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# Transthoracic Echocardiography and Carotid Doppler Ultrasound for Detection of Cardiac and Carotid Artery Disease in Patients with Acute Retinal Artery Obstruction

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**We reviewed the medical records of patients with acute retinal artery obstruction (RAO) and evaluated the importance of transthoracic echocardiography (TTE) and carotid Doppler ultrasound in determining causes of cardiac and carotid artery origin in RAO. A retrospective case study conducted in the Department of Ophthalmology, Inha University Hospital, Korea comprised 26 patients presenting with acute RAO who underwent systemic evaluation, TTE and carotid Doppler ultrasound between June 1, 1997 and December 31, 2003. Among these 26 patients, abnormal cardiac findings were detected in 12 (46%) and abnormal carotid findings in 4 (15%). Furthermore, other risk factors for RAO were found in 2 (8%) and stroke broke out within 7 months after experiencing RAO in 4 (15%) of the 26 patients. In patients with acute RAO, TTE and carotid Doppler ultrasound play an important role in pinpointing the origins of retinal emboli. It is thought that TTE and carotid Doppler ultrasound may be essential examinations for determining the underlying cause, planning treatment strategies, and preventing stroke and death.**

**Key words:** acute retinal artery obstruction, branched retinal artery obstruction, carotid Doppler ultrasound, central retinal artery obstruction, transthoracic echocardiography

## INTRODUCTION

Acute retinal artery obstruction (RAO) presents with sudden and profound visual loss, which may be irreversible. Acute RAO is an important clinical event because an RAO is often a precursor of recurrent ischemic events involving the retina or brain. A workup is usually initiated when patients present with symptoms of retinal ischemia or have evidence of asymptomatic embolism, and imaging studies of the carotid arteries and a through systemic evaluation are required.[1] Patients with acute RAO often have one or more systemic or ocular possible causes.[2] Cardiac valvular, structural disease and carotid artery disease are known causes of acute RAO.[3] Transthoracic echocardiography (TTE) is commonly used to detect cardiac sources of embolism. In addition, Doppler ultrasound is used to detect possible flow changes in the carotid arteries and sources of embolism.[4] We evaluated the systemic risk factors and diagnostic yield and importance of TTE and carotid Doppler ultrasound in detecting possible sources of emboli in patients with RAO.

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## PATIENTS AND METHODS

We retrospectively reviewed the charts of 26 consecutive patients with acute RAO who visited the Department of Ophthalmology, Inha University Hospital, Korea between June 1, 1997 and August 31, 2003. The medical records were reviewed for current findings on electrocardiography, coagulation status, blood chemistry, and for medical history. A diagnosis of central RAO (CRAO) was made if the patient had experienced a sudden, painless loss of vision in one eye associated with the typical ophthalmoscopic findings of CRAO including a pale retina, narrowing of the retinal arteries, and a macular cherry red spot. A patient was diagnosed with branched RAO (BRAO) if the territory of a branch retinal artery appeared pale on ophthalmoscopic examination.

TTE was performed with a Philips Sonos (MCMD02AA). In all cases, the cardiologist performing and interpreting the echocardiogram was aware of the patient's ophthalmological diagnosis. Each patient was thoroughly evaluated for the presence of cardiac thrombi, valvular lesions, structural lesions, and other cardiac tumors. Doppler ultra-

**Table 1.** Patient characteristics

| Sex | Age | Type of RAO | HT | DM | TO | Cardiac history | Other history | Anticoagulant | Abnormal findings | | | Stroke | F/U (month) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | Hematologic | TTE | Doppler | | |
| M | 52 | CRAO | + | - | + | - | - | - | - | - | - | - | 20 |
| M | 63 | CRAO | - | + | + | MI, PTCA with stent | DVT | + | - | + | - | - | 46 |
| F | 57 | CRAO | + | - | - | - | - | - | - | - | - | + | 28 |
| M | 44 | CRAO | - | - | + | - | - | - | - | - | - | - | 17 |
| F | 65 | CRAO | - | - | - | - | - | - | - | - | + | - | 18 |
| F | 62 | CRAO | - | + | - | - | - | - | - | - | - | - | 24 |
| M | 62 | CRAO | - | - | - | - | - | - | - | - | - | - | 12 |
| M | 55 | CRAO | - | + | + | - | - | - | - | + | + | - | 18 |
| M | 55 | CRAO | - | + | - | MI, CABG | - | + | - | + | - | - | 17 |
| F | 63 | CRAO | - | - | + | - | Trauma | - | - | - | - | - | 26 |
| M | 60 | CRAO | - | - | + | - | - | - | - | - | - | + | 33 |
| M | 69 | CRAO | + | - | + | - | - | - | - | + | + | - | 48 |
| F | 84 | CRAO | + | - | - | - | - | - | - | + | - | - | 25 |
| M | 81 | CRAO | - | - | + | - | - | - | - | - | - | - | 8 |
| F | 28 | CRAO | - | - | - | SBE | - | - | - | + | - | - | 17 |
| M | 45 | CRAO | - | - | + | - | - | - | - | - | - | - | 27 |
| F | 71 | CRAO | - | + | - | - | - | - | - | + | - | - | 31 |
| M | 42 | CRAO | - | - | + | Prosthetic-MV | - | + | - | + | - | - | 25 |
| F | 39 | CRAO | + | - | - | - | - | - | - | - | - | - | 19 |
| F | 52 | BRAO | + | - | - | - | - | - | - | - | - | - | 25 |
| M | 73 | BRAO | + | - | + | - | - | - | - | + | - | - | 14 |
| F | 49 | BRAO | - | - | - | - | - | - | - | + | - | - | 7 |
| F | 20 | BRAO | - | - | - | - | SLE | - | + | - | - | - | 24 |
| F | 57 | BRAO | + | - | - | - | - | - | - | - | - | + | 31 |
| F | 74 | BRAO | + | - | + | MI, PTCA with stent | - | + | - | + | - | - | 23 |
| F | 65 | BRAO | + | - | - | - | - | - | - | + | + | + | 32 |

