OBJECTIVE BRAIN DIAGNOSTICS FOR ATTORNEYS

TRAUMATIC BRAIN INJURY CASE STUDY

Clinical History
A 46-year-old male with a history of multiple head injuries that was referred for brain qSPECT evaluation. Patient was experiencing a variety of progressive emotional, behavioral and cognitive symptoms since his second brain injury in the fall of 2012.

Chief Complaints
- Confusion
- Short-term memory problems
- Decreased judgment
- Personality changes
- Impulse control problems
- Difficulty with concentration
- Difficulty following instructions
- Making careless mistakes
- Distractibility
- Risky behavior
- Compulsive behavior
- Frequent dizziness
- Balance problems
- Blurred/double vision

Brain qSPECT® Findings
Focal areas of abnormal cortical hypoperfusion in the frontal, temporal, occipital and cerebellar areas (signature pattern for traumatic brain injury). In addition, focal areas of abnormal subcortical hypoperfusion were noted in the basal ganglia and thalamic nuclei.

The frontal/occipital findings are suggestive of a coup/countercoup mechanism of injury frequently seen in trauma. The apparent functional widening of the anterior interhemispheric fissure is suggestive of underlying functional atrophy of the frontal lobes.

The pattern of these abnormalities is most consistent with the scientific literature pertaining to traumatic brain injury and the patient’s clinical history.
Clinical History

52-year-old male patient referred for evaluation for toxic brain injury due to carbon monoxide poisoning. As a result of a faulty installation, the patient was exposed to low levels of carbon monoxide from 2005 to 2007. The patient was diagnosed with idiopathic Parkinson’s disease in 2008, after the incident in question.

Chief Complaints

- Cognitive decline and fatigue
- Short-term memory problems
- Balance problems
- Personality changes
- Tremors
- Loss of facial expressions
- Left-sided weakness
- Vocal cord paralysis
- Difficulty concentrating, learning new things and following instructions

Brain qSPECT® Findings

Diffuse, patchy decrease in cortical activity as well as focal areas of abnormal cortical hypoperfusion in the frontal, temporal, cerebellar and occipital lobes. In addition, focal areas of abnormal subcortical hypoperfusion were noted in the pontine portion of the brainstem and the bilateral caudate nuclei. This pattern was consistent with a diffuse toxic/hypoxic encephalopathic process and the patient’s history of carbon monoxide (CO) poisoning.

Outcome

Overcame defense’s attempt to attribute all symptoms to Parkinson’s disease, which was diagnosed after the CO poisoning. CereScan differentiated the Parkinson’s from CO poisoning by 1. visualizing the signature CO poisoning pattern, 2. differentiated abnormal vocal cord paralysis (resulted from a flaccid rather than spastic paralysis), and 3. fusing the functional and structural imaging to confirm a brainstem abnormality.