Virchows Arch. A Path. Anat. and Histol. 384, 325–335 (1979)

Virchows Archiv A

Dissecting Aneurysm of Cerebral Arteries in Childhood and Adolescence

Case Report and Literature Review of 20 Cases

Herbert J. Manz[1], John Vester[2] and Bennett Lavenstein[3]

Departments of Pathology[1], Neurology[2], Pediatrics[3], Georgetown University Medical Center, Washington, D.C., USA

Summary. A nine year old girl died with a massive infarct of the right cerebral hemisphere causing transtentorial and foramen magnum herniation. The infarction was secondary to an idiopathic dissecting aneurysm. The case is unusual in that the supraclinoid segment of the right internal carotid artery, the anterior cerebral artery, and the middle cerebral artery and its three branches were embedded and sectioned longitudinally. The dissection commenced in the supraclinoid segment of the right internal carotid artery, extended into the middle and anterior cerebral arteries, and was accompanied by thrombosis of the false lumen. A literature review of 20 pediatric cases indicates the malignant natural course of the disease (76% mortality in the first two months), and emphasizes the characteristic angiographic "string sign". The diagnosis of cerebral arterial dissection during life depends on angiography and a high index of suspicion.

Key words: Dissecting aneurysm – Pediatric stroke – Intracranial vascular disease – Angiographic "string sign".

Introduction

Among the relatively uncommon strokes afflicting infants, children, and adolescents (1) is a small group of dissecting aneurysms of intracranial arteries reported with increasing frequency in recent years. A literature review discloses 20 cases under 19 years of age: to 1959, there were three; in the decade 1960–1969, there were six; and from 1970 onward eleven cases have been reported. With the exception of Wolman's (1959) and Fisher et al.'s (1978) description of two cases, all the others have been single case reports.

The more frequent publication of cases in the present decade suggests several possibilities: (a) the entity of intracranial dissecting aneurysms in the pediatric

Address offprint requests to: Dr. H.J. Manz, Georgetown University Medical Center, Department of Pathology, 3900 Reservoir Rd., N.W. Washington, D.C. 20007, USA

0340-1227/79/0384/0325/$02.20

age group is becoming more widely recognized (adult cases are more prevalent, though still uncommon): (b) anatomic pathologists, particularly pediatric pathologists and neuropathologists, are examining the cerebral vasculature more carefully; (c) the entity is becoming more prevalent, with the implication that environmental factors, including psychologic stress, diet, and various pollutants, play a contributory role, since it appears improbable that a dramatic change in the genetic pool has occurred.

In reporting another case, we wish to publicize the occurrence of intracranial arterial dissection in the pediatric population; by analyzing the reported cases, common etiologic, pathologic, or pathogenetic features may be uncovered which might prove of assistance in diagnosis and management of such patients. Our case concerns a nine-year old girl who died with massive infarction of the right cerebral hemisphere secondary to dissection and thrombosis of the supraclinoid segment of the internal carotid artery and its major branches.

Case Report

A previously healthy 9 year old Black girl developed sudden, right-sided headache followed by lethargy and twitching of the left arm and left side of the mouth. Physical examination revealed lethargy, left hemiparesis, a left Babinski sign, and left homonymous hemianopia. Lumbar puncture at another hospital demonstrated clear CSF under normal pressure, containing 140 erythrocytes per mm^3, and normal levels of protein and glucose.

At transfer to Georgetown University Hospital the next day she was obtunded and her head and eyes deviated to the right. Initial studies showed normal erythrocyte sedimentation rate, clotting studies and echocardiogram, and negative sickle cell preparation and vasculitis screen. A urinary sodium nitroprusside test was weakly positive, but a quantitative 24 h amino acid study revealed no homocystine. Computerized tomography demonstrated a low density zone consistent with infarction in the distribution of the right middle and anterior cerebral arteries and midline shift to the left. A tapering occlusion of the supraclinoid portion of the internal carotid artery without collateral flow was evident on selective angiography (Fig. 1). The patient was placed on Dexamethasone for elevated intracranial pressure due to edema.

