IS CAROTID ULTRASOUND NECESSARY IN THE CLINICAL EVALUATION OF THE ASYMPTOMATIC HOLLENHORST PLAQUE? (AN AMERICAN OPHTHALMOLOGICAL SOCIETY THESIS)

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ABSTRACT

Purpose: To evaluate the utility of carotid ultrasound in patients with asymptomatic Hollenhorst plaques.

Methods: Retrospective chart review of 237 patients diagnosed with Hollenhorst plaques between 1996 and 2004. The baseline cardiovascular risk profile, medications, and carotid ultrasound findings were documented. Retinal ischemia, myocardial ischemia, and cerebrovascular events during follow-up were noted.

Results: There was no statistically significant difference in the proportion of patients with carotid stenosis >40% between symptomatic (n=60) and asymptomatic (n=177) patients (32.7% vs 22.7%; *P*=.192, one-way ANOVA). However, symptomatic patients were statistically more likely to have stenosis >69% (25% compared with 9.2% in the asymptomatic group; *P*=.008, one-way ANOVA).

Among asymptomatic patients, those with carotid bruit (27.1%) were more likely to have moderate carotid stenosis >40% (55.6% vs 18.6% in patients without bruit; *P*=.0008, one-way ANOVA) and significant stenosis >69% (37% vs 4.3% in patients without bruit; *P*=.0001, one-way ANOVA). Follow-up data was obtained from 32 symptomatic patients (39.6 ± 22.9 months) and 100 asymptomatic patients (41.3 ± 21.8 months). Vascular and neurologic event rates were similar between the two groups.

Conclusions: Hollenhorst plaques are a marker of significant carotid disease irrespective of retinal symptoms. Carotid auscultation remains important in the examination of patients with Hollenhorst plaques and increases the yield of asymptomatic patients diagnosed with carotid stenosis. The presence of visual symptoms on presentation did not correlate with an increased risk of death or stroke compared to asymptomatic patients during follow-up. Therefore all patients with asymptomatic plaques should have a medical workup, including carotid ultrasonography.

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INTRODUCTION

Hollenhorst plaques are cholesterol emboli deposited within retinal arterioles. These were first described by Dr Robert Hollenhorst in 1961 in 31 patients with known occlusive carotid or vertebral artery disease.[1] Multiple studies have since been performed to evaluate the relationship between Hollenhorst plaques and surgically correctable carotid disease.[2-8]

The literature on patients with Hollenhorst plaques and symptoms of retinal ischemia is established. Earlier studies utilized carotid angiography to evaluate the carotid artery of patients with symptomatic Hollenhorst plaques. In 1979, Wilson and colleagues[2] studied 103 patients with permanent visual field defects secondary to cholesterol emboli. Up to 55% of the patients were found to have operable carotid stenosis. These findings were confirmed by Chawluk and colleagues[4] in 1988 by ultrasound evaluation of 105 patients with symptoms of retinal ischemia. Forty percent of the 10 patients with Hollenhorst plaques were found to have carotid stenosis greater than 60%. Chawluk and colleagues found an association between Hollenhorst plaques and a stenotic or ulcerated carotid plaque (*P*=.04).

There is a paucity of literature on the utility of carotid ultrasonography and the workup and outcomes of patients with asymptomatic Hollenhorst plaques. No consensus or established guidelines exist for the workup of these patients. Bunt[3] evaluated 60 patients referred after ocular examination or visual complaints for cerebral angiography. Of the 60 patients, 18 had asymptomatic Hollenhorst plaques; 9 of those (50%) had carotid disease and 5 of those patients required carotid endarterectomy. None of the patients with Hollenhorst plaques developed cerebral symptoms. Of the 26 patients with amaurosis fugax in that study, 70% had carotid disease and 50% required carotid endarterectomy. Follow-up fundus examinations indicated that the Hollenhorst plaque persisted in 5 of 9 patients. Bunt concluded that an incidental Hollenhorst plaque could be a marker of a past embolic event and that the asymptomatic Hollenhorst plaque was a poor predictor of future embolic events.

