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157. The differential use of physostigmine by toxicologists in anticholinergic toxicity based on causative agent

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Background: The anticholinergic toxidrome is well described and relatively common, seen over 800 times by toxicologists report- ing to the ToxIC registry over a 27 month period. Physostigmine is generally regarded as the antidote to anticholinergic toxicity, yet previous studies suggest physostigmine is underutilized. Case reports have described adverse events related to physostigmine in TCA overdose, therefore, we would expect a differential use of physostigmine in TCAs versus other agents.

Methods: We retrospectively analyzed data in the ToxIC registry, a repository of data reported by bedside Medical Toxicologists in 44 institutions nationwide. We searched for patients diagnosed with an anticholinergic toxidrome, determining what agent(s) were likely causative based on "primary agent #1" as recorded by the Toxicologist. We then calculated the rate of physostigmine use in the following agents: antihistamines, antipsychotics, cyclic anti- depressants, botanicals, SSRI/SNRI, anti epileptics, benztropine, cyclobenzaprine, mixed, others and left blank. Results: 815 patients were seen by Toxicologists for anticholinergic toxidromes from January 2012 through March 2014, of which 173 (21%) received physostigmine. The rate of physostigmine use for those in which antihistamines were the primary agent was 78 of 350 (22%), 20 of 114 for antipsychotics (18%), 4 of 57 (7%) for TCAs, 23 of 44 (52%) for cyclobenzaprine, 8 of 21 (38%) for benztropine, 2 of 26 (8%) SSRI/SNRIs, 1 of 13 (8%) anti epileptics, and 6 of 17 (35%) for botanical exposures. Among 173 patients with the causative agent left blank, with mixed agents, or with agents with no identi- fied anticholinergic effects, 31 (18%) received physostigmine.

Discussion: These data suggest there is a differential use of physostigmine based on likely causative agent. Anticholinergic toxidromes caused by cyclobenzaprine overdoses were nine times more likely to receive physostigmine than those caused by a TCA. It is interesting to note the relatively low rate of physostigmine use in antihistamine overdose (22%) given its frequency. This may represent a selection bias in patients with mild toxidromes.

Conclusion: Overall, the rate of physostigmine use in anticholin- ergic toxidromes was lower than we expected. When stratified by causative agent, it appears physostigmine was used more readily for agents that exert a pure anticholinergic effect such as cycloben- zaprine, benztropine, and botanicals; whereas agents with multiple actions (antipsychotics), mixed ingestions, or tricyclic antidepres- sants were less likely to receive physostigmine.