

Prevalence and Incidence of Carotid-Fetal-Posterior Syndrome

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Keywords

Carotid stenosis · Symptomatic carotid stenosis · Fetal posterior cerebral artery · CT angiography

Abstract

Introduction: Carotid-fetal-posterior (CFP) syndrome is a posterior cerebral artery (PCA) territory ischemic stroke/TIA caused by symptomatic $\geq 50\%$ carotid stenosis or occlusion via fetal posterior communicating artery. We aimed to assess the incidence of CFP syndrome and prevalence of CFP syndrome among symptomatic $\geq 50\%$ carotid stenosis or occlusion as these are unknown. **Methods:** We reassessed consecutive CTAs from 4,042 persons and included locally admitted patients with $\geq 50\%$ carotid stenosis or occlusion. These were assessed for symptoms and signs of possible posterior circulation stroke/TIA (suspicion of CFP syndrome). Among these, those with unilateral PCA territory stroke/TIA, ipsilateral stenosis, and fetal/fetal-type PCA were considered CFP syndrome. **Results:** We included 208 locally admitted patients with $\geq 50\%$ carotid stenosis or occlusion; 33 (16%) patients had suspicion of CFP syndrome, of which 3 (9%) had CFP syndrome. The prevalence of CFP syndrome was 2.9% of symptomatic $\geq 50\%$ carotid stenosis or occlusion; incidence was 4.23 per 1,000,000 person-years. Also, we found a lower prevalence of CFP syndrome (0.9%, $p = 0.047$) among

referred patients with symptomatic $\geq 50\%$ carotid stenosis or occlusion than among locally admitted patients with symptomatic $\geq 50\%$ carotid stenosis or occlusion. **Discussion/Conclusion:** CFP syndrome has a low incidence and low prevalence among symptomatic carotid stenosis cases. Given lower prevalence of CFP syndrome among referred cases than local, CFP syndrome seems susceptible to underdiagnosis. On the other hand, few cases with suspicion of CFP syndrome had CFP syndrome, why CFP syndrome also seems susceptible to overdiagnosis if detailed assessment is not employed.

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Introduction

To assess if a carotid stenosis is symptomatic is a routine duty in clinical neurology. Stroke and TIA in the posterior cerebral artery (PCA) territory are usually not considered to be caused by carotid stenosis as the PCA is usually fed by the vertebrobasilar arteries. There are several anatomical variants that allow for a carotid stenosis to embolize to PCA territory, most commonly a fetal PCA but also a persistent trigeminal artery [1–8]. We consider emboli from a carotid stenosis that

pass through a fetal PCA to reach the PCA territory as “CFP syndrome” (carotid-fetal-posterior syndrome).

Most recent European Society for Vascular Surgery (ESVS) guidelines acknowledges CFP syndrome as symptomatic carotid stenosis, but without any reference [9]. Large trials and other guidelines have been vague about whether CFP syndrome should be considered symptomatic stenosis, but none refutes it [10–13]. Literature of CFP syndrome we could identify was limited to 11 patients in 4 case reports [1–4] and 3 studies: Yang et al. [5] reported that among 21 patients with both recent middle cerebral artery (MCA)/anterior cerebral artery infarction and PCA infarction on MRI, 5 (24%) had CFP syndrome. In two reports totaling 117 cases with PCA stroke, 2 (2%) had CFP syndrome [6, 7]. We found no study that assessed prevalence of CFP syndrome among carotid stenosis, nor estimated the incidence of CFP syndrome. The aim of this study was to assess the prevalence of CFP syndrome among symptomatic $\geq 50\%$ carotid stenosis or occlusion and the incidence of CFP syndrome.

Materials and Methods

Participants

This is an additional analysis of previously reported cohort. We reassessed consecutive CTA exams from 4,042 persons aged >18 years. The exams were performed for various indications at or sent to the University Hospital of Northern Sweden (that houses a tertiary stroke unit) between 2010 and 2014. The scans were performed at 12 hospitals with the tertiary stroke unit being only available for carotid stenosis issues. In addition, all evaluations for carotid stenosis and referrals for carotid ultrasound were assessed to detect cases with symptomatic stenosis not examined with CTA (for selection bias assessment). During the study period, approximately 0.71 million person-years occurred in the local uptake area.

