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Retinal Arterial Occlusion Due to Embolism of Suspected Cardiac Tumors – Report on Two Patients and Review of the Topic

D. Schmidt[1], A. Hetzel[2], A. Geibel-Zehender[3]

[1]Department of Ophthalmology,
[2]Department of Neurology,
[3]Department of Internal Medicine,
University of Freiburg, Germany

Abstract
Background: Ophthalmic complications due to heart tumors are rare.
Patients: This case report describes two patients with ocular complications, caused by a suspected cardiac tumor. A 56-year-old woman with arterial hypertension had a severe episode of headache coinciding with an acute loss of vision in her right eye and left-sided hemiparesis. A 20-year-old woman noticed sudden transient visual loss in her right eye.
Result: The 56-year-old woman had an infarction on the right side of the middle and posterior cerebral arteries and, simultaneously, a central retinal artery occlusion (CRAO) in her right eye. Echocardiography revealed a tumor in the left atrium. The tumor disappeared after treatment with phenprocoumon within a few days. The diagnosis of a cardiac thrombus was made. The 20-year-old woman noticed recurrent episodes of sudden, transient visual loss in her right eye. A branch retinal arterial occlusion (BRAO) in her right eye was diagnosed. Echocardiography revealed a myxoma in the left atrium. The tumor was successfully excised surgically.
Conclusion: In any vascular disturbance in the eye suspected to be embolic in origin, echocardiography should be carried out in order to exclude the presence of a heart disease.

Key words: central retinal artery occlusion, branch retinal artery occlusion, myxoma, intracardial thrombus, echocardiography

Introduction

Sudden visual disturbances are always suspected to be caused by severe vascular diseases. Diseases of the carotid artery or the heart may lead to embolic events in the eye, i.e. central retinal artery occlusion or branch retinal artery occlusion. It is mandatory to perform echocardiography and Doppler sonography of the extracranial and intracranial carotid arteries to identify the cause of the ocular vascular disease. Atrial myxomas are rare. Surgery offers the possibility to prevent early re-embolism. Therefore, early diagnosis is essential to reduce morbidity or mortality from cardiac dysfunction and embolic complications. Diagnosis of cardiac tumors are non-specific; cardiac imaging by echocardiography, computer tomography, or angiography are important. However, transesophageal echocardiography is superior to other methods of examination, as was demonstrated by Saurbier et al. (1997). Benign tumors of the heart can occur as "myxoma syndrome", defined as cardiac myxoma with lentiginosis, extracardiac tumors, and family clustering of cardiac myxomas. However, most cardiac tumors are "sporadic myxomas". Geibel et al. (1996) evaluated 72 consecutive patients with cardiac and/or mediastinal tumor lesions echocardiographically. Twenty-three patients had *benign* tumors located in the hearts of 20 patients. Three tumor lesions were situated outside the heart, namely a pericardial cyst, dermoid cyst, and teratoma. Echocardiography is important for the localization, however histology is necessary for final assessment of the tumor.

For decades, cardiogenic embolism has accounted for about 15% of all ischemic strokes in clinical studies (Cerebral Embolism Task Force 1986).

The severity of embolisms in retinal arteries depends on their type, quantity and size. The first author to provide a detailed description of central retinal artery occlusion (CRAO) in a patient with a cardial myxoma was Marchand (1894). We describe the findings diagnosed by echocardiography of two patients with ocular complications due to embolic events of cardiac tumors.

Case Reports

1. Central Retinal Artery Occlusion (CRAO) in a Patient with Suspected Myxoma

A 56-year-old woman with arterial hypertension had suffered from migraine for about 30 years. The patient had sinus rhythm and no tachycardia. She developed a severe episode of headache together with an acute loss of vision in her right eye concurrent with a left-sided hemiparesis in November 1998. The totally blind right eye showed the typical signs of a central retinal artery occlusion. CT revealed a right-sided infarction of the middle and posterior cerebral arteries. Echocardiography showed the signs typical of a tumor in the left atrium.

