The literature published during the last ten years period regarding the vascular parkinsonism (VP) was reviewed. We reviewed articles published from 2006 to 2015 in English, cited in PUBMED database and in the web site Googlescholar.com. The key words for searching were: vascular parkinsonism (VP); cerebrovascular disease (CVD); lower body parkinsonism. The inclusion criteria for reviewing an article were: vascular parkinsonism and its definition, diagnostic criteria, epidemiological data, clinical features, symptoms and signs, brain imaging, scales, pathogenesis, risk factors, treatment, differential diagnosis.

**Definition**

The atypical parkinsonism (in particular postural and gait impairment, lower body symmetrical bradykinesia, rigidity and absence of resting tremor, and poor response to dopaminergic therapy) that is present concomitantly with CVD (clinical and in MRI and/or CT) and other clinical signs (dementia, pyramidal and pseudobulbar symptoms) is often referred to as vascular parkinsonism (VP). The clinical entity vascular parkinsonism has not yet been precisely defined [Hughes et al. 1992; Rektor et al. 2006]. Prevalence of VP from all parkinsonism is 8.8% [Horvath et al. 2011]. Pure VP is due to ischemic or hemorrhagic lesions in the substantia nigra, globus pallidus pars externa, thalamic ventral lateral nuclei, or nigrostriatal pathway, leading to presynaptic dopamine transporter deficiency as measured by single photon emission CT. The term pseudovascular parkinsonism was suggested for neurodegenerative parkinsonism with nonspecific neuroimaging signal abnormalities, vascular pseudoparkinsonism for akinetic mutism resulting from bilateral mesial frontal strokes or apathetic depression from bilateral striatal lacunar strokes, and pseudovascular pseudoparkinsonism for higher-level gait disorders, including normal pressure hydrocephalus [Vizcarra et al. 2015]. It was also suggested to replace the term lower body parkinsonism with cerebrovascular gait disorder [Rektor et al. 2006]. There were reported patients with dural arteriovenous fistula manifesting as parkinsonism and akinetic mutism and freezing of gait with dramatic improvement after endovascular treatment of fistula [Hattori et al. 2013, Luo et al. 2014, Netravathi et al. 2010, Shahar et al. 2012].

**Clinical features and differential diagnosis**

The magnitude of leukoaraiosis in MRI bears no correlation with the clinical phenotype and there is poor correlation between brain MRI hyperintensities and microangiopathic brain disease and VP from available clinicopathologic data [Vizcarra et al. 2015]. Clinical features of VP could include pyramidal signs, pseudobulbar palsy, cognitive impairment, history of stroke, rapid progression, urinary dysfunction [Korczyn 2015]. There are important red flags in the clinical diagnosis of VP as are the early onset or presentation with early postural instability and falls, freezing, lesser symptoms in the upper limbs, absence of classic 4-5 Hz pill-rolling resting tremor, presence of upper motor neuron signs as spasticity, extensor plantar(s) and other pathological reflexes, (snout, palnomental, corneomandibular, jaw, Hoffmann), and hemiparesis, ‘lead pipe’ rigidity more appreciable than ‘cogwheeling’ on examination, early or prominent dysphagia, dysarthria or involuntary laughter or crying, early onset of ‘subcortical’ dementia and no downgaze palsy, short shuffling parkinsonian gait with a wider base of stance and variable stride length, absence of festination [Gupta et al.2011, Kalra et al.2010, Vale et al. 2015, Okuda et al. 2008]. VP patients developed cognitive impairment with a significantly higher frequency than healthy control of a similar age with global pattern of cognitive impairment, including executive function, verbal memory and language. Only visuospatial function was more impaired.
in PD than in HC [Benítez-Rivero et al., 2014]. The presence of visual hallucination should be considered as a red flag for underlying Lewy body pathology [Williams et al. 2007]. VP patients had an extremely low frequency of visual hallucinations compared with PD [Glass et al. 2012]. Nocturnal manifestations (nocturnal hypokinesia, urinary incontinence, nocturnal dystonia, cramps and sleep disturbance are more experienced by patients with neurodegenerative parkinsonisms than VP [Bhidayasiri et al. 2014]. The PRIAMO study showed higher prevalence of non-motor symptoms in VP and other parkinsonisms (CBD, PSP, MSA) as compared to the more common PD [Colosimo et al. 2009]. Diferential diagnosis of lower body parkinsonism is VP, normal pressure hydrocephalus (idiopathic, secondary due to menigitis, head trauma), frontal lobe lesions (tumors, ischemia, demyelination) [Espay et al. 2007, Vizcarra et al., 2015].

**Biochemical markers**

Levels of homovanillic acid (HVA) in cerebrospinal fluid (CSF) were reduced in PD compared with both VP and controls but did not differ significantly between VP and controls indicating that dopamine deficiency was less pronounced in VP [Herbert et al. 2013]. Consistent evidence indicates the involvement of the brain-derived neurotrophic factor (BDNF) in neurodegenerative disorders. The results showed lower BDNF serum levels in VP patients and a higher BDNF concentration in patients affected by PD [Ventriglia et al. 2013].

**Imaging**

MRI scans of patients with VP and PSP had significantly smaller areas of midbrain than the patients with PD. As for the pons area there was a significant difference only between the VP and the PD [Choi et al. 2011]. Use of scan by123 I-Ioflupane (FP-CIT) SPECT is accurate to differentiate patients with PD from those with essential tremor (ET), and PD from VP and drug induced parkinsonism. The accuracy of both FP-CIT and 123 I-Iodobenzamide (IBZM) SPECT scans to differentiate between PD and atypical parkinsonism is low [Zijlmans et al. 2007, Scherfler et al. 2007, Vlaar et al. 2008, Huertas-Fernández et al. 2011, Contrafatto et al. 2012, Benitez-Rivero et al. 2012, Funke et al. 2013]. Myocardial postganglionic sympathetic dysfunction found in PD is absent in most patients with VP and cardiac [123I]metiodobenzylguanidine scintigraphy (MIBG) SPECT imaging may be useful to help distinguish between PD and VP patients [Kim et al. 2006 and J. Navarro-Otano et al 2013]. One of the predominant clinical features that differentiates VP from PD is the pyramidal sign. The triple stimulation technique (TST) is one of the most sensitive methods for comparing upper motor neuron involvement in patients with VP and PD [Jang et al. 2014].

**Therapy**

Besides the reduction vascular risk factors, external lumbar drainange (ELD), or ventriculo-peritoneal shunt (VPS) placement can be useful [Espay et al. 2007]. The repetitive magnetic stimulation could improve gait in a measurable way for up to 6 weeks without any significant side-effects [Yip et al. 2012]

**Conclusion**

Vascular parkinsonism is a heterogeneous clinical unit. Many aspects related to VP continue to be a source of confusion. Diagnostic criteria are not clearly defined and, consequently, patients with widely varied clinical pictures may be diagnosed with VP. Another critical stage is the interpretation of a finding of vascular lesions.