All persons with a $\geq 50\%$ carotid stenosis or occlusion on a CTA scan of sufficient quality to be interpreted were initially included. However, after all data had been assessed, we excluded referred patients due to suspected selection bias with low CFP prevalence and high use of carotid ultrasound alone. Among participants with several scans, the first scan of first episode was used. Exceptions were when CTA was redone due to poor image quality and when a later episode was more relevant (including some aspect of CFP syndrome or was the only symptomatic).

Clinical Data

Four observers (A.C.R., T.G., E.K., E.J.) reviewed the medical records of all participants. All events within 3 months before and after the presenting event were assessed when categorizing cases. All cases with any visual symptoms were reviewed by at least two observers. Cases with any aspect of posterior circulation (brain stem, cerebellum, and PCA territory) symptoms were systematically reevaluated by a neurologist with carotid stenosis expertise (E.J.). We systematically reassessed symptoms, signs, and recent infarction. Assessments done at the time of clinical management were used per default but were overruled when there was sufficient evidence to do so.

We recognized that MCA territory stroke can have homonymous anopia (usually not hemianopia but can be hemianopia in cases with major stroke). Stroke and TIA were considered as being from PCA territory when either (1) isolated homonymous anopia (often hemianopia), recent PCA territory infarction not required; (2) homonymous hemianopia combined with either hemisymptoms (motor or sensory – could be thalamic, hence could be PCA), aphasia (could be thalamic), and/or memory/delirium symptoms (could be hippocampus). Recent PCA territory infarction was not required. However, when no recent infarction confirmed PCA territory, the hemianopia had to be pronounced compared to other symptoms (i.e., vice versa compared to MCA territory); (3) any recent PCA territory infarction, even without hemianopia, but was usually associated with hemisymptoms, aphasia, and/or memory/delirium symptoms. For case synthesis and categorization, we did not limit the assessment to if a PCA territory was affected but assessed all symptoms from all recent events and all recent infarctions.

Radiological Data

For all cases with any clinical or radiological suggestion of CFP syndrome, native CT and MRI were reassessed by E.J. Whenever available, serial imaging was assessed. This was done unblinded to clinical data (as this was used for case synthesis). For the commonly affected borderzone between MCA and PCA (behind the posterior ventricle horn), we used a conservative approach: infarction in this location (which can vary in exact location [14]), was considered as MCA territory unless the infarction also clearly involved the usual PCA territory. PCA territory was considered as the posterior occipital lobe medial to the borderzone (usually including the medial edge of the occipital lobe), the inferiomedial part of the hemisphere, medial-posterior thalamus and the hippocampus. “Recent infarction” was defined as restricted diffusion on MRI and not yet chronic appearance on CT.

On CTA, the carotid stenoses were graded by NASCET criteria, as presented elsewhere [15]. E.J. assessed all for degree of stenosis images, AJF a subset. Care was taken to not confuse Pcom and nearby veins, especially in cases with missing Pcom. Pcom status was assessed similar to Coulier [16]: fetal PCA was defined as Pcom supplying the P2 segment with no visible P1 segment. Fetal-type PCA was similar, but P1 segment was visible but smaller than the Pcom. Hence, Pcom not visible, smaller, or similar to P1 was considered as not fetal/fetal type. All stenosis grading and Pcom/PCA assessments were done blinded to clinical data.

Definitions

Stroke was defined as previously described [15]: symptoms and signs of loss of cerebral function lasting >24 h of presumed vascular origin. TIA was similarly defined but lasting <24 h. Retinal event was defined as monocular blindness of presumed vascular origin (not subdivided by duration). We use “infarction” to describe the radiological finding, whereas “ischemia” is used to describe the clinical case.

CFP syndrome was defined as a TIA or ischemic stroke that fulfilled 5 of 5: (1) PCA territory affected (one, not both), which defined relevant side; (2) ipsilateral extracranial $\geq 50\%$ carotid stenosis or occlusion; (3) ipsilateral fetal or fetal-type PCA; (4) did not include symptoms or signs clearly indicating infratentorial stroke/TIA as this would indicate emboli passing vertebrobasilar arteries or multiple emboli from another source; (5) did not include any stroke/TIA from