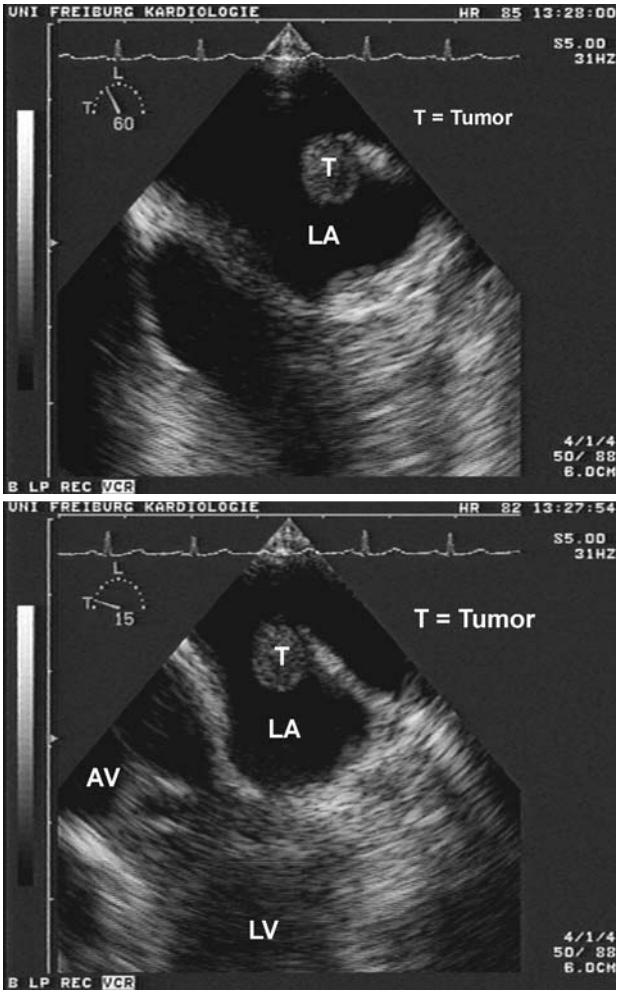

Fig. 1. Tumor in the left atrium.

A myxoma was suspected, but before surgery was planned, the patient was treated with phenprocoumon (Marcumar®) (Fig. 1). With this treatment, the tumor completely resolved within a few days, revealing that a large thrombus was the explanation for the echographically-diagnosed finding. Upon examination took place several months and years later, the eye remained totally blind and the hemiparesis persisted unchanged.

2. Branch Retinal Artery Occlusion in a Patient with a True Atrial Myxoma

A 20-year-old woman noticed a sudden transient visual loss in her right eye in 1997. About three years prior to that episode, she had noted visual disturbances such as glittering in her right eye together with headache that were interpreted as migraine attacks. There were recurrences of these episodes, and in the summer of 2000, a branch retinal arterial occlusion in her right eye was diagnosed. Echocardiography showed a tumor in the left atrium that proved to be a typical myxoma which was surgically removed (Fig. 2).

The visual field examination four weeks after the operation showed a defect of the nasal superior quadrant. Retinal edema had completely resolved and the artery showed regular blood flow.

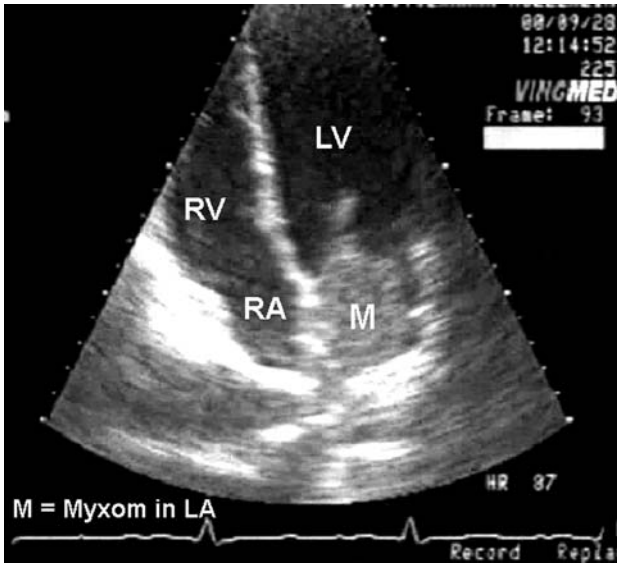

Fig. 2. Myxoma in the left atrium (cardiac myxoma).

