Original Article

# High-Resolution Vessel Wall Imaging in Primary Angiitis of Central Nervous System

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## Abstract

**Background:** High-resolution vessel wall imaging (HRVWI) can aid in differentiating the various intracranial vasculopathies, but has been sparingly used in the diagnosis of primary angiitis of central nervous system (PACNS). This study is aimed to describe the vessel wall imaging characteristics of PACNS. **Materials and Methods:** Patients with confirmed diagnosis of PACNS according to the Calabrese and Mallek criteria who had abnormal HRVWI were included in this retrospective descriptive study. Magnetic resonance image of brain, conventional four-vessel cerebral digital subtraction angiogram, and HRVWI were read by a neuroradiologist. The vessel wall parameters assessed were T1W and T2W appearances, pattern of wall thickening and contrast enhancement, and remodeling index. **Results:** HRVWI done in 21 patients with PACNS yielded abnormality in 20 (95.2%) who were included in the analysis. The mean age at presentation was 42.55 ± 9.48 years and 14 (70%) were males. The median number of vessels involved were four (range 2–12). The commonest vessels affected were proximal middle cerebral artery (70%) and internal carotid artery (55%). Vessel wall thickening was concentric, eccentric, and absent in 12 (60%), 1 (5%), and 7 (35%) patients, respectively. Vessel wall enhancement was diffuse in 17 (85%), eccentric in 1 (5%), and absent in 2 (10%) patients. One patient had T2W hyperintense stenotic lesion. Remodeling index was negative in 11 (55%) patients. **Conclusion:** Distinctive vessel wall appearances were observed by HRVWI in PACNS, concentric vessel wall thickening and enhancement being more frequent. Hence, HRVWI can be considered as an additional noninvasive imaging modality in the diagnosis of PACNS.

**Keywords:** HRVWI, PACNS, primary angiitis of CNS, vessel wall enhancement, vessel wall imaging, vessel wall thickening

## Introduction

Primary angiitis of central nervous system (PACNS) is a rare form of single organ vasculitis that is limited to the brain and spinal cord, the exact cause of which is largely obscure.[1] The neurological manifestations of PACNS, such as headache, cognitive decline, seizures, and focal deficits, are nonspecific with a broad range of differential diagnosis. In spite of an improved understanding about the natural course, neuroimaging and histopathological features, and factors determining the outcome and prognosis, PACNS still poses a diagnostic and therapeutic challenge to clinicians.[2-4]

Currently, the diagnosis of PACNS is established by demonstrating alternate areas of stenosis and dilatation of multiple arteries by conventional four-vessel cerebral digital subtraction angiogram (DSA) and/or transmural inflammation of cerebral blood vessels by meningocortical biopsy (MCB).[5] However, the diagnostic yield of MCB and DSA is only in the range of 60%–70% and 70%–88%, respectively, in the three large cohorts of PACNS.[2–4] Despite being the gold standard for the diagnosis of CNS vasculitis, MCB may fail to reveal any abnormality due to either a patchy involvement of the inflammatory process, or predominant large vessel involvement.[1–3] Moreover, there is a considerable delay in reaching a diagnosis of PACNS by MCB when compared to DSA.[2,4] Mild neurological deficits, initial response to treatment, invasiveness of the procedure with perioperative complications, and low sensitivity are a few reasons cited for delaying or not considering biopsy.[1,4] Since PACNS is a catastrophic inflammatory disorder, delayed diagnosis and suboptimal treatment can lead to significant morbidity and mortality.[6] Hence, early diagnosis gives an opportunity for treating these patients more effectively.

