Multiple aneurysms of the anterior communicating artery

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Multiple aneurysms of the anterior communicating artery are rare. The authors describe a patient who was admitted with a sudden, severe headache with radiation to the neck. Angiograms demonstrated filling of both anterior cerebral arteries and bilateral aneurysms arising from the origin of the anterior communicating artery. Both aneurysms were surgically verified and clipped.

(Key words: Anterior communicating artery, angiography, cerebral aneurysm)

Multiple cerebral aneurysms occur in approximately 20% of patients with cerebral aneurysms, but multiple aneurysms of the same artery are much less common.^{1,2} We present the angiographic findings in a rare case of multiple aneurysms of the anterior communicating artery.

Report of case

A 46-year-old right-handed woman suddenly had a severe headache with radiation to the neck. She did not lose consciousness but vomited once. On initial examination, her blood pressure was 170/110 mm Hg. She was sleepy but arousable and had slight nuchal rigidity. The patient was neurologi-

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cally intact and oriented with the exception of place. A computed tomographic scan of the head, performed shortly after the onset of symptoms, demonstrated blood in the anterior interhemispheric and sylvian fissures bilaterally.

Left internal carotid angiography demonstrated filling of both anterior cerebral arteries and aneurysms arising from the origin of the anterior communicating artery (ACoA) bilaterally. A 5 × 4-mm aneurysm from the ACoA origin on the left and a second aneurysm, measuring 5 × 3 mm, on the right, were seen (Figure). An angiogram of the right internal carotid artery revealed a hypoplastic horizontal (A1) segment of the right anterior cerebral artery. Findings of left vertebral angiography were normal.

Four days after admission, the patient had right-sided weakness with increased lethargy. She gradually improved until 16 days after admission. She then had a recurrent intracranial hemorrhage. The aneurysms were verified surgically as aneurysms of bilateral ACoA origin and were successfully clipped.

Discussion

The ACoAs account for 23% to 35% of all cerebral aneurysms and 90% of all anterior cerebral artery aneurysms.^{3,4} Previous studies have demonstrated that in patients with aneurysms 17% had multiple aneurysms and 2.8% had multiple aneurysms of the same artery, with involvement of the internal carotid artery being the most common.⁵

Multiple aneurysms of the same artery have previously been noted to have an association with familial aneurysms and cerebral arteriovenous malformations.¹ Anomalies of the horizontal segment of the anterior cerebral artery, including hypoplasia of the A1 segment, have been noted to show a slightly increased incidence of ACoA aneurysms.⁶

In our literature review, only eight cases of bilateral ACoA aneurysms were found. In one report, only one aneurysm was apparent angi-

Figure. Left internal carotid angiogram. Posterior-anterior (Left) and oblique (Right) views demonstrate two aneurysms arising from the region of the anterior communicating artery. They were surgically proved to be aneurysms of bilateral anterior-communicating artery origin.

ographically while the second aneurysm was identified at surgery.⁵ The remaining seven cases were identified either angiographically or at autopsy.² They were contained within a study of 2,951 patients with proved aneurysms. The method of detection for each individual case was not specifically stated, and no such cases were illustrated angiographically. To our knowledge, the case reported here is the first case of bilateral ACoA aneurysms demonstrated angiographically to be described in the medical literature.

Typically, in patients with multiple aneurysms, the larger aneurysm is the most likely to rupture.^{4,5} The aneurysms in our patient were nearly the same size, and no determination was made as to which aneurysm had bled. There were no clues (that is, spasm, mass ef-

fect, or irregularity of the aneurysm) to identify which aneurysm had ruptured.

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BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see **Warnings**), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in

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patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°, 2°, 3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes, reversible non-obstructive paralytic ileus. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecostasia, galactorrhea/hyperprolactinemia, increased urination, spotty menstruation, impotence.

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[†] Constipation, which is easily managed in most patients, is the most commonly reported side effect of Calan SR.

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