

Delayed-Onset of Movement Disorder in an Individual with Anoxic Encephalopathy Due to Cardiac Arrest: A Case Report

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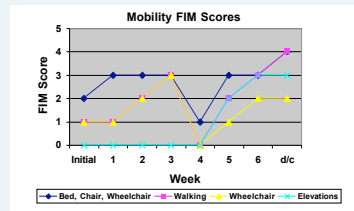
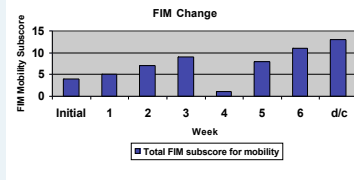
Purpose

A rare complication of anoxic injury is delayed onset or worsening of symptoms with a documented occurrence of 0.1-2.8% of all anoxic injuries.¹ While delayed onset of deficits is well researched and documented in anoxic encephalopathy due to carbon monoxide poisoning, this pattern is beginning to be recognized as a result of anoxic injuries of other mechanism, including those from childbirth, drug overdose, respiratory depression, cyanosis, shock, seizures, strangulation, perinatal hypoxia, stroke, encephalitis, and radiation necrosis.^{2,3} This report illustrates a case of anoxic encephalopathy due to cardiac arrest with symptoms consistent with delayed post-hypoxic encephalopathy.

Case Description

The patient was a 45 year-old male who was admitted following an unwitnessed cardiac arrest. This patient demonstrated consistent progress with functional mobility in physical therapy during weeks one through two. Variability in performance was noted beginning in week three with a maximal decline occurring during week four. This patient who had previously been able to ambulate more than 45 meters with moderate assistance and propel a wheelchair with complete inability to ambulate or participate in wheelchair mobility. In addition, his presentation was significant for emergence of extrapyramidal motor signs, including dyskinetic posturing and athetoid-like movements of bilateral upper extremities, whole-body akinesia, and oral preoccupation. Upon review of the literature, his symptoms and the time frame of decline were consistent with other cases of delayed post-hypoxic encephalopathy.^{2,3}

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Outcomes

Timely observations of functional decline in PT, as well as other disciplines, facilitated referral to neurology and a change in medications. This medication change coincides with resumption of trends toward improvement in functional mobility. Between weeks five and seven, this patient gained the ability to ambulate over 300 meters with close supervision and required minimal assistance for turns. In addition, he was able to ascend/descend 18 stairs with one handrail and minimal assistance, which far exceeded his optimal performance prior to decline. These improvements in activity limitation represented the difference between long term-care placement and discharge to home.

Medication Interactions

The time frame for initial presentation of delayed-onset of movement disorder in this case is consistent with time frames noted in other case reports. A review of the chart, however, suggests that medication interactions, previously unreported in the literature, may have played a role in this case.

The medical course with this patient had been complicated throughout his stay by urinary retention, which became one of the primary areas of medical intervention based on multiple failed attempts to remove the foley catheter. This patient was treated initially with Flomax, however, Urecholine (an acetylcholine agonist) was added in the third week, which coincided with the observed increase in variability of functional performance during week three followed by a dramatic decline by week four as seen on the graphs.

Medical treatment of known movement disorders frequently focuses on the balance between the acetylcholine and dopamine systems. This explains why hypokinetic movement disorders such as Parkinson's Disease is commonly managed by either dopaminergics (ex. levodopa, carbidopa) or anticholinergics (like Benztexol) Urecholine which was given to address the urinary retention in this system could have effectively decreased the amount of dopamine relative to acetylcholine and could explain the increased expression of extrapyramidal symptoms noted in this case.

A Neurology consult completed three days following initial observation of functional decline recommended that Urecholine be discontinued and an opposite medication initiated (Cogentin; an anticholinergic) which may have acted to correct the acetylcholine/dopamine balance. This final change in medication was initiated in week four, which coincides with a resumed trend of functional improvement that continued until discharge that can also be seen on the graphs.

Conclusion

A thorough search of the literature suggests that delayed-onset post-hypoxic encephalopathy may not be as rare as once believed. Multiple theories of pathomechanism to explain this phenomenon are presented in the literature. Several of these, including no "reflow" phenomenon, demyelination, metabolic toxicity, basal ganglia maturation, and trans-synaptic neuronal degeneration, as well as previously unreported medication interactions, may correlate with this case.²⁻⁹ Improved knowledge of medication actions and interactions by physical therapists may facilitate timely team communication regarding functional changes for improved overall patient care.

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