High-resolution vessel wall imaging (HRVWI) using multiplanar three-dimensional (3-D) acquisition, multiple tissue weighting, and suppression of luminal blood and cerebrospinal fluid (CSF) has been found to be a promising novel technique that could aid in the early diagnosis of PACNS.[7,8] The key findings observed in PACNS are concentric thickening and diffuse homogeneous, concentric

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**Submitted:** 04-Feb-2021 **Revised:** 21-Feb-2021 **Accepted:** 04-Mar-2021
**Published:** 21-May-2021



**DOI:** 10.4103/aian.AIAN_106_21

enhancement of the vessel wall. Since angiographic findings of PACNS closely mimic reversible cerebral vasoconstriction syndrome (RCVS), intracranial atherosclerotic disease (ICAD), and radiation vasculopathy, the distinctive changes directly visualized in the intracranial vessel wall by HRVWI can help in differentiating these intracranial vasculopathies and confer a precise diagnosis.[8] Remodeling of the arterial lumen due to underlying pathology can be estimated using remodeling index, which can also give an additional clue towards diagnosis.[9]

Atypical vessel wall imaging features of eccentric wall thickening and T2W hyperintensity of the vessel wall are also rarely reported in PACNS, which requires further elucidation.[8,9] Additionally, very few studies have explored the clinical utility of HRVWI in PACNS.[10,11] The aim of this study was a descriptive analysis of the vessel wall imaging features in PACNS.

## Materials and Methods

### Subjects

This is a retrospective descriptive study of patients satisfying the Calabrese and Mallek diagnostic criteria for PACNS from the Comprehensive Stroke Care Unit and Department of Neurology between January 2016 and December 2019.[5] We searched the electronic medical records of our institute with key words "CNS vasculitis," "primary angiitis of CNS," and "PACNS" for the selection of the cases. Patients with neurological manifestations suggestive of PACNS were included in this study when they had (i) either DSA and/or biopsy evidence for vasculitis and (ii) abnormal HRVWI findings. Patients with CNS vasculitis mimics such as RCVS and ICAD and secondary causes of CNS vasculitis were excluded from the study. The details of the investigations done in patients with PACNS for secondary causes have been previously described.[4] The demographic and clinical data were recorded from case files. Laboratory parameters including CSF analysis and MCB reports were noted. The outcomes studied were presence of relapse, modified Rankin scale (mRS), and mortality at 6 months after initiating treatment for PACNS after confirming the diagnosis and at last follow-up.[2]

### Neuroimaging

Magnetic resonance imaging (MRI) of brain at 1.5 or 3 Tesla done during the initial visit to our institute and DSA were reviewed by a neuroradiologist. The pattern of infarcts (single and multiple), grading of small vessel ischemic changes (Fazekas grading),[12] microbleeds, and contrast enhancement (leptomeningeal and parenchymal) were noted. The type of vessel involvement in DSA was classified as large, medium, or small, based on the size of the affected vessels.[4,13]

### High-Resolution Vessel Wall Imaging (HRVWI)

HRVWI was acquired with 3 Tesla MRI machine according to the standard protocol at our institute which was published previously. Time-of-flight magnetic resonance angiogram as a localizer followed by 3-D variable refocusing flip angle (VRFA) CUBE sequences was done in all patients. The imaging sequences included were T1 and T2 sagittal Cube, sagittal Cube proton density, and fat-suppressed sagittal Cube T1 pre- and postcontrast.[14,15] The images were reviewed by a neuroradiologist (blinded) for vessel wall changes in the distal internal carotid artery (ICA), proximal middle cerebral artery (MCA—M1 and M2), anterior cerebral artery (ACA) and posterior cerebral artery (PCA), posterior–inferior cerebellar artery, superior cerebellar artery, vertebral artery (VA), and basilar artery (BA). The vessel wall parameters assessed were T1W and T2W vessel wall appearance (isointense, hypointense, and hyperintense), T1W vessel wall thickening and pattern of thickening, vessel wall enhancement, and remodeling index. The vessel wall thickening, when present, was classified into concentric and eccentric. When the thickening was circumferential and uniform, it was labeled as concentric, and focal or less uniform thickening as eccentric.[7] Vessel wall enhancement was graded into grade 0 (no enhancement), grade I (mild; when the signal intensity of vessel wall was less when compared to pituitary infundibulum), and grade II (severe; when the signal intensity was equal to or greater than that of the pituitary infundibulum).[15,16] The pattern of enhancement was also classified into concentric (circumferential and uniform) and eccentric (focal or less uniform).[15,17] The remodeling index was calculated as the outer diameter of a narrowed vessel divided by the caliber of the normal proximal reference vessel. The ratio of >1.05 was taken as positive remodeling, <0.95 as negative remodeling, and normal when ratio was in between these two values. The caliber of the vessel was calculated in the sagittal-oblique images perpendicular to the vessel in axial sections of T1 fat-suppressed sequences.[15]