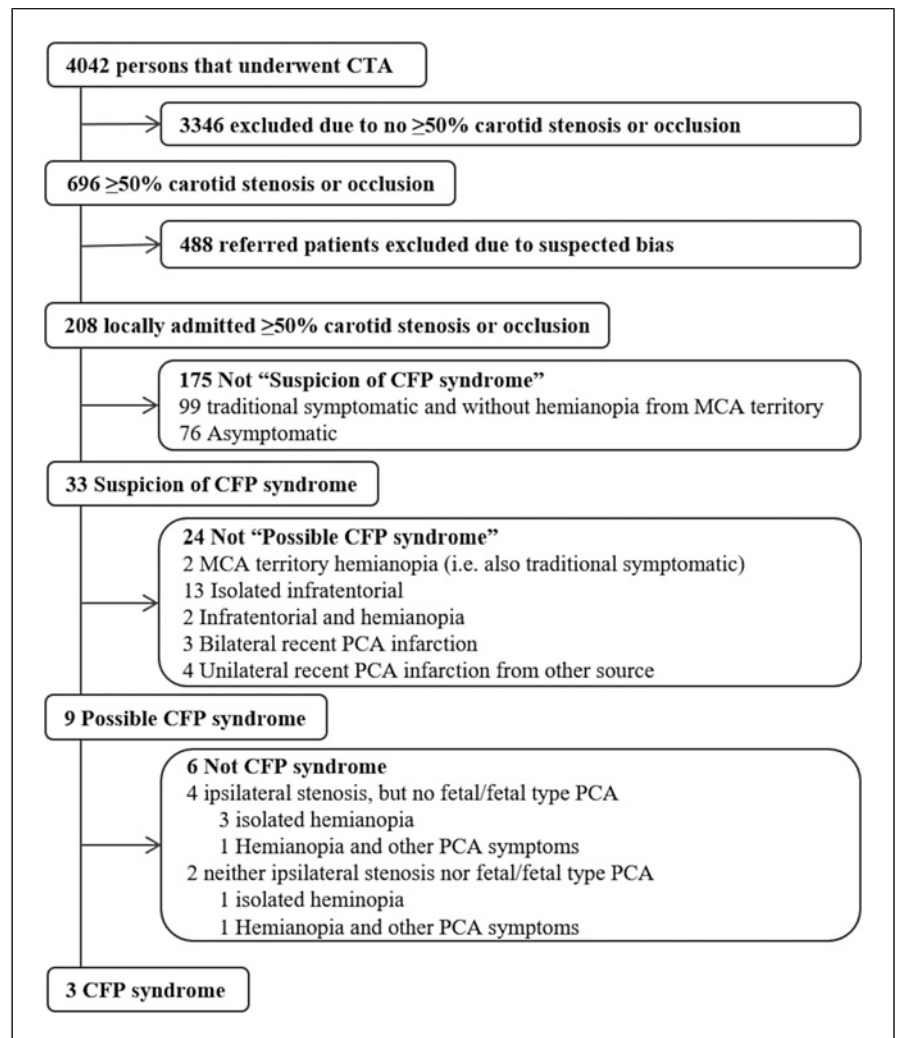


Fig. 1. Study flowchart.

contralateral carotid territory as this would indicate multiple emboli from another source. However, additional ipsilateral events from the carotid stenosis to traditional carotid territory (retinal, MCA, anterior cerebral artery) did not exclude CFP syndrome.

To facilitate results presentation, we defined “suspicion of CFP syndrome” as any symptom or sign that might reasonably be from posterior circulation (PCA, infratentorial and homonymous anopia from MCA territory). Also, we defined “possible CFP syndrome” as a stroke/TIA in a single PCA territory but regardless of stenosis or Pcom appearance. That is, of possible CFP syndrome cases, those with matching stenosis and fetal/fetal-type PCA had the full CFP syndrome.

Statistics

Case classifications, incidence, and prevalence were presented using descriptive methods. When assessing prevalence (n/N) of CFP syndrome among symptomatic stenosis, “n” was CFP syndrome cases and “N” was CFP syndrome cases plus typical symptomatic stenosis cases. We also compared clinical factors between patients with CFP syndrome,

symptomatic carotid stenosis, and asymptomatic carotid stenosis. Where appropriate, we used mean, median, standard deviation, interquartile range, 95% confidence intervals, 2-sided χ^2 test, and one-way ANOVA. We assessed the reliability of fetal/fetal PCA assessment between the observers using kappa values. We used IBS SPSS 28.0 for the calculations.

