closure increased stroke volume and thereby functional capacity.

This case highlights the potential for RV remodeling after transcatheter ASD closure even in elderly adults with severe RV enlargement and tricuspid regurgitation. Improvement in the functional capacity and quality of life outweigh potential procedural complications in this population.

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DERAILED DEMENTIA: ALZHEIMER'S DISEASE VERSUS POSTERIOR CORTICAL ATROPHY

To the Editor: Benson's syndrome, or posterior cortical atrophy (PCA), is a progressive dementia that is distinguished by early and prominent visuoperceptual and visuospatial symptoms. Comorbidities such as posttraumatic stress disorder (PTSD) may obscure its recognition, diagnosis, and treatment.

CASE REPORT

A 71-year-old man with Benson's syndrome presented to the geriatric clinic for evaluation and management of behavioral disturbances. He was receiving quetiapine 75 mg twice daily and armodafinil 125 mg/d. His neurologist had "nothing further to offer." He had failed prior donepezil and memantine trials. Past medical history included hip replacement and genital herpes, with a family history of a mother with Alzheimer's disease (AD). His wife reported alexia, such as losing his place on the page while reading, with preserved accurate reading of small print but not large print, and misjudgment of distances when driving, resulting in minor car accidents. His wife described unusual activities such as placing watermelon rather than tomatoes on his hamburger, putting clothing on backward, donning two pairs of pants, and applying toothpaste to the cap rather than the toothbrush. He was depressed and anxious because of numerous prior optometry consultations during which no diagnosis was found for his visual abnormalities. Prior magnetic resonance imaging showed cortical volume loss in bilateral parietal lobes. Records documented dementia diagnosed at age 65. His Montreal Cognitive Assessment (MoCA) score was 5 out of 30 at the clinic appointment, which included a clock drawing showing an unusual constructional apraxia (Figure 1); his dementia laboratory examination was normal. He was verbally appropriate and described working with railroad cranes and four near-death experiences due to train accidents and derailment. He reported nightmares with flashbacks, night sweats, and a history of self-medicating with alcohol. Sertraline was initiated for undiagnosed PTSD and mood and behavior disturbances, with plans to taper off quetiapine because of

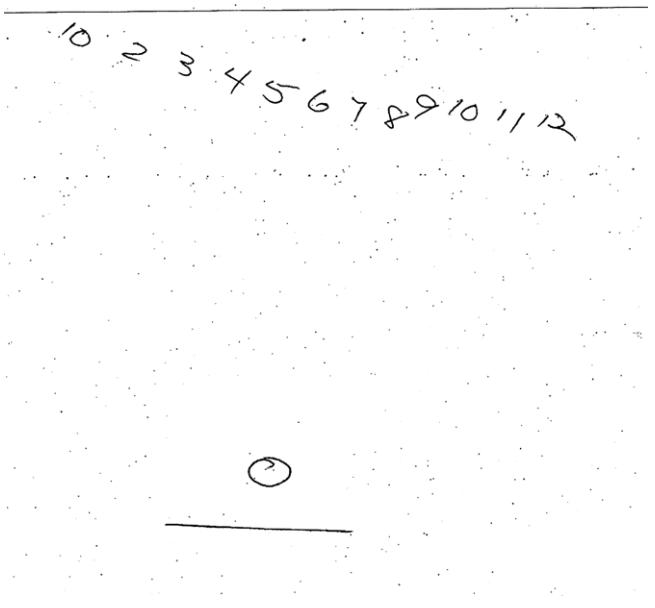

Figure 1. Clock drawing by individual at geriatric clinic (part of Montreal Cognitive Assessment).

orthostatic hypotension (supine blood pressure 130/84 mmHg; standing blood pressure 114/79 mmHg) and armodafinil. Referrals to a low vision clinic and caregiver support were made. Five weeks later, his wife reported that his behavioral symptoms improved significantly, stating, "I have my husband back now."

DISCUSSION

Dr. Frank Benson first described Benson's syndrome, or PCA, in a five-person case series in 1988.[1] Individuals present with early visual and visuospatial complaints in the absence of significant primary ocular disease[2] and may present as functionally blind.[3] Distinct from AD, memory, language, insight, and judgment are relatively preserved until late in the disease course.[1] Age of onset is typically 50 to 65, earlier than with typical AD. The prevalence and incidence of PCA are unknown, but it is considered an uncommon disease.[4] On imaging, there is atrophy of posterior brain regions,[5] with more than 80% of individuals with PCA found to have AD pathology as the cause of dementia at autopsy.[2,4]

No consensus criteria exist at present,[2] and there is disagreement as to whether PCA is linked to AD as a visual variant or subgroup of AD or perhaps a distinct nosological entity.[6] A recent multidisciplinary group of PCA research clinicians worked to develop a consensus on diagnostic criteria.[4] Core features included prominent visual impairment without significant impairment of vision itself, relative preservation of memory and insight, complex visual disorders such as Balint syndrome (consisting of ocular dysmetria (misreaching), sticky fixation (cannot shift gaze), and simultanagnosia (inability to perceive more than a single object at a time), Gerstmann syndrome (finger agnosia, left–right disorientation, acalculia, agraphia), visual field defects, visual agnosia, and environmental disorientation. Insidious onset and gradual progression with absence of stroke or tumor are other core features. There are also reports of strange manifestations of objects or letters suddenly moving, disappearing, or popping up when looking at them and reading small letters more easily than big ones.[7]

There are no PCA management guidelines, and only a few articles have mentioned using cholinesterase inhibitors.[3,7] In this man's case, worsening cognition may have triggered decompensation of his undiagnosed PTSD, and treatment with an antidepressant was instituted.[8] Peer support groups may be beneficial in initial and intermediate stages. Resources for blind and partially sighted people can be recommended.[9] Occupational and physical therapy are other resources, with a 10-week course of cognitive rehabilitation leading to some improvement in everyday functioning.[10]

PCA is an uncommon progressive dementia with an atypical cluster of symptoms. Mental health comorbidities may obscure diagnosis. The MoCA and other screening tests are heavily weighted using visual content. It is likely that clinicians will see more cases with the "graying" of the population.

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COMMENTS/RESPONSES

IMPROVEMENT OF AGE-RELATED MEMORY IMPAIRMENT WITH INFUSION OF YOUNG PLASMA: A ROLE FOR THE PERIPHERAL AMYLOID SINK?

To the Editor: A recent study attracted considerable interest in the field of geriatrics when it demonstrated that infusion of plasma from young mice improved age-related memory impairment in older mice. Its authors proposed that the presence of a "pro-youthful" factor in the blood of young mice or, alternately, removal of an "aging" factor in the blood of the aged mice caused the rejuvenating effect of plasma from younger mice.[1] A previous study that showed that infusion of plasma from aged mice impaired neurogenesis and spatial memory in young mice supported this hypothesis.[2] As a result of this new study, two investigations have been proposed: the search for