.Dissection of vertebral and carotid arteries are dynamic processes. Hence radiographic findings change dramatically within a period of days or even hours.Although the radiographic appearance may worsen during the acute phase of dissection, about 90% of stenosis eventually resolve and 2/3 occlusion recanalised and 1/3 of aneurysms decrease in size. Improvement takes place largely within the first 2-3 months after dissections and is rare after 6 months. In rare cases persistent extracranial aneurysms may cause thromboembolic complications years after dissection. Risk of recurrent dissection in healthy, unaffected artery is about 2% during the 1st month and after that 1% per year.

Treatment

As regards the treatment there have not so far been any randomized controlled trials on which recommendations can be based,although antithrombotic treatment has been advocated since the 1970's.To prevent thromboembolic complications anticoagulation is recommended in various studies regardless of the type of symptoms,unless there are contraindications such as intracranial extension. Main argument for the same is to prevent cerebral embolisation from dissected artery and to prevent permanent occlusion in tight stenosis. Sreenivasan et al has shown microemboli signals in middle cerebral artery in patients with ICAD ,by using transcranial doppler study, giving impetus to the role of anticoagulant therapy,and also helps to resolve the dilemma regarding the exact duration of anticoagulant treatment. Lucas et al has shown contribution of heamodynamic changes as a minor cause of cerebral ischemia. Hemodynamic changes are a more important cause of ischemia in intracranial dissections.

Consensus regarding anticoagulation from various studies are ,anticoagulation with a target international normalized ratio of 2-3 is used for 3-6 months.Optimal duration of anticoagulation has not been established . Anticoagulation may be stopped earlier if complete recanalisation is demonstrated on duplex imaging or MRA on follow up. And may be continued in cases of persistent aneurysm or remaining luminal irregularities of the vessel wall. Despite anticoagulation recurrence of symptoms can occur in 7-14% of patients.Endovascular stenting or placement of coils is preferred in cases of persistent aneurysm, which acts as a source of emboli or if patient develops recurrent symptoms despite optimal anticoagulation. Surgery in the acute setting of dissection is least preferred,because of friability of vessel wall.Surgical excision of aneurysms also tried in various small series of patients with VAD.

PATIENTS AND METHODS

We reviewed the medical records of patients admitted to neurology department of Sree Chitra institute Of Medical sciences &Technology ,which is a tertiary neurology care centre in South India,during January 1995 to January 2003, retrospectively ,who were detected to have CCAD on Intraarterial Digital substraction angiography or MRI/MRA and at least 3months follow up.Clinical features of the patients were assessed by reviewing the case files

.Cervicocranial artery dissection was defined as evidence of dissection such as string sign (tapered long segment stenosis), double lumen intimal flaps, flame shaped occlusion or an occlusion associated with mural haematoma on cervical MRI.

Angiographic characterstics were reviewed by an experienced neuroradiologist, who was blinded to the clinical features. Other comorbid risk factors like Hypertension, tobacco use, migraine, dyslipidemia and diabetes mellitus were also looked into.

Risk factor definition:

1)Arterial HTN: Defined as BP≥140/90mm on 2 different occasions outside the acute phase of stroke (after 1 week) or treatment with antihypertensive drugs during last 2 weeks prior to recruitment.

2)Diabetes mellitus: Documented by medical records before recruitment or fasting venous plasma> 120mg/dl out of acute phase.

3)Dyslipidemia:- If fasting blood cholesterol level > 220mg/dl, TG> 150/dl or LDL cholesterol > 130 or receiving cholesterol lowering agents prior to recruitment.

4)Migraine:- Defined according to the IHS criteria for migraine with and without aura.

5)Smoking- More than 10 cigarettes or bidies for at least 2 years prior to the onset of the stroke.

Outcome scale was defined similar to Glasgow outcome scale(GOS) and disability scoring was done by reviewing the case files 3months/ at last follow up as below

(Grade 1 Death.,2.Severe disability- Unable to walk without assistance or unable to attend to ADL without assistance.3. Moderate disability-Requires some help to look after ADL, but able to walk without assistance.4. Slight disability- Unable to carry out previous activities,but able to look after ADL.5.Normal)

RESULTS

A total of 30 patients were included in the study.

Of these 30 patients, 20(67%) were males and 10(33%) were females.

Internal carotid artery dissection were seen in 20patients(67%) and vertebral artery dissection in 10(33%) patients.

Mean age of the patients was 41.8 ±13yrs (Range 17-80 yrs).

Etiological association of trivial head/neck trauma in 5/30(16 %).