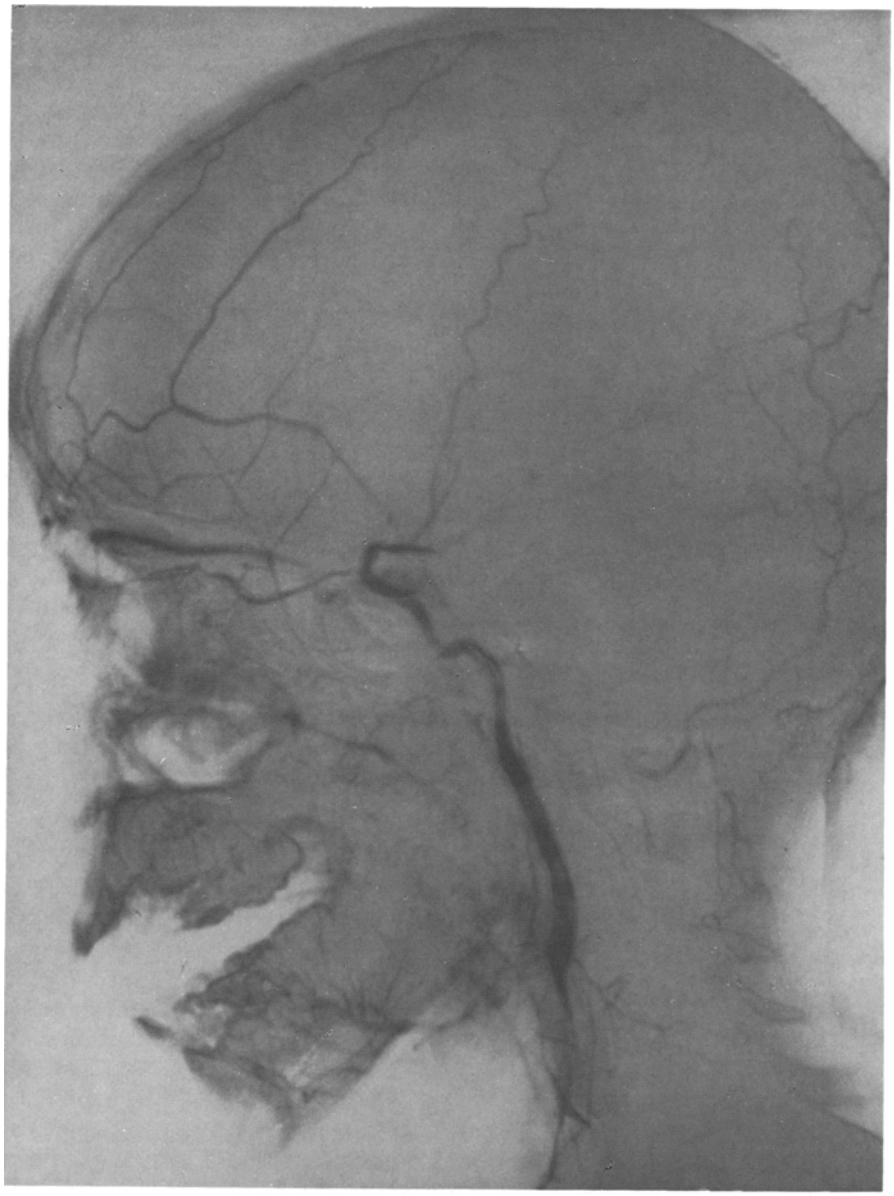
The following morning the patient became increasingly more stuporous; dilatation of the right pupil indicated transtentorial herniation. A subdural pressure monitor registered an initial pressure of 37 mm Hg, which, after head elevation and Mannitol infusion, fell to 5 mm Hg. Later that evening further pupillary dilatation and decerebrate posturing were noted. Intubation and hyperventilation (to a pCO_2 of 20–25 mm Hg) reduced intracranial pressure from 80 to 40 mm Hg. In spite of steroids, mannitol and hyperventilation, the patient had fixed, dilated pupils and no spontaneous respirations, 12 h later. Four days after the initial event she was pronounced dead and respirator care was terminated.