Results

Case Ascertainment and Exclusion of Referred Patients

We ascertained 696 participants with $\geq 50\%$ carotid stenosis or occlusion examined with CTA, of which 424 (61%) were symptomatic and 208 (30%) were locally admitted. The prevalence CFP syndrome among symptomatic $\geq 50\%$ stenosis or occlusion was higher among locally admitted patients (3/104; 2.9%) than among referred patients (1/320; 0.3%, $p = 0.047$). In addition, we ascertained 142 cases with

Table 1. Case classifications

Suspicion of CFP syndrome	Group	Symptom	<i>n</i>	Ipsilateral $\geq 50\%$ stenosis or occlusion ^a , <i>n</i> (%)	Confirming recent infarction, <i>n</i> (%)	Ipsilateral fetal/fetal-type PCA, <i>n</i> (%)	CFP syndrome
No	Symptomatic	Various in ipsilateral retinal, MCA, or ACA territory	99	99 (100)	Not assessed	14 (14)	NA
Yes	Asymptomatic ^b	Various or none	76	NA	Not assessed	18 (17) ^c	NA
	Hemianopia from MCA territory	Hemianopia + ipsilateral hemisymptoms	2	2 (100)	2 (100)	0 (0)	NA
	Isolated infratentorial	Various ^d	13	NA	2 (15)	2 (15) ^c	NA
	Infratentorial + PCA	Hemianopia + infratentorial ^f	2	1 (50)	0 (0)	0 (0)	NA
	Bilateral recent PCA infarction	Mixed	3	NA	3 (100)	0 (0)	NA
	Unilateral recent PCA infarction from another source ^g	Various	4	3 (75)	4 (100)	2 (50)	NA
Possible CFP syndrome	Isolated hemianopia	Isolated hemianopia	4	3 (75)	3 (75)	0 (0)	0 (0)
	Hemianopia + other PCA symptoms	Hemianopia + other PCA symptoms	5	4 (80)	4 (80)	3 (60)	3 (60)

ACA, anterior cerebral artery; CFP, carotid fetal posterior; MCA, middle cerebral artery; NA, not applicable; PCA, posterior cerebral artery. ^aWhen no ipsilateral $\geq 50\%$ stenosis or occlusion, the existing $\geq 50\%$ stenosis or occlusion was contralateral. ^bCTA indications for the asymptomatic participants were contralateral stroke/TIA with $< 50\%$ stenosis ($n = 25$), hemorrhagic stroke ($n = 4$), iatrogenic stroke ($n = 1$), suspected stroke/TIA but got other final diagnosis (such as syncope or seizure; $n = 33$), other diseases ($n = 2$), carotid bruit ($n = 5$), research ($n = 2$), follow-up of known stenosis ($n = 4$). ^cOn the side with most severe stenosis. ^dLikely affecting only brainstem and/or cerebellum (not PCA): 7 gaze disorders (nonconjugated gaze, diplopia, and/or nystagmus), 3 vertigo with paresis, 2 isolated vertigo, 1 quadriplegia. ^fLikely affecting brainstem and/or cerebellum and also a PCA territory, both had vertigo and hemianopia. ^gRecent infarction and/or symptoms from several territories, not fitting CFP syndrome. Two cases with fetal/fetal-type PCA: one fetal-type PCA and presentation suggested emboli via the small P1: single stroke with bilateral PCA-territory symptoms and signs and recent infarction in PCA territory only on the side with fetal-type PCA. One had fatal bilateral ischemic stroke: embolic occlusion terminal ICA on a side with neither stenosis nor fetal PCA. Contralateral chronic ICA occlusion and fetal PCA, i.e., a contralateral noncarotid origin embolus.

symptomatic $\geq 50\%$ stenosis or occlusion examined with ultrasound alone. Ultrasound alone was more common among referred patients with symptomatic $\geq 50\%$ stenosis or occlusion (120/440; 27%) than among locally admitted patients with symptomatic $\geq 50\%$ stenosis or occlusion (22/126; 17%, $p = 0.03$). Given these possible sources of selection bias, referred patients were excluded (Figure 1).