Other associated risk factors found were migraine 6 (20 %),tobacco use in 13 patients(43 %),hypertension in 11(37 %) ,dyslipidemia 11(37%) and Diabetes mellitus in 9(30%)

clinical characteristics of the patients were summarized in table1 below.

Table1.Clinical presentation.

Clinical features	Carotid Artery	Vertebral Artery
Stroke	14(70%)	8(80%)
TIA	1(5%)	1(10%)
Associated Headache	9(45%)	4(40%)
Associated Neckpain	5(25%)	3(30%)
Horner's Syndrome	7(35%0	3(30%)

Contrary to classical teaching of the triad of horner's syndrome, headache &ischemic stroke, as a characteristic feature of carotid artery dissection ,it was seen only in 25% of our patients.

In majority of the patients initial presentation was with ischemic symptoms rather than local symptoms.In those patients with inaugural local symptoms ,ischemic symptoms developed within mean duration of several minutes to less than 48 hrs..

Clinical suspicion of dissection was present in 10 patients(30%).One patient ,who had intracranial extension in the carotid territory,presented with subarachnoid hemorrhage .

Imaging details of the patients were summarized in table.2.

All of the patients had either CT brain or MRI brain for evaluation. More than 90% patients presented with evidence of thromboembolic strokes and <10% patients presented with hemodynamic strokes.

Table.2.Imaging characteristics.

CT/MRI Scan	30patients
Water shed infarcts	2(6%)
Thromboembolic infarcts	27(91%)
Subarchnoid hemorhage	1(3%)

Doppler study suggested dissection in 15(75%) patients for carotid territory dissection in the acute setting,while only in 3 (30%) in vertebral artery dissections.

All of our patients had either 4 vessel angiogram or MRI/MRA(only 3 patients) for definite diagnosis of dissection .Majority of our patient underwent 4 vessel angiogram within 2 weeks of onset of symptoms.

Angiographic characteristics of all the patients were summarized in table 3 and 4 down.

In carotid artery dissection the commonest site of dissection was distal to carotid bifurcation and in vertebral arteries the commonest site was the V3 segment(segment winding over atlantooccipital joint).

Classic signs of dissection like string sign was the commonest angiographic sign in our patients.Intracranial extension of dissection was seen in only 1(5%)patient of carotid dissection compared to 5(50%) patients in vertebral artery dissection. Bilaterality of dissection was seen in only minority of patients.

Table 3.Angiographic characteristics of carotid artery dissection.

Carotid artery (20 patients)
Site of dissection
Carotid bulb- 4 (20%)
ICA (2 cm from bifurcation) -16 (80%)
Extent of dissection
Intracranial extension – 1 (5 %)
Bilateral dissection – 1(5%)
Angiographic signs
String sign – 13 (65%)
Flame sign – 6 (30%)
Intimal flaps –1 (5%)

Table 4.Angiographic characteristics of vertebral artery dissection.

Vertebral artery (10 patients)
Site of dissection
V1 segment - 1 (10%)
V2segment - 2 (20 %)
V3segment - 4 (40%)
V4segment - 3 (30%)
Extent of dissection
Intracranial extension – 5 (50 %)
Bilateral dissection – 2(20%)
Angiographic signs
Stenosis (String sign) -3 (30%)
Occlusion -4 (40%)
Aneurysm -2 (20%)
Intimal flaps -1 (10%)

OUTCOME.

Mean duration of follow up is 17 months in our study. A total of 5(16%) patients lost to follow up despite repeated communications. Eventhough CCAD is a potentially serious condition the outcome seems to be benign with early diagnosis and intervention, which is evident in the present study. All of our patients received anticoagulants for at least 3 months from the time of diagnosis,if not otherwise contraindicated,followed by aspirin depending on status of recanalisation at 3months without reference to the time of onset of symptoms,.In cases of partial recanalisation anticoagulant is continued for next 3 months.Clinical outcome is assessed by using GOS, by reviewing the case records.Excellent outcome is seen in around 60% patients at last visit which ranged from 3-40 months in our study.During the follow up period mortality &recurrence is seen in only 1(3%) patient each in our study,confirming the benign long term prognosis in CCAD.Overall outcome is summarized in table5 below.

Table .5.Outcome

Outcome (GOS)	No. of patients (%)
Excellent outcome	15 (60)
Moderate disability	5(24)
Severe disability	4(16)
Recanalisation rate at 3 months	9(30)
Recanalisation rate at 6months	12(48)
Mortality	1(3)

DISCUSSION

This study is the largest series of patients with cervicocranial artery dissection(CCAD) from the Indian subcontinent .Diagnosis of CCAD is usually presumptive and based on the clinical presentation, angiography, MRI or CT and exclusion of other specific arterial wall pathology like atherosclerosis.