Autopsy Findings

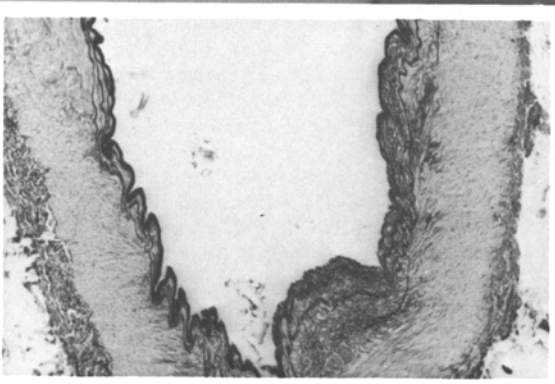
The body was that of a well-nourished, well-developed nine year old Black female without unusual physiognomy or body habitus. Except for a few lipid

Fig. 1. Right lateral carotid arteriogram demonstrates that the the lumen of the supraclinoid segment of the internal carotid arthery is markedly narrowed ("string sign") and the middle and anterior cerebral arteries occluded

Fig. 2. Transverse section of intracavernous segment of right internal carotid artery, showing splaying and reduplication of elastic lamina (VVG; ×17)



1



2

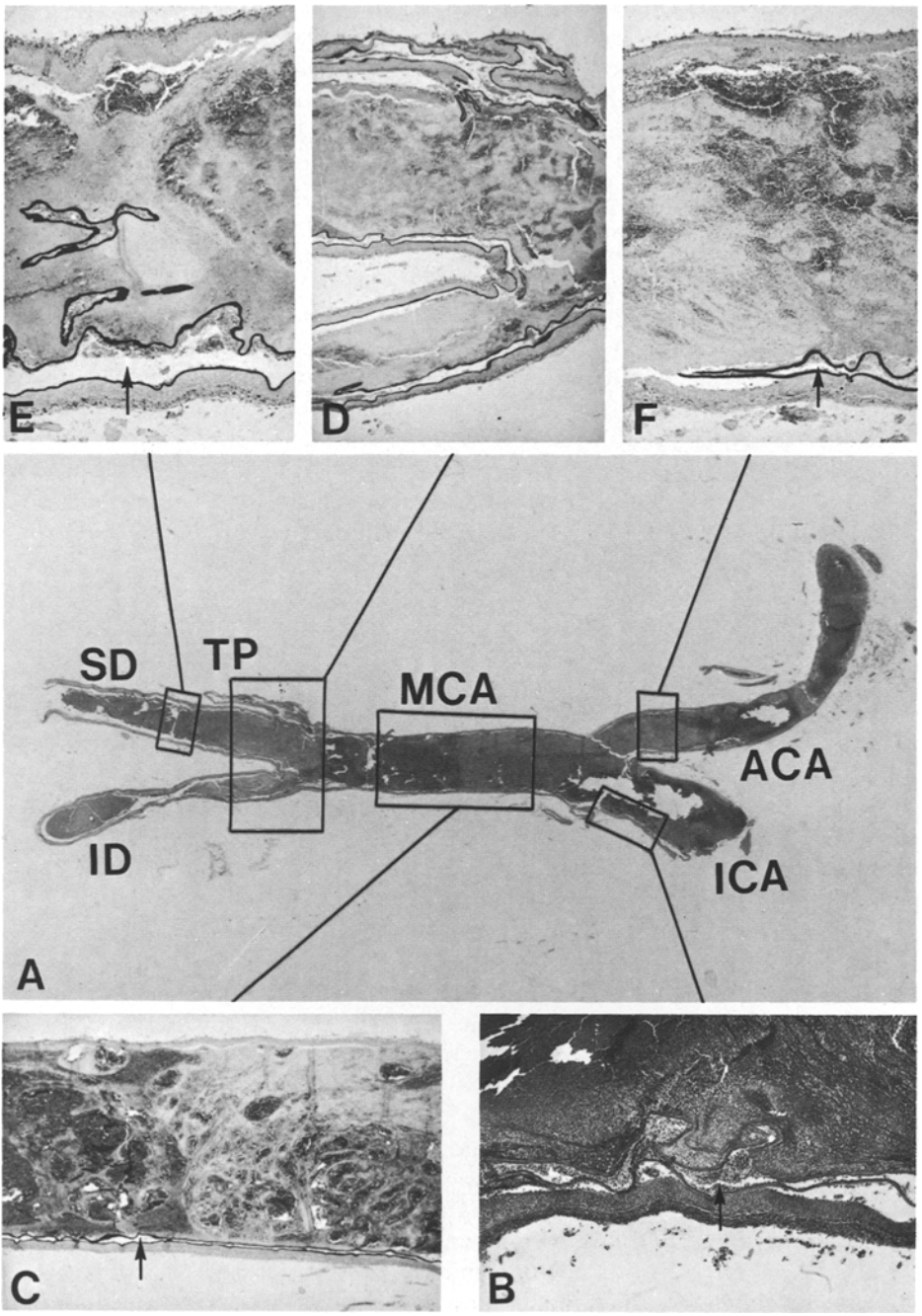
plaques in the aortic arch there were no lesions in the heart or large vessels from which emboli could derive. Patchy staphylococcal bronchopneumonia was present bilaterally. There were supernumerary renal arteries, but no evidence of fibromuscular dysplasia, cystic medial degeneration, or vasculitis in the major arteries of the body.