Case Classification

Of the 208 included patients, 33 (16%) had suspicion of CFP syndrome of which 9 (27%) had possible CFP syndrome (Table 1). Of the 9 with possible CFP syndrome, 4 (44%) had an ipsilateral $\geq 50\%$ stenosis or occlusion but no fetal/fetal-type PCA (Pcom not visible in all four), 2 (22%) had neither an ipsilateral $\geq 50\%$ stenosis or occlusion nor ipsilateral fetal/fetal-type PCA, and 3 (33%) had CFP syndrome (see Table 2 for baseline comparisons and

Figure 2 for case presentations). 16 patients had recent PCA infarction: 2 had CFP syndrome, 2 bilateral PCA infarction, 4 another source, and 6 not associated with ipsilateral $\geq 50\%$ stenosis or occlusion and ipsilateral fetal/fetal-type PCA (Table 1; Fig. 2). One patient with CFP syndrome had possible recent ischemia (Fig. 2).

Prevalence and Incidence

The prevalence of CFP syndrome was 2.9% (3/104; 95% CI: 0.0–6.1%) of symptomatic $\geq 50\%$ stenosis or occlusion and 2.2% (95% CI: 0.0–5.1%; 2/93) of symptomatic $\geq 50\%$ stenosis. Incidence of CFP syndrome was 4.23 (95% CI: 0.0–9.01) per 1,000,000 person-years.

Reliability

After excluding cases with pathological findings, such as basilar tip/P1 occlusion, 1259 Pcoms were assessed by the

Table 2. Baseline data

	Traditional symptomatic ^a (n = 101)	CFP syndrome (n = 3)	Asymptomatic (n = 104)	p value ^b
Age, mean (SD)	74 (9)	74 (10)	73 (9)	0.91
Men, n (%)	69 (69)	2 (67)	65 (63)	0.76
Previous myocardial infarction, n (%)	23 (23)	1 (33)	22 (21)	0.95
Current angina, n (%)	20 (20)	1 (33)	18 (17)	0.90
Current heart failure, n (%)	12 (12)	0 (0)	10 (10)	0.76
Current claudication, n (%)	7 (7)	1 (33)	6 (6)	0.19
Previous arterial revascularization, n (%)	27 (27)	0 (0)	36 (35)	0.20
Atrial fibrillation, n (%)	16 (16)	0 (0)	14 (14)	0.81
Current smoking, n (%)	13 (13)	1 (33)	1 (18)	0.33
Hypertension, n (%) ^c	98 (97)	3 (100)	101 (97)	1.0
Diabetes, n (%)	24 (24)	0 (0)	18 (17)	0.32
Total cholesterol, mean (SD)	5.0 (1.3)	4.7 (0.4)	4.8 (1.3)	0.58
LDL cholesterol, mean (SD)	3.1 (1.1)	3.0 (0.6)	2.7 (1.1)	0.07
HDL cholesterol, mean (SD)	1.21 (0.30)	1.32 (0.34)	1.25 (0.41)	0.74
Presenting event				
Stroke, n (%)	50 (51)	3 (100)	–	0.29
TIA, n (%)	32 (32)	0 (0)	–	
Retinal, n (%)	17 (17)	0 (0)	–	
Ipsilateral stenosis on CTA ^d				
50–69%	38 (39)	1 (33)	62 (60)	0.06
≥70%	29 (30)	0 (0)	21 (20)	
Near occlusion	21 (21)	1 (33)	14 (14)	
Occlusion	10 (10)	1 (33)	7 (7)	
Ipsilateral fetal/fetal-type PCA	16 (16)	3 (100)	18 (17)	0.005
Contralateral fetal/fetal-type PCA	23 (23)	2 (67)	26 (25)	0.22
CEA/CAS treatment	44 (44)	0 (0)	12 (12)	<0.001

CFP, carotid fetal posterior; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SD, standard deviation; PCA, posterior cerebral artery. ^aSymptomatic: 99 cases +2 with MCA hemianopia and an ipsilateral stenosis, i.e., excluding CFP syndrome. ^b2-sided χ^2 test for categorical, one-way ANOVA for continuous. ^cBlood pressure >140/90 and/or use of blood pressure medication. ^dMissing data for 3 cases with conventional $\geq 50\%$ stenosis due to too calcified stenosis for assessment, all symptomatic.

two observers. Overall agreement on fetal/fetal PCA versus not was 92% (1154/1259), kappa 0.68 (95% CI: 0.63–0.74).