### Statistical analysis

Descriptive statistics were used for analysis. Continuous and categorical variables were reported as mean ± standard deviation or as median [interquartile range (IQR)] and proportions, respectively. Based on mRS score, the outcome at 6 months was dichotomized into good (mRS ≤2) and poor (mRS >2 including death).

### Ethics approval

This study was approved by the Institutional Ethics Committee (IEC) of Sree Chitra Tirunal Institute for Medical Sciences and Technology (IEC/830).

## Results

There were 45 suspected cases of CNS vasculitis during the study period and 27 cases were confirmed to have PACNS based on DSA (24) and MCB (3) [Figure 1]. HRVWI done in 21 patients showed vessel wall changes in 20 (95.2%) and were included in the final analysis. The one patient with normal HRVWI was excluded from the study. The mean age at presentation was 42.55 ± 9.48 years and 14 (70%) were male. Stroke was the main clinical presentation seen in 18 patients (90%) followed by dementia in 2 (10%). The demographic profile, clinical features, and laboratory

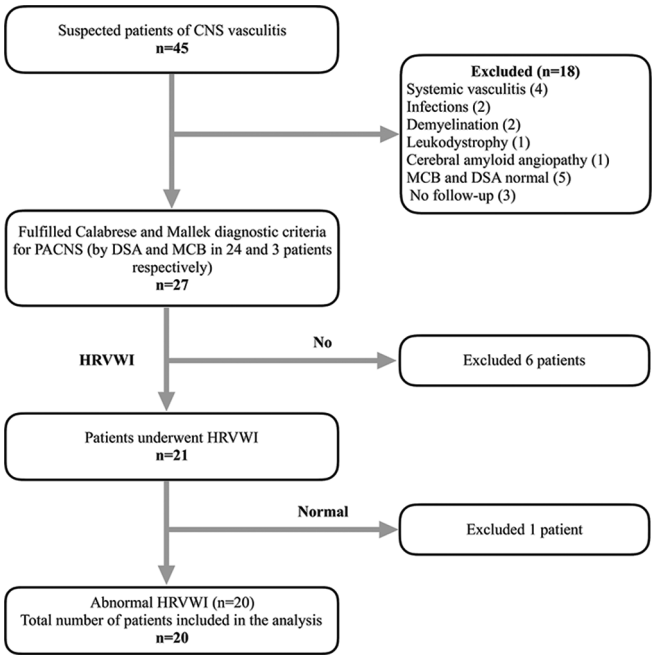


**Figure 1:** Flowchart depicting the inclusion and exclusion of patients in this study. Abbreviations: DSA-digital subtraction angiography; HRVWI-high-resolution vessel wall imaging; MCB-meningocortical biopsy; *n*-number of patients; PACNS-primary angiitis of central nervous system

parameters are described in Table 1. Poor outcome at 6-month follow-up was seen in 5 (25%) patients, and 6 patients (30%) had relapse and no mortality at last follow-up.

In 19 patients (95%), diagnosis was confirmed by DSA and in 1 patient (5%) by MCB. In the single patient who was diagnosed by MCB, the diagnosis was made after 142 days. Twelve patients with angiographic evidence for PACNS underwent MCB, of which two patients underwent targeted biopsy; but none of them had histopathological confirmation for CNS vasculitis. Table 2 depicts the MRI and DSA findings in the study group. In 19 patients who had evidence of vasculitis by DSA, large, medium, and small vessel involvement were seen in 13 (68.4%), 16 (84.3%), and 11 patients (57.9%), respectively.