Review of Case Histories Described in the Literature

In the literature we found 60 case histories of patients with ocular involvement due to embolic events caused by *atrial myxomas*.

Group I. Patients with Minor Ocular Deficits (Table 1)

We compiled from the literature the data from 13 patients who had no massive ocular changes, such as amaurosis fugax or blurring of vision (6), BRAO (2), retinal infarction without a detailed description of the extent of the infarction (2), "pallor of the fundus" (2), and visual field defects (1).

The mean age of the 13 patients was 29.6 years (8-63), seven female and six male patients.

However, we found that eight the 13 patients also had severe neurological deficits, either hemiplegia or cerebellar signs (8), loss of consciousness (1), an embolic event in a lower extremity (1), and one patient died.

Group II. Patients with Massive Ocular Deficits (Table 2)

In addition, we summarized the data from the case histories in the literature of 27 patients who went blind due to central retinal artery occlusion (CRAO) (22), unilateral optic atrophy (4), anterior ischemic optic neuropathy (AION) (1).

In this overview of the literature, we demonstrate that most patients had severe neurological deficits, most of them suffered hemiparesis in addition to visual loss (16), embolic events in the extremities (3), dysphasia (1), syncopal attack (1), transient aphasia (1), prosopagnosia (1), transient dizziness (1), and two patients died.

The mean age of the 27 patients was 37.26 years (8-75), 14 females and 13 males, indicating that the pa-

Table 1. Retinal infarctions or blurring of vision due to myxoma of the heart.

Author	Age, sex	signs of generalized embolic infarctions	Ocular signs
1. Al-Mateen et al. (2003)	11, f	acute right hemiplegia, red spots of the nail beds and on the soles of the feet, right facial paresis, dysarthria	LE: BRAO
2. Al-Mateen et al. (2003)	10, m	transient dizziness, unsteady gait, left-sided headache, MRI: lesions in the cerebellar hemispheres	transient visual disturbances, double vision, dysconjugate eye movements
3. Donaldson et al. (1981)	36, m	atrial arrhythmia, progressive dyspnoea	LE: transient blindness "curtain crossing the visual field", emboli in retinal vessels
4. Goodwin et al. (1962)	63, m	five episodes of vertigo, followed by loss of consciousness for up to 10 minutes	episodes of blurring of vision
5. Landers et al. (2000)	8, f	right shoulder pain and cough, right-sided hemiparesis due to embolus of the left middle cerebral artery	right visual field defect
6. MacGregor and Cullen (1959)	56, m	increasing tiredness, breathlessness, febrile temperature, headache, crepitations at both lungs, anginal pain, coma, death	RE: amaurosis fugax (five minutes)
7. Maroon and Campbell (1969)	13, f	1. sudden confusion, ataxia, dysarthria, right supranuclear facial palsy, right hemiparesis, 2. additional syncopal episodes 3. left hemiparesis	"marked pallor of the left fundus"
8. Newman et al. (1966)	33, f	sudden onset of pain, coldness and blanching in her left foot	episode of temporary loss of the upper half of the visual field
9. Sandok et al. (1980)	25, m	two right TIAs, vertigo alone, left lateral medullary infarction	"retinal infarction"
10. Sandok et al. (1980)	52, f	left cerebral infarction	"retinal infarction"
11. Stoane et al. (1966)	13, f	syncope, right facial weakness, hemiparesis right side	left fundus: "striking pallor"
12. Thompson and Simmons (1974)	46, f	sudden left-sided hemiparesis, complete occlusion of the right internal carotid artery and left brachial artery	bilateral visual blurring, more severe on the right
13. Yasuma et al. (1989)	19, m	no signs of generalized disease	LE: BRAO
	29.6 years (8-63) **f: 7** **m: 6**	hemiplegia and/or cerebral or cerebellar infarction: **8** loss of consciousness: **1** embolic event of the lower extremity: **1** progressive dyspnoea: **1** death: **1** no generalized disease: **1**	Amaurosis fugax or visual blurring: **6** BRAO: **2** "retinal infarction": **2** "pallor of the fundus": **2** visual field defect: **1**

tients in this group were older than the 13 patients from Table 1.