The brain weighed 1,350 g and was asymmetric, with the right cerebral hemisphere larger, softer, and paler. The gyri of both hemispheres were flattened and the sulci obliterated; a prominent right uncal groove and angulation and bruising of the right oculomotor nerve indicated transtentorial herniation. Both cerebellar tonsils were wrapped about the lower medulla oblongata and were displaced into the foramen magnum. The right internal carotid artery in its supraclinoid portion as well as the right middle cerebral artery were distended with solid, dark blue thrombus.

Cross sections of the terminal segments of each carotid siphon showed duplication and irregularity in thickness of the internal elastic lamina (Fig. 2). The supraclinoid segment of the right internal carotid artery, the anterior and middle cerebral arteries, and the proximal stems of the branches at the middle cerebral artery trifurcation were embedded longitudinally as a unit (Fig. 3A). Multiple sections were obtained and stained with hematoxylin-eosin (HE), Masson's trichrome, luxol-fast blue hematoxylin-eosin (LFB-HE), Verhoeff-van Gieson elastica (VVG), phosphotungstic acid-hematoxylin (PTAH), periodic acid-Schiff, Alcian blue, and Hale's colloidal iron.

The distal 7 mm of the supraclinoid segment of the right internal carotid artery showed dissection between the internal elastic lamina and the media, largely in the anterior and medial wall; the true lumen was compressed to a narrow slit, while the false channel was filled with laminated thrombus (Fig. 3B). The right middle cerebral artery was dissected for the entire length of 11 mm. along the anterior wall, and thrombus occluded the false lumen (Fig. 3C). At the trifurcation of the middle cerebral artery, the superior (anterior) division showed extension of the dissection for over 9 mm, with thrombosis of the false lumen; the posterior (inferior) division was occluded by thrombus, and a segment of dislodged elastica projected "downstream" from the middle

Fig. 3. A Longitudinal section of right internal carotid artery *(ICA)*; middle cerebral artery *(MCA)* and its major Sylvian branches: Temporo-polar *(TP)*, superior division *(SD)*, and inferior division *(ID)*; and anterior cerebral artery *(ACA)*. The false lumina of these vessels, except the temporopolar branch, are occluded with thrombus (PTAH; ×4.4). **B** Section of internal carotid artery illustrating the closely apposed elastic laminae, virtually obliterating the true lumen *(arrow)*. The false channel is filled with laminated thrombus. (PTAH; ×67). **C** Longitudinal section of middle cerebral artery demonstrating slit-like true lumen *(arrow)* outlined by apposed elastic laminae. The elastic lamina has been stripped from the superior wall of the vessel and the false channel is filled with thrombus (VVG; ×17). **D** The temporopolar branch is patent, but the superior and inferior divisions are occluded. The elastica has been stripped from the superior wall of the superior division. A segment of elastica from the middle cerebral artery has been carried "downstream" into the inferior division. (VVG; ×17). **E** In the superior division, the lumen *(arrow)* is again reduced to a narrow slit by the dissection along the superior wall. The intervening false channel is occluded by thrombus, in which curled-up fragments of dislocated elastica are embedded (VVG; ×44). **F** Longitudinal section of the anterior cerebral artery again demonstrates subintimal dissection along the upper wall with compression of the true lumen by thrombus in the false lumen. (VVG; ×44)


E
D
F
SD
TP
MCA
ACA
ID
ICA
A
C
B

cerebral artery; the temporopolar branch was uninvolved (Fig. 3D). A polymorphonuclear leukocytic infiltrate was present in the wall at the trifurcation of the MCA. In its branches, flap-like sheets of the internal elastic membrane were seen closely applied to the opposing wall; curled-up fragments of the elastica were embedded in the thrombus (Fig. 3E). The anterior cerebral artery was dissected for 14 mm along its superior wall (Fig. 3F), though focally the dissection appeared to be circumferential. The recurrent artery of Heubner and anterior communicating artery were uninvolved.