The median time from onset of clinical manifestations to the HRVWI was 115 (IQR, 157.5) days. T2W sequences were not available for review in two patients. The vessel wall thickening was concentric in 12 (60%) patients and eccentric in 1 patient (5%). Seven patients did not have any vessel wall thickening. Postgadolinium contrast T1WI showed diffuse concentric enhancement [Figure 2] in 17 (85%), eccentric enhancement in 1 (5%), and no enhancement in 2 patients (10%). The details of T1W and T2W vessel wall appearance, grade of hyperintensity, and remodeling characteristics are mentioned in Table 3. The median number of vessels involved were 4 (range 2–12). The most common vessels affected in the decreasing order were proximal MCA (70%), ICA (55%), ACA (40%), PCA (35%), and BA (35%).

Two patients had atypical HRVWI findings. Focal eccentric vessel wall thickening and enhancement was seen in a 44-year-old hypertensive male who presented with hemorrhagic stroke [Figure 3]. MRI showed multiple microbleeds (60 in number) and he had classical features of CNS vasculitis in DSA. There was focal thickening and enhancement of bilateral ICA, M1, VA, and BA with M1 having grade 2 enhancement in HRVWI. The other patient was a 34-year-old female, who had T2 hyperintensity of left petrous and supraclinoid ICA. She was presented with bihemispheric stroke and had no vascular risk factors. MRI brain showed multiple infarcts and grade 2 Fazekas changes. She had angiographic evidence for vasculitis involving multiple vessels of the anterior circulation. Her HRVWI showed concentric vessel wall thickening and enhancement of bilateral ICA, MCA, and ACA. Except for left ICA, no other vessels were T2 hyperintense.

## Discussion

Very few studies have looked into the application of HRVWI with advanced sequences (3D-VRFA T1 and T2W CUBE) and utilizing the remodeling index exclusively in PACNS.[10,11,18,19] Here, we described the salient abnormalities seen in HRVWI in 20 patients with PACNS. The commonly observed vessel wall appearances were as follows: (i) concentric wall thickening; (ii) concentric enhancement with Grade I–II hyperintensity, and (iii) negative remodeling. Eccentric wall thickening with enhancement and T2 hyperintense vessel wall appearance were less frequent.

More than half of the patients with PACNS had vascular risk factors in our cohort and their presentation was stroke. Hence, confirmation of diagnosis of PACNS was as important as excluding ICAD, RCVS, and arterial dissection as alternative causes. Schematic analysis of the HRVWI could solve this puzzle. First and foremost is to look for T2 signal changes of the vessel wall.[10] In our cohort, T2WI showed hyperintensity in the stenotic lesions of left ICA in a single patient only. T2W hyperintense vessel wall appearance has a sensitivity of 79% and specificity of 100% in differentiating ICAD from PACNS/RCVS.[10] T2 hyperintensity with underlying hypointensity in the arterial wall is a pointer toward a diagnosis of ICAD, although CNS vasculitis has been rarely reported to have this finding.[10,11] Apart from a single vessel with atypical T2 signal changes, other affected vessels in this patient had concentric wall thickening and enhancement that was suggestive of CNS vasculitis and absence of vascular risk factors supporting the diagnosis. Pathologically, the juxtaluminal T2 hyperintensity and underlying hypointensity in ICAD correspond to the fibrous cap and the lipid rich core, respectively.[20]

Identifying the pattern of wall thickening and enhancement is the next step in evaluating cerebral vasculopathies.[14] In our study, almost 60% of the patients had concentric wall thickening while 35% had none. Concentric enhancement and wall thickening favor PACNS.[7,18] However, this finding has