Group III. Patients with Severe Neuro-ophthalmological Signs Deficits (Table 3)

Furthermore, we collected the data from 20 case histories of the literature with neuro-ophthalmological signs, such as hemianopia (most were homonymous) (9), diplopia or gaze paresis (4), nystagmus (3), skew deviation (1), Parinaud syndrome (1), absent corneal reflexes (1), or papilledema (1). We found 19 patients in this group who also had severe neurological deficits; most of them revealed hemiplegia due to stroke (16), loss of consciousness (1), weakness in one arm (1), ataxia (1), and TIA (1).

The mean age of the 20 patients was 46 (21-64) years, 12 females and eight males. In this group, patient mean age was also higher than that of patients in group I.

Discussion

Various diseases can lead to vascular retinal obstructions, and any non-inflammatory changes in the retinal arteries may be significant. They may be caused by a *local thrombotic* mechanism, but it is more often due to an *embolic* process originating from the the heart or the carotid artery (Mangat et al. 1995). We describe

Table 2. Central retinal artery occlusion (CRAO) or unilateral optic atrophy due to myxoma of the heart.

Author	Age, sex	signs of generalized embolic infarctions	Ophthalmological signs
1. Anderson and Lubow (1973)	13, m	loss of consciousness, left hemiparesis, aphasia, painful right foot, red spots on the sole of his foot	impaired saccadic movements to the left, RE: CRAO
2. Budzilovich et al. (1979)	62, f	1. right hemiparesis, aphasia, right central facial weakness 2. loss of consciousness, decerebrate posture, death due to occlusion of the left middle cerebral artery	LE: "optic atrophy" and empty arterioles in the fundus, ptosis 2. LE: third nerve palsy
3. Campbell (1974)	45, m	partial right hemiparesis	LE: embolic CRAO
4. Cogan and Wray (1975)	17, m	1. malaise, night sweats, anorexia, pains in the legs, red spots on the hands, 2. right hemiplegia, aphasia 3. bilateral occlusion of iliac arteries (saddle embolus)	LE: CRAO
5. Cogan and Wray (1975)	20, f	1. vertigo due to vascular occlusion of the brain stem, diffuse aneurysms of the cerebral cortical arteries, transient swelling and discoloration of one foot, 2. seizures, tetraplegia, death due to embolic infarction in the cerebrum, cerebellum and brain stem	1. third nerve paralysis, 2. right-sided internuclear ophthalmoplegia, branch retinal artery occlusion 3. left homonymous hemianopia, LE: CRAO
6. Donoso et al. (1981)	55, m	pain in the right leg, obstruction of the popliteal artery, mild dysphasia, decreased sensation over the right side of the face	LE: CRAO
7. Furlong and Verdile (1995)	65, f	numbness in right upper and lower extremities, left frontal headache	LE: pale fundus (suspected CRAO)
8. Gleason (1955)	13, f	syncopal attack, splinter hemorrhages in the nailbeds, low-grade fever, leukocytosis, gangrene of the right toes, auricular fibrillation	LE: CRAO
9. Jampol et al. (1973)	37, f	episodic joint pains, low-grade fewer, rash on the sole of the feet, infarct of the kidney, episode of hearing loss, transient aphasia	LE: CRAO
10. Knepper et al. (1988)	75, m	confabulation, denial of visual loss, prosopagnosia	1. right homonymous hemianopia 2. LE: pale optic disc (blind eye),
11. Leonhardt and Kullenberg (1977)	35, m	diffuse muscle and joint pain, bilateral pleuritis, myocardial infarction 2. recurrent attacks of headache and dizziness, transient hemiparesis	RE: BRAO, LE: incomplete CRAO
12. Lewis (1994)	53, f	right hemiparesis, aphasia due to a left lenticostriate infarct	LE: CRAO and choroidal infarction, no cherry-red spot
13. Marchand (1894)	37, m	vertigo, right hemiparesis, aphasia	LE: CRAO
14. Maroon and Campbell (1969)	29, m	pain and tenderness in the legs, minute haemorrhagic spots on the palms of both hands, splinter haemorrhages in the nail beds, sudden hemiparesis, "pseudoaneurysms" on branches of the right middle cerebral artery	RE: optic atrophy
15. Nettl et al. (1957)	31, f	headache, left hemiplegia, death due to embolic occlusion of the right internal carotid artery	papilledema, RE: CRAO
16. New et al. (1970)	17, m	tender red spots on the hands, stabbing chest pain, aching and pain in the legs, diminished left carotid pulse, diminished left superficial temporal pulse	LE: CRAO

