



Clinical Research

The Impact of Biochemical Markers on Major Adverse Cardiovascular Events and Contralateral Carotid Artery Stenosis Progression Following Carotid Interventions

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Background: To determine if elevated preintervention high-sensitivity C-reactive protein (hsCRP) and B-type natriuretic peptide (BNP) levels associate with major adverse cardiovascular events (MACE) or disease progression after carotid revascularization.

Methods: We retrospectively examined patients receiving elective carotid endarterectomy (CEA) or carotid artery stenting (CAS) at our institution from 2007 to 2014. All included patients had preintervention hsCRP and BNP levels. Examined outcomes of interest included contralateral carotid disease progression (increased stenosis or need for revascularization) and MACE (composite of death, stroke, myocardial infarction, need for coronary artery bypass graft or percutaneous coronary intervention) at 3 years after procedure. The relationship between baseline hsCRP and BNP levels and time to event was examined by univariate and multivariate Cox proportional hazard regression analyses.

Results: A total of 248 patients were included in the analysis (mean age: 68 ± 10 years), with 14% receiving CAS and 86% CEA. A total of 61 patients (25%) had 1 or more MACE by 3 years. Elevated hsCRP (>3 mg/L) trended toward associating with MACE but failed to reach significance (hazard ratio [HR]: 1.6 [1.0–2.7], $P = 0.07$). Multivariate analysis found that elevated BNP (>100 pg/mL, HR: 2.2 [1.3–3.7], $P = 0.002$) and diabetes mellitus (HR: 1.9 [1.2–3.2], $P = 0.01$) predicted MACE. Having elevated preprocedural levels of both hsCRP and BNP significantly increased patients' likelihood of experiencing MACE (HR: 3.4 [1.6–7.1], $P = 0.001$). About 175 patients received contralateral carotid imaging postprocedure and of those patients, 31 (18%) experienced stenosis progression and/or revascularization within 3 years. However, neither elevated hsCRP (HR: 1.2 [0.6–2.3], $P = 0.68$) nor BNP (HR: 1.1 [0.5–2.5], $P = 0.88$) associated with disease progression.

Conclusions: BNP elevation at the time of carotid intervention is associated with MACE in long-term follow-up. hsCRP does not appear to correlate with either disease progression of the contralateral artery or MACE.

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INTRODUCTION

Carotid artery occlusive disease accounts for a significant portion of vascular specialist care. Fundamental evidence suggests inflammation as a culprit involved with atherosclerotic lesion pathogenesis. The inflammation gradation as demonstrated by serum biochemical markers likely correlates with disease severity and progressive plaque morphology. The Inflammation and Carotid Artery—Risk for Atherosclerosis Study showed a temporal correlation between inflammatory biomarkers and worsening atherosclerosis of the carotid artery.¹ Others have demonstrated via prospective evaluation of carotid stenosis by duplex surveillance, a correlation between serum c-reactive protein (CRP) and stenosis progression.²

Cardiovascular morbidity accounts for the lion share of major adverse events following carotid interventions. In addition to inflammation contributing to progressive stenosis, it likely also contributes to the risk of cardiac-related events. Another biomarker linked to cardiovascular events after noncardiac surgery is B-type natriuretic peptide (BNP).³ Previously, we have reported on the association of combined preoperative biochemical markers on future major adverse cardiovascular events (MACE) in patients undergoing lower extremity endovascular revascularization.⁴

This study sought to evaluate the association of contralateral carotid artery stenosis progression and risk of future cardiovascular morbidity after either surgical or endovascular revascularization with respect to the baseline serologic markers, high-sensitivity CRP (hsCRP), and BNP.

METHODS

We retrospectively examined patients who received elective carotid revascularization by a single operator at Charleston Area Medical Center (CAMC), Charleston, WV, between January 1, 2007 and December 31, 2014. We then compared preprocedure hsCRP and BNP levels to the patients' postoperative course. Patients were identified using procedure codes for carotid endarterectomy (CEA; 38.12) and carotid artery stenting (CAS; 00.61, 00.63). Inclusion criteria included the presence of preintervention hsCRP and BNP levels obtained within 30 days before the procedure, elective CEA, angioplasty, or stenting of the internal carotid artery performed by the primary author (Patrick A. Stone), and at least one postoperative follow-up carotid duplex or angiogram. Exclusion criteria included patients younger than 18 years,

emergency revascularization, lack of hsCRP or BNP levels before procedure, and lack of postoperative carotid duplex or angiogram. All aspects of the study were reviewed and approved by the CAMC/West Virginia University, Charleston Division, Institutional Review Board.

Our primary outcome was major adverse cardiovascular events (MACE) within 3 years of procedure. MACE was the composite endpoint comprised all-cause death, myocardial infarction (MI), stroke, and need for coronary artery bypass graft (CABG) or percutaneous coronary intervention (PCI). All-cause mortality was determined via hospital records, Social Security Death Index, and online obituary search engines. Follow-up for MACE consisted from the time of procedure until the occurrence of the initial MACE or the most recent admission/visit at CAMC or an affiliated clinic/office (mean follow-up: 33 ± 25 months).

A secondary outcome of interest was carotid disease progression contralateral to the index CAS or CEA. To be included in the analysis of this outcome, patients had to received 2 or more duplexes or undergo revascularization of the internal carotid artery contralateral to the index procedure. Carotid stenosis was stratified into the following categories: no stenosis (0–15%), mild stenosis (16–49%), moderate stenosis (50–79%), severe stenosis (80–99%), and occlusion (100%).⁵ In agreement with previous studies,^{6–8} we defined carotid disease progression as an increase in internal carotid artery (ICA) stenosis of 50% or greater for ICAs with a baseline lesion less than 50% or an increase to a higher category of stenosis if the baseline lesion was 50% or greater. Therefore, the transition from no stenosis (0–15%) to mild stenosis (16–49%) was not deemed as progression, whereas all other increases in severity classification were considered as disease progression. Contralateral carotid disease progression was additionally defined as increase of ≥ 100 cm/sec in peak systolic velocity or by the patient receiving revascularization (CEA or CAS) in the contralateral ICA. Follow-up for contralateral carotid disease progression continued from the time of the procedure until event or most recent angiography or duplex, (mean follow-up: 32 ± 22 months).

Collected demographic information included age, gender, race, BMI, and current tobacco use. Comorbidity information included obesity (BMI > 30), diabetes mellitus, hypertension, hyperlipidemia, coronary artery disease, chronic kidney disease, and heart failure. Recent events or conditions, with recent defined as thirty days or less before procedure, that can induce a systemic inflammation response were examined and included trauma or

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