**Table 1: Baseline, clinical, and laboratory parameters of patients with PACNS**

| Baseline parameters | Total 20 patients, *n* (%) |
|---|---|
| Age of symptom onset, mean (SD) in years | 42.55 (9.48) |
| Sex, male | 14 (70) |
| Clinical features | |
| Headache | 7 (35) |
| Cognitive decline | 5 (25) |
| Seizures | 8 (40) |
| Hemiparesis | 16 (80) |
| Dysarthria | 16 (80) |
| Aphasia | 5 (25) |
| Sensory | 7 (35) |
| Ataxia | 5 (25) |
| Visual | 3 (15) |
| Vascular risk factors | |
| Diabetes mellitus | 5 (25) |
| Hypertension | 11 (55) |
| Dyslipidemia | 3 (15) |
| mRS, median (range) | 2 (0-4) |
| NIHSS, median (range) | 3.5 (0-18) |
| Cerebrospinal fluid analysis, abnormal | 14 |
| Median white blood cells/mm$^3$ (range) | 2 (2-105) |
| White blood cells >5/mm$^3$ | 3 (15) |
| Median protein levels, mg/dL (range) | 49 (25-95) |
| Protein levels ≥45 mg/dL | 14 (70) |
| First line treatment | |
| Glucocorticoids only | 8 (40) |
| Glucocorticoids and cyclophosphamide | 12 (60) |
| Median time between first visit at our institute and diagnosis in days, (range), IQR | 18 (6-142), 14.5 |
| 6-month outcome | |
| Relapse | 3 (15) |
| mRS, median (range) | 2 (0-4) |
| Poor outcome (mRS >2) | 5 (25) |
| Duration of follow up in days, median (range) | 409, range (118-1240) |

IQR-interquartile range; mRS-modified Rankin scale; NIHSS-National Institutes of Health Stroke Scale; *n*-number; SD-standard deviation

also been reported in 10% of stenotic lesions due to ICAD. The contrast enhancement patterns described in ICAD are diffuse (60%) in the majority, followed by heterogenous (25%) and focal (15%).[10] Another recent study did not observe any concentric thickening or enhancement in ICAD.[11] Absence of concentric vessel wall thickening was seen in seven patients in this study and in five of them, HRVWI was done almost 1 month after the ictus. The absence of vessel wall thickening could be related to the lack of vessel wall edema while on treatment. Eccentric vessel wall thickening and enhancement was observed in one patient in this study. Focal thickening and enhancement are usually reported in atherosclerotic process and has a sensitivity and specificity of 90.1% and 86.5%, respectively, in distinguishing ICAD from CNS vasculitis.[10] But this finding can also be rarely seen in PACNS.[10,18] When atypical vessel wall features are encountered in suspected PACNS, vessel wall appearance in other involved blood vessels, MRI brain (microbleeds and leptomeningeal enhancement), typical angiographic appearance in DSA or histopathology have to be relied upon for confirming the diagnosis.

The angiographic appearance of PACNS is almost indistinguishable from RCVS. Differentiating these two conditions in the acute phase is extremely important as the treatment strategies vastly differ among them. HRVWI can be especially useful in this situation as the presence of vessel wall enhancement can exclude RCVS.[8,14] However, lack of vessel wall enhancement in PACNS is not uncommon as was noted in two of our patients.[19,21] In one patient, HRVWI was done after 6 months of symptom onset while on treatment which could explain the lack of vessel wall enhancement seen in this case. Resolution of the vessel wall enhancement in PACNS was noticed in two of the six patients while on treatment during follow-up.[18] Thus, HRVWI can also give information on the temporal course of the vasculitic process. This could be another advantage of HRVWI as it can be coupled with MRI brain for assessing the radiological progression. Vessel wall imaging is already reported to be a useful prognostic marker in Moyamoya