Discussion

The main findings of this study were that CFP syndrome is rare in general (low incidence), rare among symptomatic carotid stenoses cases, and uncommon among patients with suspicion of CFP syndrome (i.e., carotid stenosis and posterior circulation presentation).

We found that local patients more often had suspicion of CFP syndrome and CFP syndrome than referred patients, suggesting selection bias. Asymptomatic CEA was done at the time of the study, why asymptomatic stenoses were reasonable to refer in general. Referral pattern of detected stenoses could still be a mechanism of selection: limiting carotid exams to stroke/TIA cases with

anterior circulation symptoms and signs and use of only carotid ultrasound might be other causes for selection, missing cases with CFP syndrome. Given the likely selection bias among referred patients, the main analysis was conducted only on locally admitted patients. Nevertheless, our CFP syndrome for locally admitted patients might be underestimated given that we had no case with CFP syndrome presenting with TIA (albeit not reaching statistical significance).

In NASCET, events had to be “in the appropriate carotid artery distribution” in the inclusion criteria, i.e., neither highlighting or excluding CFP syndrome [10]. In ECST, cerebral events for eligibility were described as, “i.e., a stroke in those parts of the brain principally supplied by branches of that internal carotid artery,” which makes CFP unlikely but not excluded [11]. We have found no NASCET or ECST analysis on CFP syndrome. However, even if it had been presented, number of cases would