Intimal fibrous plaques in the siphon of both internal carotid arteries stained very intensely with Alcian blue and Hale's colloidal iron; significant deposits of acid mucopolysaccharides were not seen in the aorta and extracranial arteries.

The cerebral hemisphere in the distribution of the right middle cerebral artery exhibited acute ischemic change of neurons, congestion of blood vessels with margination and early infiltration of polymorphonuclear leukocytes, extensive edema, and pallor of myelin staining in the white matter. Sommer's sector of the right hippocampus also appeared acutely ischemic, but diencephalon and lentiform nucleus were intact. The right occulomotor nerve demonstrated extravasation of erythrocytes and smudged, pale-staining myelin, as commonly seen with transtentorial herniation.

Discussion

Review of Table 1 reveals that fourteen males and seven females were affected; whether this apparent predilection for males indeed indicates a trend or only chance variation in a small sample cannot be determined. Two patients had bilateral dissections of the internal carotid artery/middle cerebral artery systems, at intervals of six weeks (Adelman et al., 1974; Chang et al., 1975). The artery at greatest risk is the middle cerebral artery, being involved nineteen times (counting the bilateral cases); either singly (seven times) or conjointly with the internal carotid artery (eleven times) and/or anterior cerebral artery (seven times). No side was preferentially affected. The basilar artery was the site of dissection in two patients; in both, the intramural hemorrhage occurred in the media, not in the subintimal plane between elastica and media observed in the other 17 cases. The two cases of Fisher et al. (1978) were diagnosed by angiography and are still living.

The morbidity and mortality data disclose that twelve of the 21 patients died within one week of the dramatic onset of an intracranial catastrophe. Four more patients died within two months of sequelae related to the arterial dissection, including the two with bilateral dissections; these two died within one week of the second intramural hemorrhage (Adelman et al., 1974; Chang et al., 1975). By contrast, angiographically proved occlusive cerebrovascular disease of childhood has a better prognosis; 14 of 16 were alive after one year and one-third had no residual hemiparesis (Solomon et al., 1970). Similarly, among 16 pediatric patients with cerebral artery aneurysms, largely giant saccular and fusiform or mycotic aneurysms, Amacher and Drake (1975) obtained excellent operative results in 50%, and an overall mortality of only 38%. Significantly,

dissecting aneurysm during infancy, at one year of age or less, permitted survival of all three patients; two are apparently still alive, up to nineteen years later (Wolman, 1959; Shillito, 1964), while the third died of tuberculosis at 15 years of age (Norman and Urich, 1957). Dissecting aneurysms during infancy thus appear less frequently and rapidly lethal than those occurring in childhood; perhaps the still open fontanels prevent excessive increase in intracranial pressure and transtentorial herniation accompanying the cerebral hemispheral infarction and edema. The two cases of Fisher et al. (1978) are adolescents in whom the diagnosis of dissecting aneurysm was made by the typical angiographic "string sign".

Diagnosis of arterial dissection during life depends on angiography and a high index of suspicion. Ojemann et al. (1972), Hochberg et al. (1975), and Fisher et al. (1978) describe the angiographic picture of the "string-sign" as characteristic – a long segment of an artery having a tapering and very narrow lumen, and accompanied by eccentric stenosis. Descriptively, this feature was also recorded in three other cases (Jacob et al., 1970; Adelman et al., 1974; Chang et al., 1975) but no significance was attached by the observers. Only Johnson et al (1977), recognized the "string sign" as indicative of arterial dissection. In adult patients, Giedke et al. (1975) maintain that the only absolutely characteristic angiographic feature of a dissecting aneurysm is the demonstration of a double lumen. In this regard, Chang et al. (1975) report, "The right anterior cerebral artery was irregularly stenotic, with what appeared to be bands across the lumen".

Management of such cases is still largely a matter of applying general principles for massive cerebral infarction with edema, intracranial compartment shifts, and elevated intracranial pressure. Attempts at definitive, reconstructive vascular surgery have not as yet been made, except by endarterectomy of the cervical portion of the internal carotid artery in adults (Ojemann et al., 1972; Roome and Aberfeld, 1977). Anticoagulant therapy converts the ischemic infarct into a fatal hemorrhagic one (Hochberg et al., 1975).