In our study CCAD constituted around 1.5% of all ischemic strokes in our institute during the study period and around 7% of stroke in young. Hence it constitutes an important cause of stroke in young,eventhough commonly it is an underdiagnosed condition.In our study there was a male predominance for CCAD, which was never documented in other series so far for both carotid and vertebral territory.

The average age of the patients with CCAD has most commonly been 41-46 years ,which agrees with the mean age of 42 years in our study.

Most common risk factor for traumatic dissection is blunt or penetrating trauma to the neck,and if trauma or a definable underlying diseases not identified,the dissection is labeled as spontaneous. Because repetitive microtrauma is possible in daily activities and because multiple vessel dissection without any reason seems unlikely,the concept of completely spontaneous dissection is questionable.In that case the underlying cause may be an unknown arterial disease,mechanical factor or both.Associated trivial head and neck trauma was present in only 4(13%)of our patients with spontaneous dissections, compared to 25-41% in various other series.

Many vascular risk factors and diseases are thought to be associated with CCAD.The major associated conditions found in our study are migraine, smoking hypertension in a similar proportion of patients in other series, even though their etiological contribution to dissection was not clear. None of our patients had associated abnormalities likefibromuscular dysplasia,cystic medial necrosis,marfan's syndrome, evidence of other connective tissue diseases or hyperhomocystinemia in contrast to association described in small percentage of patients in western literature.

The symptoms and signs of CCAD have a wide spectrum in literature.The symptoms reportedly considered typical of internal carotid artery dissection (ICAD) are head or neck ache and horner's syndrome followed hours or days later by focal motor or sensory deficits.The most typical symptoms of Vertebral artery dissection (VAD) are sudden occipital headache or neck pain on the side of dissection and symptoms of vertebrobasilar TIA or stroke.But the associated local symptoms are seen in only minority of our patients.

More than 70% of our patients presented with cerebral ischemic symptoms rather than local symptoms ,which was seen in only less than 20%.

Based on the stroke patterns of ICAD, Lucas et al described cortical and large subcortical infarcts >1.5cm were considered to be embolic in origin and small subcortical infarcts and junctional infarcts were of hemodynamic origin and also concluded that most of the infarcts were embolic rather than hemodynamic which is the case in our study also.

In our study >90% patients , cerebral ischemia appears to be thromboembolic compared to <10% patients in whom the ischemia appears hemodynamic considering above said imaging criteria.

Mokri et al has described an incidence of presentation with cerebral ischemic symptoms in 63-90% of their patients with dissection.

Clinical suspicion of dissection is in only 10 (33%) of our patients.

Conventional 4 vessel angiogram or MRA (only 3 patients)is required for confirmation of diagnosis in all of our patients.

Doppler study suggested dissection in 75% patients of carotid territory dissection compared to less than 30 % in vertebral territory,helping as a good screening device in carotid artery dissection for recruitment of patients for invasive angiogram.

Angiography showed varying signs in our patients ,but the commonest sign is of a tapered stenosis(string sign).Aneurysms are seen in vertebral artery territory more than carotid territory.

ICAD may be caused by a sudden stretching of ICA over the transverse process of upper cervical vertae in hyperextension and sudden flexion or or rotation of the neck.The most usual site of dissection is the cranial part of carotid bulb up to the foramen lacerum.In our study it is distal to carotid bifurcation in 80 % of patients comparable to existing datas.

Vertebral artery dissection may be caused by by stretching and compression of vertebral artery (VA) over the lateral masses of the C1 and C2 vertebral bodies in neck movements.This could be the reason of involvement of V3 segment of VA commonly in VAD ,as in our study.Third and fourth segments of the vertebral arteries are more prone for intimal tear and dissection in view of the marked mobility of the region and related tight fascial planes.

Intra cranial extension was uncommon for carotid territory compared to vertebral territory, because of the small size of transmitting foramen for carotid artery which limits the extension which is seen in our study also.

Treatment of patients with CCAD is controversial,as controlled studies are lacking in view of the rarity as well as the lack of recurrence in the natural course of dissections.Aiming at preventing cerebral embolism in patients with a stenotic artery in dissection, as documented by Sreenivasan et al by using transcranial Doppler sonography of frequent microemboli signal in carotid dissection,the most frequent therapy was heparin for a week followed by oral anticoagulants for 3-6 months often followed by aspirin, which is the treatment strategy in our study.

All of our patients received anticoagulants for at least 3 months from the time of diagnosis by angiogram, without any reference to the time of onset of symptoms,eventhough angiogram was delayed up to 1 year.

Endovascular stenting may be considered in patients who remain symptomatic due to thromboembolic events or progression despite anticoagulation and in cases of persisting aneurysm.