McCullough and colleagues[7] reviewed the records of 105 patients referred for a carotid ultrasound to evaluate ocular manifestations. Of those, 11 patients, some of whom were asymptomatic, had been referred for evaluation of Hollenhorst plaques. The rate of significant carotid disease >60% was 18.2%. The investigators concluded that Hollenhorst plaques had moderate predictive value for significant carotid disease. Bull and colleagues[5] utilized duplex ultrasound and found that 9% of 23 patients with asymptomatic Hollenhorst plaques had significant carotid stenosis. They concluded that the presence of asymptomatic Hollenhorst plaques appears not to have a significant association with carotid disease and may not necessarily require routine screening unless other risk factors for carotid stenosis are present.

Wakefield and colleagues[6] retrospectively reviewed the records of 3560 patients that presented to the vascular clinic for ultrasound between 1996 and 2002 and found 18 patients with asymptomatic Hollenhorst plaques. The rate of significant stenosis (>60%) found on ultrasound was 5.6% (2 of 36 carotid arteries). They concluded that carotid ultrasound should be performed in this patient population, as a small percentage will have surgically correctable disease, but that if results were normal, follow-up ultrasound was

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not required. This study included only 18 patients with asymptomatic plaques, and those were from patients referred for an ultrasound. Our study is different in that it is larger, is more comprehensive, and looks at all patients diagnosed with an asymptomatic Hollenhorst plaque by the ophthalmology department, regardless of whether or not they were referred for a carotid ultrasound.

In reviewing this literature, it appears that the relationship between Hollenhorst plaques and carotid disease has been established but the degree of this relationship has been the matter of controversy. A report on physicians' response to a survey regarding the management of patients referred to them for a medical workup of a Hollenhorst plaque detected during a routine eye examination demonstrated a wide variability in patient management. This indicates that more research is needed to understand the utility of testing in order to help establish guidelines, particularly for asymptomatic patients.[9] The lack of consensus on the utility of ultrasound in evaluating patients with asymptomatic Hollenhorst plaques is the result of multiple factors. The largest number of patients with asymptomatic Hollenhorst plaques studied thus far was 23.[5] Dunlap and colleagues[10] studied 50 asymptomatic patients with retinal artery occlusion and Hollenhorst plaque but did not separate these into distinctive groups. Furthermore, most studies were approached from a radiological viewpoint. Our study looked forward at the workup and fate of those patients with symptomatic vs asymptomatic Hollenhorst plaques.

The guidelines for carotid endarterectomy have changed since the publication of the earlier studies, and they now include recommendations for asymptomatic patients. For many years, vascular surgeons performed carotid endarterectomy on patients with an asymptomatic Hollenhorst plaque.[11] Later literature recommended against this practice, as it showed that carotid endarterectomy did not appear to affect the incidence of late cerebrovascular events.[12] It is important to diagnose the stenosis on ultrasound to enable classification of stenosis and to determine optimal medical or surgical management. Carotid endarterectomy is recommended for symptomatic patients with moderate to severe stenosis (≥50%).[1] In the North American Symptomatic Carotid Endarterectomy Trial (NASCET),[14,15] symptomatic patients with 70% to 99% carotid stenosis had a cumulative risk of ipsilateral stroke at 2 years of 9% compared with 26% for those treated medically. The European Carotid Surgery Trial[16] similarly supported endarterectomy in this group of patients. Among symptomatic patients in the NASCET study, with stenosis 50% to 69%, the 5-year rate of ipsilateral stroke was 15.7% in those who had an endarterectomy vs 22.2% in those treated medically.[15] In asymptomatic patients with moderate to severe stenosis (>60%), endarterectomy is recommended if perioperative risk is low.[10] Meta-analysis that pooled results from three randomized clinical trials[17] included 5223 patients with asymptomatic moderate to severe stenosis (≥50% in the Veteran Affairs Cooperative Study[18] and ≥60% in the Asymptomatic Carotid Atherosclerosis Study[19]and the Asymptomatic Carotid Surgery Trial).[20] The relative risk of perioperative or subsequent stroke or mortality was 0.69, favoring endarterectomy.