17. New et al. (1970)	21, f	1. headache, confusion, aneurysms in the parietal division of the right middle cerebral artery, 2. disturbance of consciousness, minor seizures, facial paresis, 3. flaccid left hemiparesis 4. right hemiplegia, left hemiparesis, death	1. diplopia, slight ptosis, mydriasis, weakness of the left medial und superior rectus muscles (incomplete oculomotor paresis) 2. old chorioretinitis, small hemorrhage in the right fundus 3. left homonymous hemianopia 4. LE: CRAO
18. Porrini et al. (2000)	42, f	paresthesia of the inferior limb	RE: occlusion of the posterior ciliary arteries with AION
19. Rafuse et al. (1997)	45, f	right-sided hemiparesis, aphasia	LE: painful eye, infarction of the ophthalmic artery
20. Reichling (1934)	38, m	1. syncopal attack 2. loss of consciousness, death due to embolic occlusion of the middle cerebral artery	LE: CRAO
21. Salehian et al. (2001)	29, m	no symptoms	LA painless loss of vision with diffusely pale retina
22. Schwarz et al. (1972)	40, f	1. right hemiparesis 2. left hemiparesis	LE: sudden blind, optic atrophy
23. Stoane et al. (1966)	20, m	numerous small red spots in the palms of both hands, stroke (partially paralyzed on the left side)	1. LE: decreased vision 2. RE: optic atrophy
24. Sybers and Boake (1971)	43, f	right hemiparesis, aphasia	LE: amaurosis fugax, later CRAO
25. Thompson et al. (1988)	60, f	1. dizziness, nausea, transient paresthesias of the left face and hand 2. aphasia, right-sided weakness in the face and arm 3. cardiac arrest, death	1. left beating gaze-evoked nystagmus 2. LE: CRAO
26. Tipton et al. (1977)	56, f	bifrontal headache, transient dizziness	LE: occlusion of several retinal arterioles
27. Tönz et al. (1992)	8, m	1. generalized seizures, recurrent red spots on both hands and feet, pain in the calves, 2. acute right hemiparesis, aphasia	1. no visual disturbances 2. LE: CRAO
	37.26 (8-75) years **f: 14** **m: 13**	hemiparesis: **16** embolic events in the extremities: **3** death: **2** dysphasia: **1** syncopal attack: **1** transient aphasia: **1** prospagnosia: **1** transient dizziness: **1** no signs or symtoms: **1**	CRAO or infarction of the ophthalmic artery: **22** optic atrophy: **4** anterior ischemic optic neuropathy (AION): **1**

herein the case histories of two patients with embolic events due to cardiac tumors and we also collected case histories from patients with cardiac myxomas from the literature. In our first patient, who showed sinus rhythm and no tachycardia, an embolic episode caused by cardiac arrhythmia was unlikely. The disappearance of the "tumor" after anticoagulation was proof that no myxoma existed. However, the second patient presented a myxoma, as revealed by surgery and histological evaluation. It is important to emphasize that even in artery occlusion of a small retinal branch of the eye, such an embolic event could be caused by a cardiac tumor. Therefore, in any vascular disturbance in the eye, cardiac disease should be suspected and echocardiography should be carried out. If no change in tumor size after conservative treatment occurs, surgical excision of the tumor is indicated.

