An 81-year-old man was admitted in emergency room for decreased general condition for three weeks with anorexia, loss of weight and insomnia. Blood tests revealed an important inflammatory syndrome with increased C-reactive protein (32 mg/dl). Because the patient was complaining of heavy headaches, a CDU of temporal arteries with a high frequency linear probe (17 MHz) was performed.

Longitudinal (Fig. 1A) and axial views (Fig. 1B) in B-mode of temporal artery revealed an important hypoechoic circumferential wall thickening (1.2 mm, Fig. 1B). The thickening starts at the origin of the temporal artery in contrast with a spared external carotid artery where thin wall is seen (Fig. 1C).

Color Doppler imaging revealed multifocal areas of aliasing (Fig. 2A) with acceleration in spectral Doppler, the peak systolic velocity was 90 cm/s (Fig. 2B) in contrast with 50 cm/s in the external carotid artery. No artery occlusion was seen but an impaired waveform downstream from these areas with low velocities (32 cm/s), monophasic appearance and diastolic positive component has been observed (Fig. 2C). Combined with the clinical presentation, these US results were suggestive of GCA. The patient received corticotherapy which produced a quick resolution of the inflammatory syndrome and the symptoms.

Discussion

GCA (Horton’s disease) is the most common primary systemic vasculitis and typically affects patients older than 50 years (1, 2). GCA is composed by two entities: temporal arteritis and polymyalgia rheumatica. Fever of unknown origin, weight loss and asthenia are often present. Temporal arteritis affects the large vessels and particularly the supra-aortic ones. Clinical presentation includes temporal or occipital headaches, jaw claudication and facial pain. The main complication is ophthalmic involvement (anterior ischemic optic neuropathy, central retina artery occlusion) with loss of vision.

GCA is often a clinical diagnosis. The American College of Rheumatology (ACR) published in 1990 a five point scoring system for the diagnosis of GCA that appears to be per-
temporal biopsy (4, 5, 6, 7, 8, 9). Combined with presence of stenosis or occlusion, sensitivity may be higher (5). The halo sign is not specific of GCA because it was described in a large range of arteritis (polymyositis nodosa, Henoch-Schönlein, Churg-Strauss, Takayasu disease), but the clinical presentation is often different. The treatment is corticotherapy with quick biologic response (diminishing of CRP) expected. Useful of CDU after corticotherapy is also under debate: some authors reported rapidly decrease of sensitivity of CDU under corticotherapy (9) and other authors reported that halo sign may persist after treatment with excellent clinical and biological response (7).

In conclusion, CDU is a performing and non-invasive first-line examination that permits, with clinical and biological findings, a relatively accurate diagnosis of GCA. Temporal biopsy remains the gold standard and should be reserved for negative scan or with positive scan with clinical discordance.

References