With evolving guidelines for the management of carotid stenosis, and with improved medical treatments, it is important to identify those patients and initiate early referral so that they can be managed appropriately. We describe the significance of clinical predictors such as carotid bruits as markers for significant carotid disease in patients with Hollenhorst plaques, and examine the utility of carotid ultrasonography in these patients.

The hypotheses are as follows: (1) there is a difference in the baseline characteristics of patients with symptomatic vs asymptomatic Hollenhorst plaques, (2) there is a difference in the relationship between Hollenhorst plaques and carotid disease in symptomatic vs asymptomatic patients, and (3) there is a difference in the rates of vascular and neurologic events that occurred during follow-up in patients with symptomatic vs asymptomatic Hollenhorst plaques.

METHODS

After Institutional Review Board approval was obtained, all patients diagnosed with Hollenhorst plaques at Mayo Clinic, Rochester, Minnesota, between January 1, 1996, and December 31, 2004, were identified. Medical records on each patient identified were obtained from the entire hospital and clinic medical records system. All diagnoses had been made on clinical fundus examination by ophthalmologists. Medical records of all patients with a diagnosis of Hollenhorst plaque, retinal vascular occlusion, retinal artery occlusion, and retinal embolism (International Classification of Diseases codes 362.30 to 362.33) were retrospectively reviewed to find those patients who had a documented Hollenhorst plaque. Medical records of the 237 patients identified to have a Hollenhorst plaque were carefully reviewed. The following demographic data were extracted from the charts: age, gender, race, body mass index, and data pertaining to cardiovascular risk factors such as hypertension, diabetes, tobacco use, hyperlipidemia, coronary artery disease, cerebrovascular disease, and peripheral vascular disease, including a history of known carotid disease. Patients who had known carotid stenosis as documented by previous ultrasound were grouped into one of two categories, >40% stenosis or >69% stenosis, according to the criteria that were reported by our vascular radiology department. Information on whether the patients underwent carotid auscultation by a physician during their initial evaluation and the results of that evaluation were specifically sought and documented. The medication list on presentation was reviewed. It was noted whether Hollenhorst plaques were discovered coincidentally in asymptomatic patients undergoing an eye examination for other reasons. In addition, patients whose Hollenhorst plaque was noted as part of a workup for visual complaints consistent with retinal ischemia were identified. These include temporary and permanent monocular visual loss and amaurosis fugax. Results of a lipid panel and fasting glucose were documented when obtained at diagnosis or within a year prior to diagnosis. Results of carotid duplex ultrasound were documented, when performed. Follow-up data was obtained from yearly patient-filled clinical surveys and clinical notes. Cause of death was obtained from physician-completed reports. Visual field defects were diagnosed by an ophthalmologist.

All continuous data were reported in mean ± SD with a confidence interval of 95%. The Fisher exact test was utilized to compare baseline characteristics and outcomes of patients in both groups. Continuous data in both groups were compared using the one-way

analysis of variance test. *P* values ≤.05 were considered significant for all statistical analyses. All data was analyzed using JMP software (version 6.0 for Windows; SAS Institute Inc, Cary, North Carolina).

RESULTS

The diagnosis of Hollenhorst plaque was made in 237 patients between 1996 and 2004. Of these 237 patients, 60 patients had symptoms (25.3%) and 177 patients (74.7%) were asymptomatic at the time of diagnosis.