A number of etiologic or pathogenetic factors has been implicated for dissecting aneurysms of intracranial arteries. It is interesting that in two of the first three cases reported, Wolman (1959) suggested congenital medial defects as causal. Trauma was deemed responsible in three (Ritchie, 1961; Dourov et al., 1964; Nelson and Styri, 1968), and a possible contributing factor in three more cases (Sato et al., 1971; Adelman et al., 1974; Johnson et al., 1977). Intense physical exertion was considered a major factor in three cases (Pikula et al., 1973; Fisher et al., 1978). Fibromuscular dysplasia (Hirsch and Roessmann, 1975), probable periarteritis nodosa (Wisoff and Rothballer, 1961), and congenital or acquired deficiency of elastica (Chang et al., 1975) were considered to be underlying causes, each in one case. In six cases the authors found no recognizable predisposing condition (Norman and Urich, 1957; Shillito, 1964; Hayman and Anderson, 1966; Jacob et al., 1970; Hochberg et al., 1975; Gagne et al., 1977).

Homocystinuria has recently been established as an inborn error of metabolism associated with intimal injury, systemic arterial and venous thrombosis, and strokes in childhood (Gerritsen and Waisman, 1972). The majority of pa-

Table 1. Literature review of intracranial dissecting aneurysms in the pediatric age group

Case	Sex	Age at dissection	Survival	Angiography	Artery dissected	Histopathology	Etiology	Comment	Reference
1	M	6 mos.	15 yr.	–	Right MCA and ACA	False & true lumen patent and communicating	?	Died of tuberculosis	Norman and Urich, 1957
2	M	16 yr.	4 da.	Right ICA block	Right ICA, MCA, ACA	Intramural aneurysm	Congenital defect	–	Wolman, 1959
3	F	Birth	19 yr. plus	–	Left MCA	Lumen organized and recanalized with open false channel	Congenital medial defect	Left hemispherectomy; alive 19 years later	Wolman, 1959
4	F	11 yr.	5 da.	Right ICA block	Right ICA, MCA, ACA	Focal medial degeneration and necrosis	Medial degeneration; polyarteritis?	–	Wisoff and Rothballer, 1961
5	M	17 yr.	5 da.	Left ICA occlusion	Left ICA MCA	Subintimal dissection	Trauma	–	Ritchie, 1961
6	M	1 yr.	1 yr. plus	Right MCA occlusion	Right MCA	Thrombus with elastica and inflammatory cells	–	Thrombectomy; alive 14 mos. later	Shillito, 1964
7	M	15 yr.	1 da.	Left MCA block	Left MCA	Subintimal dissection	Trauma	–	Dourov et al., 1964
8	M	15 yr.	8 wks.	Normal bilateral carotid	Basilar	Medial dissection	–	Slight mental retardation	Hayman and Anderson, 1966
9	M	5 yr.	3 da.	No filling, left ACA	Left ACA	Subintimal dissection	Trauma	–	Nelson and Styri, 1968
10	F	7 yr.	3 da.	Left MCA occlusion; proximal segment irregular	Left MCA	Subintimal dissection	–	–	Jacob et al., 1970
11	M	6 yr.	6 da.	No direct filling left MCA	Left MCA	Subintimal dissection	Trauma 2–3 months earlier	–	Sato et al., 1971

12	M	17 yr.	3 da.	Left ICA occlusion	Left ICA, MCA, ACA	Subintimal dissection	Exertion hypoxia?	–	Pikula et al., 1973
13	M	14 yr.	7 wks.	Right ICA supraclinoid occlusion, irregular lumen	Right ICA, MCA	Absence and duplication of elastica	Trauma? developmental abnormality	–	Adelman et al., 1974
			1 wk.	Abrupt left MCA occlusion	Left ICA, MCA				
14	F	11 yr.	Hours	–	Giant basilar	Arterial fibromuscular dysplasia, medial dissection	Fibromuscular dysplasia	–	Hirsch and Roessmann, 1975
15	F	15 yr.	14 da.	Right ICA "string sign"	Right ICA MCA, ACA	Subintimal dissection	–	–	Hochberg et al., 1975
16	M	8 yr.	6 wks.	Left ICA siphon irregular, narrowed and occluded branches	Left ICA, MCA	Elsastica split and frayed; false lumen organized	Congenital or acquired elastica deficiency?	–	Chang et al., 1975
			6 da.	Irregular lumen right MCA and ACA	Right ICA, MCA, ACA	Subintimal dissection		–	
17	F	6 yr.	4 da.	Right MCA and ACA branches occluded	Right MCA	Subintimal dissection	–	–	Gagne et al., 1977
18	M	8 yr.	2 da.	Right ICA siphon attenuation ("string sign")	Right ICA, MCA, ACA	Subintimal dissection with thrombosis	Trauma plus congenital abnormality	–	Johnson et al., 1977
19	M	13 yr.	?	Left MCA narrowing ("string sign")	Left MCA	–	strenous physical exercise	alive	Fisher et al., 1978 (case 20)
20	M	18 yr.	>3 mos.	Right PCA narrowing ("string sign")	Right PCA	–	strenous physical exercise	alive	Fisher et al., 1978 (case 21)
21	F	9 yr.	4 da.	Right ICA occlusion	Right ICA, MCA, ACA	Subintimal dissection	–	–	Present case