None of our patients underwent any surgical intervention during the follow up period. Recurrence of dissection is seen in only 1 patient in our study and is in the same vascular territory as the first dissection,which is similar to described literature..

Our mean follow up period is 17 months and follow up regime included Doppler ultrasonography for ICAD and 4-Vessel DSA for VAD at 3 months ,which helps in knowing the recanalisation of the vessels,to take decision regarding continuation of anticoagulants for next 3months if there is only partial recanalisation.

Up to 80 % of dissections are reported to normalize or recanalise various series.In our study the overall recanalisation rate is around 50%at the end of 6 months .Disabilty scale assessed by review of the case records and using a scale like GOS.there is no significant difference in the recanalisation rate at 3 and 6 months in our study.

Around 60% of our patients made an excellent recovery at last follow up in our study.Mortality is in only one patient in our study for a mean follow up of 17 months, which occurred in the acute setting as a consequence of the severity of acute vascular insult.

Thus eventhough CCAD was considered to be a potentially serious condition with a benign outcome.Our study confirmed the benign nature of its outcome in the long term, which ranged from 3- 40 months in our study.

BIBLIOGRAPHY

1. Jentzer-1954. Dissecting aneurysm of left ICA ;Angiology 5:235-234.
2. Fisher CM, Ojemann RG, Roberson GH.et al Sponteneous dissection of CCA. Canadian Journal of neurological sciences 1978:5:9-19.
3. Mokri B, Sundt TM jr, Houserow, Spontaneous ICAD, Hemicrania and Horner's syndrome. Arch. Neurol 1979:36: 677-680.
4. Spont carotid and vertebral artery dissection. Wouter I.S. NEJM 2001, 344:12:898-906.
5. Schievink WI, Mokri B, whisnant JP et al. ICAD in a community stroke 1993:24:1678-1680
6. Giroud M, Fayolle H, Andre N et al : Incidence of ACAD in a community of Dyon JNNP 1994 : 57 ;1443
7. Hart RG, Easton JD. Dissections of cervical and cerebral arteries: Neurologic clinics Vol. 1. 1983 : 155-182
8. Bougousslavsky J, Despland PA, Regli F: Spontaneous Carotid dissections with acute stroke: Arch. Neurol 1987 :44 : 137-140
9. Bostrom K, Liliequist B. Primary dissecting aneurysm of EC part of ICA and VA:report of 3 cases; Neurology 1967:17:179-186
10. Michael T. Hanelane, Arthur C C, Benjamin MF. Neurologist 2003:9:35-44
11. Hart RG, Easton JD, Dissections: stroke 1985:16:925-927
12. Grau A j, Brandt T, Buggle F et al. Association of cervical artery dissection with recent infection. Arch. Neurol. 1999:56:851-856,
13. Alessandro P, Elisabetta DZ, Alessandro P et al. Stroke 2002:33:664-669
14. WI Schievink, Molori P, O Fallon WM. Spontaneous recurrent cervical artery dissection. NEJM 1994:330:393-397.
15. WI Schievink , Mokri B, Kuieper BS et al. Stroke 1996:27:622:624
16. Mokri B, Silbert PL, Schievink WI et al. Cranial nerve palsy in SICAD.Neurology 1996:46:356-359
17. Sturzenegger M, Huber P. Cranial nerve palsies in SICAD. JNNP1993:56:1191-1199.

18. Biousse V, Massiou H, Bousser MG, Series of 80 Patients. Stroke 1995:26:235-239.
19. L R Caplan,C K Zarins,M Hemmati .Spontaneous dissection of extracranial vertebral arteries .Stroke ;16:No.6 1985 .
20. A Auer,S Ffelber,C Schmiauer et al MRA and clinical features of VAD JNNP;1998;64;474-481.
21. O Pelkonen,T.Tikkakoski ,S.Leinonen et al ICAD and VAD course and prognosis.Neuroradilogy 2003;45;71-77.
22. D Milhaud,Gabriel R,Bogousslavsky et al ;Occlusion due to carotid dissection.
23. Michael T H,DC,Arthur C,Benjamin M F.Association of ICAD and chiropractic manipulation.Neurologist;2003;9;35-44.
24. O Pelkonen,T.Tikkakoski ,S.Leinonen et al.Intracranial arterial dissection Neuroradilogy 1998;40:442-447.
25. Zetterling M,Cristrom C,Konrad P ;Internal carotid artery dissection ,Actaneurologica Scandinavia 2000;101:1-7.
26. Hart RG,Miller VT ;Crebral infarction in young adults:A practical approach;Stroke 1983;14;110-114.