The baseline characteristics of symptomatic and asymptomatic patients with Hollenhorst plaques are listed in Table 1. There was a statistically greater number of females (39.0% vs 21.7%, *P*=.018) and a greater number of patients currently taking aspirin (62.7% vs 43.3%, *P*=.01) in the asymptomatic group vs the symptomatic group. In both groups, there was a similar rate of hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease, cerebrovascular disease, peripheral vascular disease, and preexisting carotid disease. The use of clopidogrel, warfarin, beta blockers, statins, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers was similar.

TABLE 1. BASELINE CHARACTERISTICS OF SYMPTOMATIC VS ASYMPTOMATIC PATIENTS WITH HOLLENHORST PLAQUES

BASELINE CHARACTERISTICS	SYMPTOMATIC (N=60)	ASYMPTOMATIC (N=177)	*P* VALUE (FISHER EXACT TEST)
Age (years)	70.7 ± 9.5	70.9 ± 9.8	.810
Gender: female/male (%)	21.7 / 78.3	39.0 / 61.0	.018*
BMI (kg/m^2)	29.5 ± 5.0	28.7 ± 7.8	.359
Hypertension (%)	56.7	69.5	.083
Diabetes mellitus (%)	18.3	26.6	.227
Hyperlipidemia (%)	58.3	55.9	.766
Current tobacco use (%)	18.3	14.1	.414
Any tobacco use (%)	74.6	61.6	.084
Coronary artery disease (%)	53.3	40.1	.097
Cerebrovascular disease (%)	23.3	23.7	1.000
Peripheral vascular disease (%)	16.7	19.8	.705
Significant carotid disease (%)	6.7	12.5	.338
Current aspirin use (%)	43.3	62.7	.010*
Current clopidogrel use (%)	1.7	5.1	.459
Current warfarin use (%)	15.0	8.5	.213
Current beta blocker use (%)	33.3	31.6	.873
Current ACE/ARB use (%)	38.3	33.3	.531
Current statin use (%)	33.3	36.7	.756

ACE, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index.
*Statistically significant (*P*<.05).

Carotid auscultation on or prior to diagnosis was reported in 75% of symptomatic patients and 61% of asymptomatic patients (*P*=.04). A carotid bruit was reported in 24.4% of symptomatic patients and 27.1% of asymptomatic patients (*P*=.8) who underwent carotid auscultation. A duplex carotid ultrasound was eventually obtained in 87% of symptomatic patients and 80% of asymptomatic patients (*P*=.3).

The carotid evaluation results are outlined in Table 2. Overall, 81.4% of patients with a Hollenhorst plaque had a carotid ultrasound performed, with no difference between symptomatic and asymptomatic patients. The majority of patients in both groups had atherosclerosis on carotid ultrasound (97.4%). There was no statistically significant difference in the proportion of patients with carotid stenosis of at least 40% (32.7% in the symptomatic and 22.7% in the asymptomatic group; *P*=.192). However, symptomatic patients were statistically more likely to have stenosis >69% (25% in the symptomatic compared with 9.2% in the asymptomatic group; *P*=.008). This difference was not reflected in the rate of endarterectomy (13.3% in the symptomatic compared with 7.9% in the asymptomatic group; *P*=.208). The decision to perform endarterectomy is based on many other factors, such as the patient's overall health and ability to undergo the surgery.

Among the 107 asymptomatic patients who underwent carotid auscultation, 29 patients (27.1%) had a carotid bruit. Baseline characteristics of asymptomatic subjects with and without a carotid bruit are outlined in Table 3. The only statistically significant differences between the two groups were that patients with a carotid bruit were more likely to have a history of cerebrovascular

disease (37.9% in patients with a bruit compared to 14.1% in patients without a bruit; *P*=.01) and more likely to have carotid disease. Among asymptomatic patients, those with a carotid bruit were more likely to have carotid stenosis of >40% (55.6% vs 18.6% in patients without a bruit; *P*=.0008) and significant stenosis >69% (37% vs 4.3% in patients without a bruit; *P*=.0001) (Table 4). During follow-up, 37.9% of the asymptomatic patients with a bruit underwent carotid endarterectomy compared with 2.6% of patients without a carotid bruit (*P*<.001).