Retinal emboli consist either of cholesterol ("Hollenhorst plaques"), or fibrin, or they are calcareous (originating from the heart valves), or a combination of these different embolic types may occur. Retinal emboli caused by myxomas are rare, as myxomas are rare cardiac tumors and - by no means does every myxoma cause retinal emboli. Heart valve vegetations (Greven et al. 1995, Schmidt and Zehender 1999), fat emboli after bone fracture, parasites, air, and even particles of replacement heart valves have also caused vascular retinal occlusions (Ffytche 1974).

Cholesterol and platelet-fibrin emboli occur more often than calcific emboli (Arruga and Sanders 1982).

Table 3. Neuro-ophthalmological signs in connection with stroke due to myxoma of the heart.

Author	Age, sex	signs of embolic infarctions, mainly neurological signs	Ophthalmological signs
1. Branch et al. (1985)	53 f	migraine headache, occasional numbness and tingling in the hand, arteriogram: multiple fusiform aneurysms in the distributions of the carotid arteries, occlusion of the right posterior cerebral artery	sudden left homonymous hemianopia
2. Browne et al. (1993)	63, f	1. ischemic stroke of the left middle cerebral artery 2. coma, no spontaneous motor movements, death due to multiple intracranial emboli	1. no eye findings 2. eyes were deviated to the right, corneal reflexes were absent
3. Burton and Johnston (1970)	41, f	right-sided hemihypalgesia, dysphasia, headache	right hemianopia (lasting for 6 hours)
4. Butler et al. (1986)	31, f	left hemiparesis, left hemianesthesia	left homonymous hemianopia (incongruous)
5. Damasio et al. (1975)	43, f	central left facial palsy (episode), left hemiparesis	diplopia (episode), limitation in upward gaze, rotatory nystagmus
6. Desousa et al. (1978)	44, f	three strokes in four years, right hemiplegia, right facial weakness	bilateral papilledema, right hemianopia
7. Frank et al. (1979)	49, f	1. progressive defects in memory, bifrontal headaches, anemia, vertigo, intermittent fever 2. right hemiparesis	right inferior homonymous quadrantanopia
8. Gorlitzer (1934)	50, m	loss of consciousness due to cerebral hemorrhages, seizures	horizontal nystagmus
9. Huston et al. (1978)	38, m	1. rash over the dorsum of the right foot, 2. cramping pain in the right forearm, discoloration of the right index finger 3. disorientation, ataxia, nausea, left facial paresis 4. left hemiparesis, ataxia	left internuclear ophthalmoplegia, homonymous hemianopia
10. Knepper et al. (1988)	57, m	ataxia, difficulty finding the correct words, apnea	1-hour episode of binocular vertical diplopia
11. Manschot (1959)	57, m	right hemiparesis, right central facial paresis, aphasia, coma	gaze paresis to the right, suspicion of juxtapapillary chorioretinitis, choked disc
12. Morton-Bours et al. (2000)	51, m	MRI: bilateral hyperintense lesions in the cerebellum, triangular hyperintense lesion in the mid-pons (history of infarction) weakness and dysmetria of the right arm and leg	horizontal nystagmus on left gaze
13. Price et al. (1970)	21,f	1. headache, confusion 2. pain, swelling, discoloration of her left foot, 3. left hemiparesis, sudden right-sided headache 4. convulsive seizures, deep coma	1. diplopia, slight ptosis, mydriasis, weakness of the left medial and superior rectus muscle 2. RE: small retinal hemorrhage, left homonymous hemianopia 3. skew deviation, vertical bobbing movements 4. fundi became pale and ischemic
14. Reichmann et al. (1992)	39, f	1. weakness, fatigue, anaemia, high ESR 2. brachiofacial hemiparesis, MRI: hyperintense area in the quadrigeminal area	2. visual disturbances, Parinaud syndrome
15. Rankin and DeSousa (1978)	44, f	1. third stroke with right hemiplegia and aphasia 2. right central facial weakness and spastic right hemiplegia, lethargia due to metastasis of myxoma in the left temporal lobe	bilateral papilledema, right hemianopia
16. Sandok et al. (1980)	30, f	vertigo, left-sided paresis and sensory loss	diplopia
17. Selzer et al. (1972)	64, m	1. sudden hemiplegia, 2. paresis of fingers of the left hand 3. right hemiplegia, aphasia	1. no eye signs 2. transient double vision 3. unilateral papilledema on the left side