tients with this hereditary disease have also ectopia lentis, skeletal deformities resembling Marfan's disease, cutaneous abnormalities and variable degrees of intellectual deficit. According to Gibson et al. (1964), the aorta and large and small arteries of patients with homocystinuria have symmetric or eccentric deposits of metachromatic material and fibrous tissue in their intima as well as irregular thickening and fraying of the elastic lamellae. Large vessels also have pools of acid mucopolysaccharides in the media. McCully (1969) reported similar intimal fibrosis and elastica degeneration but without metachromasia, i.e., acid mucopolysaccharides. These observations must be balanced with those of Chang et al. (1975); in a systematic study of the middle cerebral arteries in two children dying from causes other than dissecting aneurysm, they found focal splitting and irregularity of the elastic lamina in addition to intimal pads and cushions at arterial branch points. The implication is that such changes may be very prevalent in the pediatric age group and not necessarily indicative of homocystinuria. These morphologic changes might nevertheless predispose to arterial dissection (Johnson et al., 1977).

In our case, intimal fibrous plaques staining metachromatically with Alcian Blue and Hale's colloidal iron were present in the cerebral arteries; the elastica of the terminal portions of both internal carotid arteries was splayed. The aorta and extracranial arteries did not demonstrate significant deposits of acid mucopolysaccharides in the media. We have no biochemical proof for the existence of homocystinuria. Our patient was of average intelligence, and did not have an abnormal body habitus; thus, the possibility of homocystinuria is excluded.

In the assessment of pediatric strokes, more common predisposing factors should be considered first, such as congenital heart disease, hematologic disorders (acute leukemias, hemophilia, sickle cell disease), trauma, infection and vasculitis, vascular malformations, migraine, and homocystinuria (Lagos and Siekert, 1969; Gold et al., 1973). Once these entities are ruled out by appropriate studies, the typical angiographic features and high index of suspicion may permit the antemortem diagnosis of dissecting aneurysm.

Acknowledgements. The authors express gratitude to Dr. Dieter Schellinger, Neuroradiologist, for the radiogram, to Mrs. S. Pulley for expert histopathologic processing, and to Mrs. M. Davis for typing the manuscript.

References

Adelman, L.S., Doe, F.D., Sarnat, H.B.: Bilateral dissecting aneurysms of the internal carotid arteries. Acta Neuropath. **29**, 93–97 (1974)

Amacher, A.L., Drake, C.G.: Cerebral artery aneurysms in infancy, childhood and adolescence. Child's Brain **1**, 72–80 (1975)

Carson, N.A.J., Neill, D.A.: Metabolic abnormalities detected in a survey of mentally backward individuals in Northern Ireland Arch. Dis. Child. **37**, 505–513 (1962)

Chang, V., Rewcastle, N.B., Harwood-Nash, D.C.F., Norman, M.G.: Bilateral dissecting aneurysms of the intracranial internal carotid arteries in an 8-year old boy. Neurol. **25**, 573–579 (1975)

Dourov, N., Locoge, M., Themelin, G., deRede, J.: Étude anatomo-clinique et radiologique d'un cas d'hématome disséquant d'une artère cérébrale chez un sujet jeune. Rev. Belge Path. **30**, 265–281 (1964)