TABLE 2. CAROTID ULTRASOUND EVALUATION RESULTS OF SYMPTOMATIC VS ASYMPTOMATIC PATIENTS WITH HOLLENHORST PLAQUES

MANAGEMENT AND WORKUP	ALL PATIENTS (N=237)	SYMPTOMATIC (N=60)	ASYMPTOMATIC (N=177)	*P* VALUE (ONE-WAY ANOVA)
Carotid ultrasound performed (%)	81.4	86.7	79.6	.254
Atherosclerosis on ultrasound (%)	97.4	96.1	97.9	.611
Stenosis >40% on ultrasound (%)	25.2	32.7	22.7	.192
Stenosis >69% on ultrasound (%)	13.2	25.0	9.2	.008*
Carotid endarterectomy (%)	9.3	13.3	7.9	.208

*Statistically significant (*P*<.05).

TABLE 3. BASELINE CHARACTERISTICS OF ASYMPTOMATIC HOLLENHORST PLAQUE PATIENTS WITH AND WITHOUT CAROTID BRUITS

BASELINE CHARACTERISTICS	NO BRUIT (N=78)	WITH BRUIT (N=29)	*P* VALUE (FISHER EXACT TEST)
Age (years)	69.8 ± 10.1	72.7 ± 8.2	.145
Gender: female/male (%)	35.9 / 64.1	44.83 / 55.17	.503
BMI (kg/m2)	29.0 ± 4.6	28.5 ± 4.9	.579
Hypertension (%)	69.2	79.3	.343
Diabetes (%)	26.9	17.4	.447
Hyperlipidemia (%)	55.1	72.4	.124
Current tobacco use (%)	11.5	10.3	1.000
Any tobacco use (%)	57.7	62.1	.826
Coronary artery disease (%)	34.6	41.4	.652
Cerebrovascular disease (%)	14.1	37.9	.014*
Peripheral vascular disease (%)	15.4	24.1	.393
Significant carotid disease (%)	6.4	24.1	.016*
On aspirin (%)	62.8	75.9	.254
On beta blocker (%)	37.2	27.6	.493
On ACE/ARB (%)	30.8	31.0	1.000
On statin (%)	34.6	51.7	.123

ACE, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index.
*Statistically significant (*P*<.05).

Among the symptomatic patients, 45 patients had carotid auscultation reported and subsequent carotid ultrasound. Thirty four patients had a carotid bruit on exam (75.6%) and 11 patients did not have a bruit (24.4%). Again, both groups had similar cardiovascular risk factors at baseline. Severe carotid stenosis (>69%) was found in 35.3% of patients with a carotid bruit compared to 18% of patients without a carotid bruit but this difference was not statistically significant (P=0.18). The rate of carotid endarterectomy was also no different between the two groups (17.6% in patients with a carotid bruit compared to 18.2% in patients without a carotid bruit; P=0.62).

Follow-up data (months ± SD) was obtained from 32 symptomatic patients (39.6 ± 22.9 months) and 100 asymptomatic patients

(41.3 ± 21.8 months) (Table 5). The only difference between the groups at baseline was aspirin use (43.3% in the symptomatic group vs 62.7% in the asymptomatic group; *P*=.01). After the diagnosis of Hollenhorst plaque was made, most patients were taking aspirin (80% of symptomatic, 75% of asymptomatic, *P*=.60). Event rates for visual field defects, transient ischemic attacks, stroke, carotid endarterectomy, myocardial infarction, cardiac death, and all cause mortality were similar between the two groups (Table 5).

