

Maduramycin Toxicity in Human: The Complete Picture

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Introduction : Maduramycin is an inophor used in poultry to prevent coccidial infection in chicks. It acts by disrupting ionic transport across cells and causes disruption of cell wall resulting in symptoms due to nerve and muscle involvement. **Methodology** : Over the last 5 years our centre has the largest experience in human toxicity due to this compound in the form of two outbreaks involving 12 persons getting affected by exposure to this compound. We reviewed the records of all these patients and are presenting our experience with the same. **Observations** : Over the last five years, a total of 12 patients have been admitted with consumption of maduramycin after making a porridge out of the toxic drug. During the 2004 episode, seven patients consumed it, two died before reaching our centre and two more died at our centre within 12 hours of admission. During the 2007 episode, five patients consumed with two dying before reaching our centre and one died at our centre within 48 hours of admission. All of them complained of profuse vomiting, intense parasthesia followed by extreme weakness in the limbs immediately after consumption of this compound. The manifestations were more pronounced in the ones consuming the largest share. Seven patients complained of respiratory distress early during the course of illness and only four of them complained of flashing lights in front of eyes. All the patients complaining of respiratory distress early in the course had a poor outcome. Sensory symptoms in the form of pin pricks and burning in the limbs were noticed in six of the eight victims reaching our centre. Five of the eight patients presented with evidence of rhabdomyolysis and established acute renal failure. Three patients presented early having dominant sensory symptoms but the neurological involvement could be documented in only one with nerve conduction study as rediculopathy. All the eight patients went on to develop rhabdomyolysis and five developed acute renal failure subsequently which recovered with conservative measures. One patient was readmitted after initial discharge following development of acute renal failure which recovered. **Conclusions** : With our experience we can postulate that the toxic manifestations of this compound are dose dependent and vary with the delay in presentation. In high dose the neuropathy is early and muscle necrosis is severe, resulting in early involvement of respiratory muscle and subsequent death. In low dose, the initial manifestations are sensory with muscle necrosis developing later in the course of illness. In these patients supportive treatment leads to a better outcome.