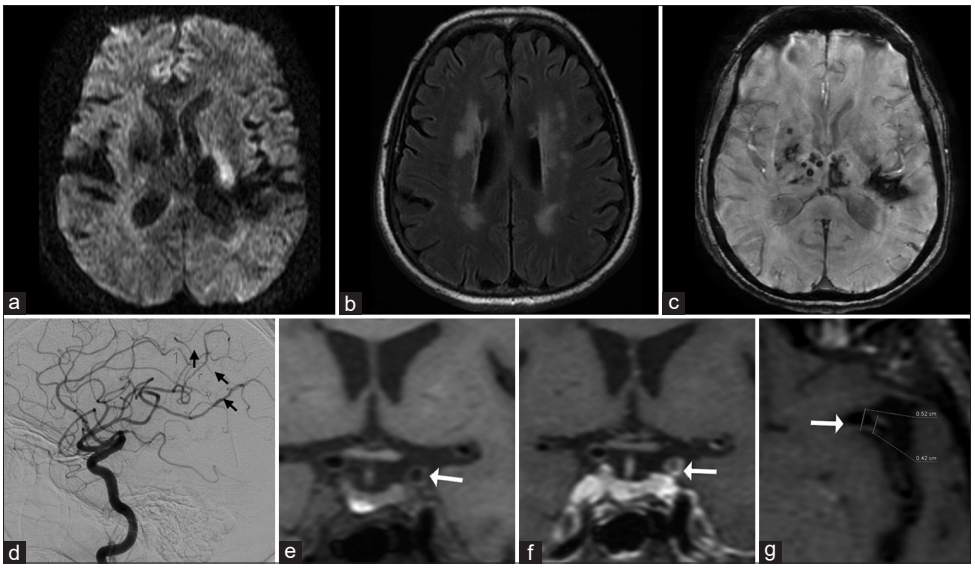


**Figure 2:** (a) DWI shows acute infarct in left lateral thalamus and posterior limb of internal capsule, (b) FLAIR images shows grade 2 Fazekas small vessel ischemic changes in periventricular white matter, (c) SWI with multiple microbleeds, (d) right ICA injection in lateral view showing multiple stenotic lesions in distal ACA and MCA branches, (e) T1W precontrast fat-suppressed (FS) image showing concentric thickening of left supraclinoid ICA, and (f) T1W postcontrast fat-suppressed image showing concentric grade 2 enhancement of the left supraclinoid ICA, and (g) T1WI (FS) showing remodeling index of 0.34 in left middle cerebral artery (MCA) suggestive of negative remodeling

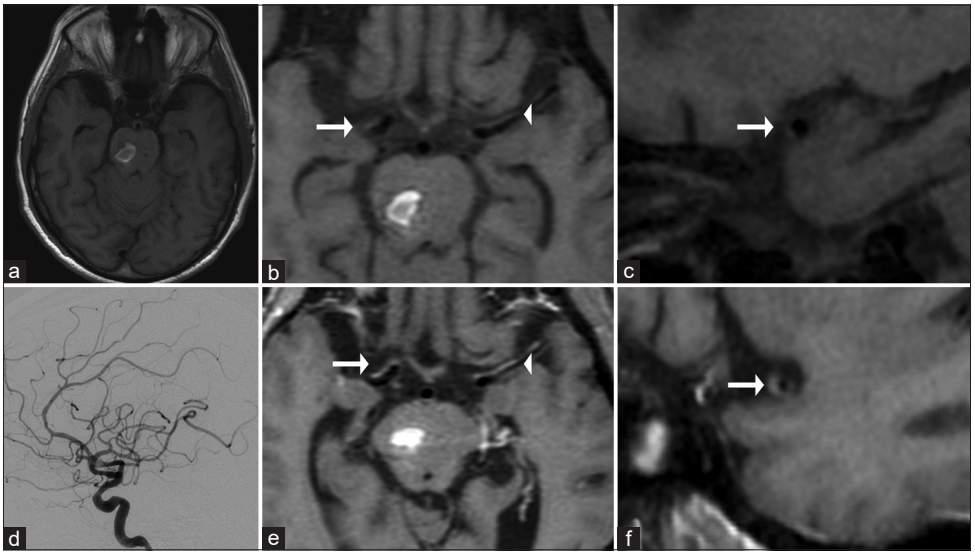


**Figure 3:** (a) T1W image shows right pontine hemorrhagic infarct and (d) left ICA injection oblique view shows multiple vessel stenosis, (b) T1W FS axial image shows right MCA (arrow) and left MCA (arrow head) eccentric wall thickening, (c) sagittal section shows left MCA eccentric wall thickening and corresponding section in post contrast images, (e) and (f) shows eccentric wall enhancement