TABLE 4. CAROTID ULTRASOUND EVALUATION RESULTS OF ASYMPTOMATIC HOLLENHORST PLAQUE PATIENTS WITH AND WITHOUT CAROTID BRUITS

MANAGEMENT AND WORKUP	NO BRUIT (N=78)	WITH BRUIT (N=29)	*P* VALUE (ONE-WAY ANOVA)
Carotid ultrasound performed (%)	89.7	92.9	1.000
Atherosclerosis on ultrasound (%)	95.7	100.0	.557
Stenosis >40% on ultrasound (%)	18.6	55.6	.0008*
Stenosis >69% on ultrasound (%)	4.3	37.0	.0001*
Carotid endarterectomy (%)	2.6	37.9	<.001*

*Statistically significant (*P*<.05).

TABLE 5. FOLLOW-UP DATA ON VASCULAR EVENTS IN PATIENTS WITH HOLLENHORST PLAQUES

VARIABLE	SYMPTOMATIC (N=32)	ASYMPTOMATIC (N=100)	*P* (FISHER EXACT TEST)
Follow-up (months) ± SD	39.6 ± 22.9	41.3 ± 21.8	.509
Combined visual events % (n)	9.4 (3)	8.0 (8)	.727
Transient ischemic attack (TIA) % (n)	12.5 (4)	7.0 (7)	.461
Stroke % (n)	6.3 (2)	7.0 (7)	1.000
Combined stroke/TIA % (n)	15.6 (5)	13.0 (13)	.769
Carotid endarterectomy % (n)	4.0 (2)	4.3 (7)	1.000
Myocardial infarction % (n)	9.4 (3)	13.0 (13)	.760
Cardiac mortality % (n)	6.3 (2)	8.0 (8)	1.000
All cause mortality % (n)	15.6 (5)	18.0 (18)	1.000
Combined stroke/TIA % (n)	15.6 (5)	13.0 (13)	.769

*Statistically significant (*P*<.05).

DISCUSSION

Our results demonstrate that Hollenhorst plaques are a marker of significant carotid disease irrespective of the presence of retinal symptoms. Overall, 25.2% of 237 patients had carotid stenosis >40%, and 13.2% of patients had carotid stenosis >69%. Symptomatic patients were more likely than asymptomatic patients to have carotid stenosis > 69% (25.0% vs. 9.2%; p=0.008).

Our results also show that carotid auscultation remains important in the examination of these patients with Hollenhorst plaques. Asymptomatic patients with a bruit were more likely than those without a bruit to have carotid stenosis >40% or >69% and had a higher rate of carotid endarterectomy. Among symptomatic patients, the percentage of patients with stenosis >69% was 25% in the group with documented carotid bruits compared with 9.2% in the group without carotid bruits (*P*=.008). Among asymptomatic patients, stenosis >69% was found in 37% of those with carotid bruits compared to only 4.3% of patient without a carotid bruit (*P*=.0001). This was an interesting finding, as recent studies have questioned the utility of carotid bruit in predicting significant carotid disease. In 1994, Sauve and colleagues[21] evaluated the predictive value of carotid bruit in patients enrolled in the NASCET. They reported that the predictive value of carotid bruit for significant carotid stenosis ranged from 18% to 94%, depending on the clinical situation. They concluded that carotid bruit alone is not a sufficient marker of significant carotid disease. A 2007 population study of 2736 asymptomatic patients showed that a carotid bruit on examination did not increase the likelihood of finding ipsilateral carotid stenosis >50%, intimal medial thickness above the median, or ipsilateral carotid plaque on carotid duplex ultrasound.[22] However, despite these reports, two large population studies have shown that carotid bruits are independent risk predictors for stroke, myocardial infarction, and death.[23,24] The community-based Fremantle Diabetes Study[23] evaluated 1181 patients with type 2 diabetes mellitus and found carotid bruits were associated with a sixfold increase in the risk of stroke at 2 years. These findings were similar to the results reported out of the Framingham cohort in 1981.[24] In that cohort, asymptomatic carotid bruits were associated with a twofold increase in the risk of stroke and myocardial infarction. A recent meta-analysis of 17,295 patients in 20 prospective studies and 2 retrospective studies evaluating the utility of carotid bruit found a twofold increase in the rates of myocardial infarction and vascular death.[25] Our results highlight the utility of carotid bruit evaluation in the evaluation of these patients. We believe it is

important to examine for a bruit in patients with Hollenhorst plaques, as this will increase the yield of patients having severe carotid stenosis.