Fisher, C.M., Ojemann, R.G., Roberson, G.H.: Spontaneous dissection of cervico-cerebral arteries. Canad. J. Neurol. Sci. **5**, 9–19 (1978)
Gagne, F., Lemay, M., Verret, S.: Hémodissection pariétale oblitérante des vaisseauz intracrâniens, une forme particulière d'anévrysme disséquant. Can. J. Neurol. Sci. **4**, 63–65 (1977)
Gerritsen, T., Waisman, H.A.: Homocystinuria. Cystathionine synthase deficiency. In: The Metabolic basis of inherited disease. Stanbury, J.B., Wyngaarden, J.B. and Fredrickson, D.S. (eds.). Third edition, pp. 404–412. New York: McGraw-Hill 1972
Gibson, J.B., Carson, N.A.J., Neill, D.W.: Pathological findings in homocystinuria. J. Clin. Path. **17**, 427–437 (1964)
Giedke, H., Kriebel, J., Sindermann, F.: Dissecting aneurysm of the petrous portion of the internal carotid artery. Case report and review of previous cases. Neuroradiology **10**, 121–124 (1975)
Gold, A.P., Challenor, Y.B., Gilles, F.H., Hilal, S.P., Leviton, A., Rollins, E.I., Solomon, G.E., Stein, B.M.: Strokes in children (Part I). Stroke **4**, 834–894 (1973)
Hayman, J.A., Anderson, R. McD.: Dissectiong aneurysm of the basilar artery. Med. J. Austral. **2**, 360–361 (1966)
Hirsch, C.S., Roessmann, U.: Arterial dysplasia with ruptured basilar artery aneurysm: Report of a case. Human Path. **6**, 749–758 (1975)
Hochberg, F.H., Bean, C., Fisher, C.M., Roberson, G.H.: Stroke in a 15 year old girl secondary to terminal carotid dissection. Neurol. **25**, 725–729 (1975)
Jacob, J.C., Maroun, F.B., Heneghan, W.D. and House, H.M.: Uncommon cerebrovascular lesions in children. Develop. Med. Child Neurol. **12**, 446–453 (1970)
Johnson, A.C., Graves, V.B., Pfaff, J.P., Jr.: Dissecting aneurysm of intracranial arteries. Surg. Neurol. **7**, 49–52 (1977)
Lagos, J.C. and Siekert, R.G.: Intracranial hemorrhage in infancy and childhood. Classification and outline of management. Clin. Pediatr. (Phil.) **8**, 90–97 (1969)
McCully, K.C.: Vascular pathology of homocystinemia: Implications for the pathogenesis of arteriosclerosis. Am. J. Path. **56**, 111–128 (1969)
Nelson, J.W., Styri, O.B.: Dissecting subintimal hematomas of the intracranial arteries: Report of a case. J. Am. Osteopath. Assoc. **67**, 512–517 (1968)
Norman, R.M., Urich, H.: Dissecting aneurysm of the middle cerebral artery as a cause of acute infantile hemiplegia. J. Path. Bact. **73**, 580–582 (1957)
Ojemann, R.G., Fisher, C.M., Rich, J.C.: Spontaneous dissecting aneurysm of the internal carotid artery. Stroke **3**, 434–440 (1972)
Pikula, B., Plamenac, P., Ferkovic, M.: Anévrisme disséquant (hématome disséquant) d'une artère cérebrale après un effort physique intense. Rev. Neurol. **128**, 125–130 (1973)
Ritchie, G.: Dissecting aneurysm of the left internal carotid and left middle cerebral arteries. Report of a case. Wisc. Med. J. **60**, 556–558 (1961)
Roome, N.S., Jr., Aberfeld, D.C.: Spontaneous dissecting aneurysm of the internal carotid artery. Arch. Neurol. **34**, 251–252 (1977)
Sato, O., Bascom, J.F., Logothetis, J.: Intracranial dissecting aneurysm. Case report. J. Neurosurg. **35**, 483–487 (1971)
Shillito, J. Jr.: Carotid arteritis: A cause of hemiplegia in childhood, J. Neurosurg. **21**, 540–551 (1964)
Solomon, G.E., Hilal, S.K., Gold, A.P., Carter, S.: Natural history of acute hemiplegia in childhood, Brain **93**, 107–120 (1970)
Wisoff, H.S., and Rothballer, A.B.: Cerebral arterial thrombosis in children. Arch. Neurol. **4**, 258–267 (1961)
Wolman, L.: Cerebral dissecting aneurysms. Brain **82**, 276–290 (1959)

Received June 15, 1979