disease. The vessel wall enhancement and thickening could predict the recurrence of ischemic events in Moyamoya disease within 3 months before and after imaging.[15] In our study, we had not investigated this aspect since HRVWI was used only as a diagnostic tool and follow-up imaging was not pursued in most. HRVWI as a tool for prognostication in PACNS needs to be explored prospectively to ascertain the applicability of this modality for monitoring disease activity.[15,18]

Another parameter in HRVWI that has been found to be helpful in differentiating vasculopathies is the remodeling index.[8] In this study, negative remodeling was seen in half of the patients with PACNS and none of them had a positive/outward remodeling index. This means that the outer diameter of the affected vessel was not increased in PACNS in spite of wall thickening. Another condition with a negative remodeling index is Moyamoya disease, which is due to the decrease in the outer diameter of the vessels that resulted from thinning of media and constriction of internal elastic lamina.[15] On the contrary, in atherosclerotic plaque, remodeling index may be positive due to arterial wall thickening, which produces an outward bulge without constriction of vessels.[8,14]

**Table 2: Magnetic resonance imaging and conventional cerebral angiogram findings in PACNS**

| Neuroimaging parameters | Total 20 patients, *n* (%) |
|---|---|
| Magnetic resonance imaging brain | |
| Single arterial territory infarct | 2 (10) |
| Multiple arterial territory infarct | 14 (70) |
| Small vessel ischemic changes (grade 2 and 3) | 12 (60) |
| SWI microbleeds | 13 (65) |
| Contrast enhancement | 10 (50) |
| Leptomeningeal | 9 (45) |
| Parenchymal | 0 (0) |
| Both leptomeningeal and parenchymal | 1 (5) |
| Digital subtraction angiography, abnormal | 19 (95) |
| Anterior circulation involvement | 7 (36.8) |
| Posterior circulation involvement | 2 (10.5) |
| Both anterior and posterior circulation involvement | 10 (52.6) |

*n*-number; SWI-susceptibility weighted imaging

**Table 3: High-resolution vessel wall imaging findings in PACNS**

| HRVWI parameters | Total 20, *n* (%) |
|---|---|
| T1W vessel wall appearance | |
| Hypointense | 8 (40) |
| Isointense | 12 (60) |
| Hyperintense | 0 (0) |
| T1W vessel wall thickening | |
| Absent | 7 (35) |
| Concentric | 12 (60) |
| Eccentric | 1 (5) |
| T2W vessel wall appearance (18 patients) | |
| Hypointense | 0 (0) |
| Isointense | 17 (94.4) |
| Hyperintense | 1 (5.6) |
| T1W post contrast vessel wall enhancement | |
| Absent | 2 (10) |
| Concentric | 17 (85) |
| Eccentric | 1 (5) |
| Number of vessels affected, median (IQR), range | 4 (4.5), 2-12 |
| Grading of hyperintensity of T1W post contrast enhancement | |
| Grade 0 | 2 (10) |
| Grade 1 | 16 (80) |
| Grade 2 | 2 (10) |
| Pattern of thickening (T1W) | |
| None | 5 (25) |
| Type 1 | 0 (0) |
| Type 2 | 15 (75) |
| Remodeling index (T1 fat-suppressed images) | |
| No remodeling | 9 (45) |
| Negative remodeling | 11 (55) |
| Positive remodeling | 0 (0) |