*TO: tobacco, MI: myocardial infarction, PTCA: percutaneous transluminal coronary angiography, DVT: deep vein thrombosis, CABG: coronary artery bypass graft, SBE: subacute bacterial endocarditis, Prosthetic MV: prosthetic mitral valve, SLE: systemic lupus erythematosus.*

**Table 2.** Results of transthoracic echocardiography

| Findings | No. of case |
|---|---|
| AR | 3 |
| AS (Non-rheumatic) | 1 |
| MAC | 1 |
| MR | 5 |
| MS (Rheumatic) | 1 |
| Prosthetic mitral valve | 1 |
| LV dysfunction * | 3 |
| Intracardiac thrombus | 1 |
| Pericardial effusion | 1 |
| Total | 17 |

*AR: aortic valve regurgitation, AS: aortic valve stenosis, MAC: mitral annulus calcification, MR: mitral valve regurgitation, MS: mitral stenosis. *LV dysfunction: left ventricular ejection fraction < 50%.*

sound was performed with a Diasonics Doppler ultrasonic instrument (VST masters series). The examination included the determination of blood flow direction and velocity, together with waveform analysis in the common, internal, and external carotid arteries. Carotid artery stenosis of more than 50% is considered severe.

## RESULTS

Patient characteristics are summarized in Table 1.

There were 12 males and 14 females, with a mean age of 57.0 $\pm$ 15.8 years (range, 28-84). Nineteen of the 26 patients with acute RAO had CRAO and 7 patients had BRAO.

According to clinical history of the 26 patients, 5 (19%) had known cardiac diseases, 3 (12%) had ischemic heart disease (2 had undergone percutaneous transluminal coronary angiography with stent insertion, and 1 had undergone coronary artery bypass surgery), and 2 (8%) had a valvular heart disease (1 had undergone valve replacement and 1 had a history of subacute bacterial endocarditis). Coagulopathies, including thrombin time, prothrombin time, partial thromboplastin time, and thrombocyte count, were found to be normal except for 1 patient. Lupus anticoagulant was positive in 1 patient with systemic lupus erythematosus. One patient had a history of head trauma and inferior orbital wall fracture, and underwent surgical reconstruction.

**Table 3.** Results of carotid doppler ultrasound

| Findings | No. of case |
|---|---|
| Both CCA stenosis | 1 |
| Both CCA, ICA stenosis | 1 |
| Ipsilateral CCA stenosis | 1 |
| Ipsilateral ICA stenosis* | 1 |
| Total | 4 (15.4%) |

*CCA: Common carotid artery, ICA: Internal carotid artery. *: Non-significant stenosis: carotid artery stenosis < 50%.*

**Table 4.** Prevalence of stroke in 26 retinal artery obstruction patients.

| Stroke | No. of event |
|---|---|
| MCA infarction | 2 |
| Pons, basal ganglia infarction | 1 |
| Cbll, pons, thalamus infarction | 1 |
| Total | 4 (15.4%) |

*MCA: middle cerebral artery, Cbll: cerebellum.*

The results of TTE are summarized in Table 2.

Abnormal findings were detected by TTE in 12 of the 26 patients (46%), and in 5 of these 12 (19%) there were multiple potential cardiac sources of retinal emboli.

The results of carotid Doppler ultrasound are summarized in Table 3.

Carotid artery stenosis was detected by carotid Doppler ultrasound in 4 of the 26 patients (15%), and was more than 50%, with multiple calcified and atheromatous plaques, in 3 (12%) of these 4. In 4 patients (15%), stroke broke out after experiencing RAO during a minimum period of follow up of 7 months (Table 4). Two of these stroke patients suffered from middle cerebral artery infarction and pons, basal ganglia infarction within 4 months after experiencing RAO. One patient suffered from cerebellum, pons, and thalamus infarction within 2 months, and the other patient suffered middle cerebral artery infarction within 4 months after experiencing RAO.

