

PAIN IN PARKINSON'S DISEASE

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Pain is defined as an unpleasant or distressing sensory experience and has been recognized as a non-motor symptom in Parkinson's disease (PD). Pain may occur in 40-50% of patients according to the sparse literature on this topic. It has also been reported that PD patients suffer from pain e twice as often as age-edcontrolsmatch individuals. In a minority of individuals it is even more prominent than motor disturbances. Pain in PD can be subdivided toin several categories. The most common form of pain is due to the increase in muscle tone (rigidity), a finding which is similar to another form of increased muscle tone (spasticity) in multiple sclerosis. An increase in rigidity is correlated with low rgicdopamine stimulation. Therefore many PD patients complain of pain when their levo-dopa levels are low, for example in the wearing-off or end of dose period (diphasic or early morning dystonia and others). High levo-dopa levels may, however, also induce pain which is sometimes the case in peak-dose dyskinesia. Apart from its association with these levo-dopa levels, pain can also be subclassified according to its quality. There is musculoskeletal pain, neuritic or radicular pain and dystonia-associated pain, akathitic discomfort, central and peripheral pain. Some PD patients present with genital and oral pain such as burning mouth. No other definable organic causes of pain apart from PD could be found in these patients PD. It is therefore tempting to speculate that dopamine has central analgesic properties in such patients. Furthermore, it could be demonstrated that the intake of levo-dopa increased pain threshold. A PET study showed abnormalities in the insular and prefrontal region and in the anterior cingulated excortices of PD patients with pain. The introduction of levo-dopa normalised this condition. Sometimes, pain is associated with panic attacks and is when resolves the underlying cause of the pain is treated. The first step should be a careful exploration of the patient with respect to levo-dopa level-associated pain. In these patients, dopaminergic treatment, preferentially continuous dopaminergic stimulation, should be initiated. Moreover, there is also evidence for improvement of PD pain by cannabinoid-receptor antagonists, amantadine and opioids, but my own experience with non-steroid analgesics is not very encouraging.