18. Yarnell et al. (1971)	46, f	left hemiplegia	left homonymous hemianopia
19. Yarnell et al. (1971)	53, m	TIA left side, 50% stenosis of the right carotid bifurcation, left-sided hyperreflexia	blurring of vision, rotatory nystagmus
20. Yufe et al. (1976)	46, m	1. myalgia, fleeting arthralgias, petechial rash on the arms, 2. impaired speech 3. weakness of the right arm, elevated ESR	1. transient blindness in both eyes. lasting 5 to 10 minutes, 2. right homonymous hemianopia
	46 years (21-64) f: 12 m: 8	hemiplegia or occlusion of cerebral arteries: **16** loss of consciousness: **1** weakness of one arm: **1** ataxia: **1** TIA: **1**	hemianopia: **9** diplopia or gaze paresis: **4** nystagmus: **3** Parinaud syndrome: **1** skew deviation: **1** absent corneal reflexes: **1** papilledema: **1**

Other rare embolic sources exist, such as talc, atrial myxoma, or thrombi from the heart or an aneurysm. Emboli in 21 young adults (under 40 years of age) were described by Greven et al. (1995). Cardiac valvular disease was the most commonly recognized etiologic agent of an arterial occlusion in the retina in young patients. Some of them presented an atrial myxoma, others bacterial endocarditis. A hypercoagulated state with oral contraceptives and cigarette smoking, or inherited protein S-deficiency were other causes of the arterial retinal disease. On the other hand, retinal arterial occlusions may also arise in arterial hypertension (Hayreh 1971), or may be due to an arterial spasm in migraine (Greven et al. 1995, Humphrey 1979).

A cardiac myxoma seems to be a rare cause of vascular disturbance in the eye, however, ocular episodes due to emboli of a cardiac tumor have often been observed in the literature (Table 1-3). Peripheral embolic events are often misleading, so that wrong diagnoses were made previously, before echocardiography became routine during medical investigations. A left atrial myxoma was frequently confused with mitral stenosis, an erroneous antemortem diagnosis (Prichard 1951, Newman et al. 1966). Left atrial myxomas have also been confused with inflammatory disease, such as lupus erythematosus (Yarnell et al. 1971), Hamman-Rich syndrome, "myocarditis", subacute bacterial endocarditis, or even acute rheumatic fever (Anderson and Lubow 1973, Bigelow et al. 1969, Goodwin et al. 1962, Manns et al. 1986, New et al. 1970). Other suspected disgnoses were tuberculous inflammatory edema of the fundus (Manschot 1959), multiple sclerosis (Cogan and Wray (1975), cerebral vasculitis (Reichmann et al. 1992), vasculitis with Raynaud´s phenomenon (Huston et al. 1978), polyarteritis nodosa (Leonhardt and Kullenberg 1977), or temporal arteritis Horton (Scala et al. 1986).

Mahar et al. (1979) found a primary cardiac *myxosarcoma* in a 30-month-old child who showed right hemiparesis with aphasia and right *homonymous hemianopia*.

Transesophageal echocardiography in particular is a useful tool for detecting cardiac tumors early. However, some tumors are clinically silent and do not show any signs or symptoms during the early growth phase. Early recognition of cardiac signs and symptoms is necessary. Pinede et al. (2001) reported on 112 patients with left atrial myxomas. They divided the data into two groups according to the pre- and post-echocardiographic periods:

1. between 1959 and 1977, when the only diagnostic methods were angiocardiography and cardiac catheterization. 14 patients (12.5%) underwent surgery.
2. since 1977 with 2-dimensional echocardiographic use (transthoracic and transesophageal), about five patients per year had undergone surgery (98 patients, 87,5%).

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Address for correspondence:
Dieter Schmidt, M.D.
Professor of Ophthalmology.
Univ.-Augenklinik
Killianstr. 5
D-79106 Freiburg, Germany
Fax: 761-270-4075
Phone: 761-270-4001
E-mail: Dieter.Schmidt@uniklinik-freiburg.de