## DISCUSSION

Acute RAO is a condition that is associated with a high prevalence of systemic disease. Recent studies have demonstrated that significant numbers of patients with acute RAO have both hemodynamically significant carotid artery stenosis and cardiac abnormalities that necessitate systemic therapy. Acute RAO is uncommon in young adults. Since most patients are more than 60 years old, it has been presumed that in most cases acute RAO is the consequence of thrombosis and embolism.[5,6] The following cardiac diseases have been associated with acute RAO: aortic and mitral valve disease,[6,7,8] atrial myxoma,[9] valvulotomy,[10] angioplasty,[11] acute myocardial infarction,[12] subacute bacterial endocarditis,[8] intravenous drug-related talc embolization,[13] prosthetic valves,[14] and mitral valve prolapse.[15,16] Various retrospective studies have noted an approximately 25% prevalence of cardiac valvular disease associated with acute RAO.[7,8]

Atherosclerotic lesions in the carotid artery are often seen in patients with RAO.[17,18,19,20,21] Evidence of a possible link between acute RAO and pathological changes in carotid arteries has been incomplete, because the examinations of the carotid arteries, whether invasive or non-invasive, have not been performed systemically in acute RAO.[4]

Accordingly, TTE should be performed in all young patients with occlusive disease of the retinal arteries, and in older patients with any of the cardioembolic risk factors.[22,23] Doppler studies of the carotid arteries are recommended in all cases of RAO, as there is a reported 19% incidence of hemodynamically significant carotid artery stenosis among these patients.[24]

Because there are numerous abnormalities detected on TTE for which no intervention is required, the important result that should be monitored is anticoagulation or cardiac surgical intervention based on the echocardiographic abnormalities. Although a retrospective study of patients with acute RAO based on TTE testing found abnormal studies in 47% of patients, in only 10% were the findings such that anticoagulation or surgical intervention was initiated.[22] In another study, a cardiac source of embolism was detected in 38% of patients acute RAO and 10% had both cardiac and carotid diseases.[8]

In our study, we analyzed the TTE and carotid Doppler ultrasound data of patients with acute RAO, as well as the decision to anticoagulate or perform surgical intervention. The most prevalent possible embolic source was cardiac abnormalities in our study (n=12, 46%), as detected by TTE. Doppler study of the carotid arteries revealed significant carotid stenosis (>50%) in 3 of the 26 patients examined (12%). Four of the 26 patients (15%) with acute RAO had TTE and carotid Doppler ultrasound findings that indicated anticoagulation or surgical intervention. The diagnoses of intracardiac thrombus and carotid artery stenosis of more than 50% are indications for anticoagulation therapy.

It should also be noted that the variable outcome of our study was based on the results of TTE rather than of transesophageal echocardiography (TEE). There have been recent reports of the detection of cardiac abnormalities by TEE that were undetected by TTE.[25,26] These include mitral valvular disease and other valvular calcifications, enlarged left atrium, and severe left ventricular dysfunction. Aortic valve calcification and mitral annular calcification indicate diffuse atherosclerotic disease, for which a more thorough investigation of the aorta by TEE is warranted.[27] In this study, TEE data were not evaluated because we had not performed TEE routinely in our center. Although the role of echocardiography in the evaluation of patients with brain ischemia has received extensive attention[28,29] and TEE has been found to be cost-effective in some investigations,[30] its importance in retinal vascular conditions is not as well studied.1 Furthermore, the disadvantages of TEE are technical considerations, inconvenience and possible rare complications, such as respiratory arrest related to sedation, arrhythmias, aggravation of heart failure, and traumatic injury to the esophagus.[31] These are the reasons for the recommendation to first perform TTE, which is a noninvasive and simple procedure, followed by TEE in cases with findings that may suggest cardiac or aortic pathologic condition.

The most common causes of death in patients who have suffered acute RAO is cardiovascular disease.[5,32] The patients with visible retinal emboli do have a significantly shorter life expectancy.[32,33]

Furthermore, RAO patients with ischemic stroke or cerebral transient ischemic attacks are thought to have similar risk factors and underlying vascular disease.[34] Therefore, patients with these ischemic events often undergo similar investigations, and the strategies used for secondary prevention of ischemic events are broadly similar.[34]

De Potter and Zografos reported that of 152 RAO patients, 26% died during a mean follow up of 9.7 years.[33] Savino and associates reported that the survival of 86 RAO patients was lower than that expected of an age-and sex-matched population.[32] In this study, 4 of the 26 (15%) patients suffered ischemic stroke after acute RAO, during a mean follow up of 2 years. Although our study was limited by the lack of a control population and the fact that only a subset of patients received TTE and carotid Doppler ultrasound studies, this incidence rate of stroke seems to be higher than that of the control population.

In conclusion, the findings in our study demonstrated the value of TTE and carotid Doppler ultrasound to detect potential sources for an unexplained artery embolism causing an acute RAO, to plan treatment strategies, and to help prevent stroke and death. We recommend both TTE and carotid Doppler ultrasound as essential examinations in all patients with acute RAO.

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