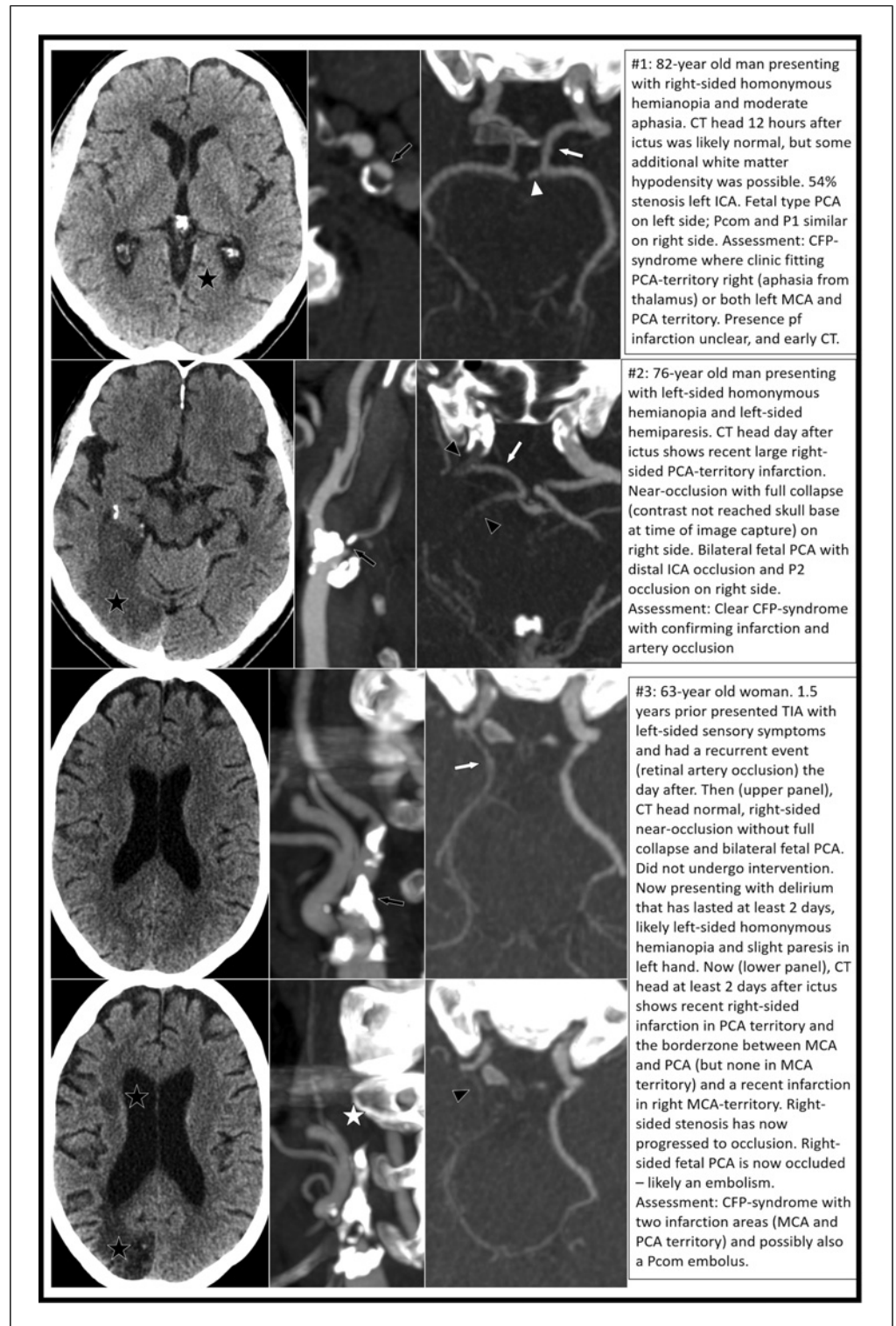


Fig. 2. Three cases of CFP syndrome (first two has a single panel/row, the third has two panels/rows). All: plain CT head, CTA of the stenosis, and of the P1-P2-Pcom part of the Circle of Willis. Black arrow: stenosis. Black arrowhead: embolic occlusion. White arrow: ipsilateral Pcom. White arrowhead: P1. Black star: recent infarction (around or adjacent to the star). White star: occluded ICA.

reasonably have been too low for reasonable conclusions about treatment effect. The most recent ESVS guidelines acknowledge CFP syndrome, albeit without any reference [9]. ESO and SVS guidelines neither refute nor acknowledge CFP syndrome [12, 13]. From a clinical-reasoning perspective, CFP syndrome is a reasonable diagnosis and relevant as it changes a presumably asymptomatic carotid stenosis to a presumably symptomatic stenosis. Four take-home messages for clinical use are as follows: (1) CFP syndrome is rare. (2) CFP syndrome is easy to miss, especially if carotid evaluations are limited to cases with anterior circulation events. In the era of thrombectomy, the presumed increased use of CTA might increase the likelihood of CFP syndrome detection as the degree of carotid stenosis and Pcom status is made available even in cases with presumed posterior circulation events. (3) CFP syndrome is often not limited to isolated hemianopia. None of our three CFP cases had isolated hemianopia. (4) Even when there is suspicion of a CFP syndrome, the full syndrome is still uncommon: 3/33 (9%) of suspected CFP syndromes had the full syndrome. Trainees should be warned not to overinterpret co-existing posterior circulation clinic and carotid stenosis as CFP syndrome. Thus, while it seems reasonable to aggressively pursue possible CFP syndromes with detailed clinical and radiological assessments, it is also reasonable to be conservative in reaching the CFP syndrome diagnosis.

A study strength was the start point with reassessment of consecutive CTAs, allowing for detection of carotid stenosis and CFP syndrome that might not have been understood at the time of clinical assessment. Limitations were the retrospective approach and that there was a selection to CTA at the time of the study, albeit not that many among locally admitted cases, i.e., a slight underestimation of CFP syndrome is possible. The reliability of fetal/fetal-type assessment was acceptable but not excellent. That we had to limit our analysis to locally admitted patients due to likely selection bias among referred patients was a limitation in the sense that it caused a smaller sample but was also an important finding.

Conclusion

CFP syndrome has a low incidence and low prevalence among symptomatic carotid stenosis cases. Given lower

prevalence of CFP syndrome among referred cases than local, CFP syndrome seems susceptible to underdiagnosis. On the other hand, few cases with suspicion of CFP syndrome had CFP syndrome, why CFP syndrome also seems susceptible to overdiagnosis if detailed assessment is not employed.

Statement of Ethics

The study was approved by the Ethics Review Board in Umeå, Sweden, approval number 2014/314-31. Need for informed consent was waived by the Ethics Review Board in Umeå, Sweden, due to the observational design.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

A.C.R. and E.J. researched the literature. A.C.R., T.G., E.K., A.J.F., and E.J. gathered data. A.N. and E.J. designed clinical assessment protocol. E.J. conceived and designed the study, acquired funding, supervised, and designed the first draft. All authors reviewed and edited the manuscript and approved the final version of the manuscript.

Data Availability Statement

The data that support the findings are not publicly available because it contains information that could compromise the privacy of research participants. All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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