IQR-interquartile range; *n*-number

HRVWI can be a useful additional tool in supporting the diagnosis of PACNS, especially when biopsy is negative. The current diagnostic criteria for PACNS still relies on either DSA or MCB after meticulous exclusion of secondary causes of CNS vasculitis and mimics.[1,5] DSA is considered the first line investigation as it is less invasive and widely available. Majority of the patients in this study were diagnosed by DSA rather than MCB, which is similar to prior studies.[2,3] Most of the patients had stroke implying large vessel involvement, which was easily confirmed through DSA; hence, the early diagnosis in this study. A similar conclusion was also drawn from the French cohort where patients with stroke had large vessel involvement and those having isolated small-vessel PACNS more often had encephalopathy like episodes.[2,22] Predominant small vessel involvement can give a false negative angiographic result as they are beyond the spatial resolution of DSA.[3,22]

The diagnostic yield of DSA and MCB in PACNS from the three large cohorts were 60%–70% and 70%–88%, respectively.[2–4] However, the specificity and positive predictive value of DSA was found to be less than 30% and the biopsy had a negative predictive value of only 70%.[23] In the current study, MCB was abnormal only in 1 out of 13 patients, quite a low yield when compared to the previous studies.[2–4] Majority underwent blinded biopsy rather than targeted biopsy (11 versus 2 patients), which could explain the low yield. Blinded biopsy was pursued due to either lack of contrast enhancement or surgically inaccessible/eloquent area when contrast enhancement was present. False negative results by biopsy in this study could also be explained by the skip lesions in PACNS.[1] Henceforth, a newer complementary noninvasive investigation like HRVWI could circumvent the disadvantages of DSA and MCB. HRVWI has shown a high concurrence rate (> 90%) for detection of vasculitis in angiographically proven cases.[24] Similar concurrence rate was also observed in our study. This technique can also be used for guiding targeted biopsy from an inflamed vessel, which had shown abnormality in 8 out of 9 patients in a recent study thereby improving the diagnostic yield.[25]

We have utilized the latest technique of 3D-VRFA CUBE imaging sequences in 3T MRI according to the current guidelines for vessel wall imaging in this study.[8] The advantages of this technique includes high spatial resolution, and it provides multiplanar reformatting to eliminate partial volume artifacts and multiple tissue weightings for better characterization of vessel wall lesions. The entire brain imaging using full protocol can be completed in less time (30–35 min).[14] However, there are a few pitfalls associated with this imaging modality and for accurate interpretation, sufficient expertise is required. The vasa vasorum of the proximal intracranial arteries can give an appearance of vessel wall thickening and enhancement. An adjacent enhancing vein can be misinterpreted as arterial wall enhancement. An apparent impression of vessel wall thickening can be due to the slow blood flow. We should also take into consideration the motion artifacts that can compromise the image quality and the effect of therapy.[8]

Our study is limited by the small sample size and retrospective description of the findings. Vessel wall imaging was interpreted by a single neuroradiologist, hence interrater reliability could not be analyzed, which is another limitation of this study. Pathological correlation could have added strength to the imaging findings, but unfortunately, all except one biopsy was normal. In spite of these limitations, this will be first publication of HRVWI in PACNS that comprehensively describes the various vessel wall imaging parameters including remodeling index. Concentric vessel wall thickening and enhancement argues for PACNS, but atypical features of focal enhancement or T2 hyperintensity warrants meticulous analysis. Based on the description of the imaging findings, HRVWI could be an additional tool in the diagnostic armamentarium that helps the clinician in the evaluation of PACNS.

## Conclusion

PACNS is a heterogenous disorder that can affect any cerebral vessel irrespective of its size and poses many diagnostic uncertainties. Concentric vessel wall thickening and enhancement in HRVWI can favor a diagnosis of PACNS among other intracranial vasculopathies. Hence, vessel wall imaging can be considered as a potential additional noninvasive imaging modality in the diagnosis of PACNS.

### Ethical approval

This study was approved by the Institutional Ethics Committee (IEC) of Sree Chitra Tirunal Institute for Medical Sciences and Technology (IEC/830).

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

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