The prevalence of significant atherosclerosis in the peripheral, cerebral, and coronary vasculature (Table 1) in our patients with Hollenhorst plaques indicates that these may be a marker for not only carotid disease but also disease in other vascular territories. Our data showed that the presence or absence of symptoms of retinal ischemia on presentation did not correlate with an increased risk of stroke, myocardial infarction, further retinal ischemic events, or death over 4 years of follow-up. This study was not designed to compare Hollenhorst plaque patients with age-matched controls to determine the difference in incidence of vascular and neurologic events. In 2006, Wang and colleagues[26] pooled the data from two large population studies and compared 111 patients with retinal cholesterol emboli with age- and risk factor–matched cohorts. They found that retinal cholesterol emboli were an independent risk factor for all cause mortality and stroke-related mortality. Because this was a population study, most of these patients were asymptomatic. This study confirmed the utility of retinal cholesterol emboli in predicting future stroke and all cause mortality. Bruno and colleagues[27] prospectively followed 70 men with asymptomatic cholesterol emboli for a mean period of 3.4 years and found higher rates of vascular events compared with age- and morbidity-matched controls. Their results also underscore the importance of Hollenhorst plaques as an early marker of significant vascular disease.

Our data shows that the presence of visual symptoms on presentation did not correlate with an increased risk for death or stroke compared to asymptomatic patients. It is interesting to note that at baseline there was a significantly higher use of aspirin in the asymptomatic group. We postulate that the lack of difference in vascular outcomes during our follow-up period maybe due to the fact that most symptomatic patients were placed on aspirin after the diagnosis, or that they may have had more aggressive medical management. This may have made both groups statistically similar in vascular risk profile.

This study has multiple limitations. Like all retrospective chart review studies, we are limited by the effective documentation of multiple physicians with various backgrounds. The symptomatic patients may have also received closer examination, which resulted in the increased reporting of carotid bruits. The diagnosis of Hollenhorst plaques is still somewhat subjective and relies on the experience of the ophthalmologist. Our study and others are limited by the interobserver variability in reporting a carotid bruit. Many cardiac murmurs can be confused for a carotid bruit.

Despite these limitations, the strength of our study is that this is the largest series to date that studies the natural history of Hollenhorst plaques and stratifies them into symptomatic and asymptomatic groups. Our results confirm the utility of detailed history and physical examination in the triage of patients with Hollenhorst plaques. Carotid ultrasound in patients with Hollenhorst plaques is important, regardless of symptoms, as it may detect carotid stenosis, which may be surgically correctable, and atherosclerosis, which may be an independent marker for future stroke and all cause mortality. The focus should be on lifestyle and risk factor modification in those patients with less than surgically correctable disease.

CONCLUSION

The results of our study indicate that asymptomatic Hollenhorst plaques are an important marker of carotid disease and should be subject to a medical workup, carotid auscultation, and carotid ultrasound in the same manner as symptomatic plaques. There was no significant difference between the proportion of symptomatic or asymptomatic patients with a carotid stenosis of at least 40%, and the minimum requirement in terms of management in both these groups is optimization of cardiovascular risk factors. A proportion of those with stenosis >69% in both groups will require carotid endarterectomy, with the difference in rate being not statistically significant in our study.

With the increasing utilization of ophthalmologic examination and telemedicine as a tool in the evaluation of patients with hypertension, diabetes, and other chronic vascular diseases, the number of patients diagnosed with Hollenhorst plaques will certainly increase. With the evolving guidelines on management of carotid stenosis, it is important to be aware of the implications of the asymptomatic plaque and to institute recommendations accordingly.

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