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THE ROLE OF CARDIOPULMONARY BYPASS IN TRICYCLIC ANTIDEPRESSANT OVERDOSE :

A Case Report

Aurangzaib Durrani Mohammad Haider Ayub

We describe a case of a 26 year old male patient with Amitriptyline poisoning. He was having malignant idio-ventricular rhythms, unresponsive to treatment. He was treated with Cardiopulmonary bypass support at our unit with initial good results.

A 26 year old man was rushed to hospital after having collapsed on the street. He had an empty bottle of 120 tablets of amitripty line of 25 mg each on him, which had been recently prescribed and he smelt of alcohol. He had a history of deliberate drug overdose previously as well, so it was presumed that he had overdosed himself again. He was seen at the scene by a GP where he was found to have a SVT of 130/min. When he was brought to A&E he had a SVT with broadening QRS complexes. The patient was brought in unconscious with a Glasgow Coma Score of 3 and dilated pupils, he had been intubated and ventilated on way to hospital. Arterial blood gases were within normal limits and there was no electrolyte imbalance, gastric washout was done and activated charcoal instilled. Soon thereafter the patient went into VT and then VF. He did not respond to Lignocaine and DC shock and became asystolic. During all this episode the patient had 25 minutes of CPR in A&E and when it became apparent that this was proving to be a futile exercise, a decision was taken to put the patient on cardiopulmonary bypass for a period of support. He was transferred to cardiac theatres from there and crashed on to bypass following which support was given for 2 hours. During this period of support his ECG slowly started normalizing. No haemofiltration was done on bypass. He was weaned successfully off bypass after two hours on moderate doses of adrenaline and nor adrenaline with a systolic blood pressure of 100 mmHg and a pulse of 145/min. The patient came off bypass in sinus rhythm, the QRS complexes were broad initially off bypass but slowly narrowed down in ITU. In ITU his haemodynamic status improved and he was initially weaned off his inotropes quite quickly. He however did not show any neurological improvement. In fact he started presenting a picture of autonomic disturbance. He became hypothermic and polyuric. The patient never regained consciousness and died still intubated on the fifth day from cerebral coning.

DISCUSSION

Which two years of Kuhns' [1975] original account of the usefulness of imipramine as an antidepressant, came the first report of its' adverse effects in overdose[2]. Today these drugs are one of the most common causes of drug overdosage, because they are prescribed to group of patients in which suicide rate is high. Tricyclic antidepressant poisoning causes some very serious symptoms which include coma, convulsions, respiratory depression, shock and a wide variety of ECG abnormalities. They affect various systems of the body, but here we will discuss only those which were responsible for the acute unstable condition of our patient. A dose of 100 tablets of 25 mg. is probably lethal [1], which is about the amount that our patient probably took.

Tricyclic antidepressants are highly concentrated in the myocardium and the myocardial toxic effects are not proportional to the blood levels [1]. The ECG changes reflect a conduction block distal to the A-V level and hence broadening of the QRS complex. Usually there is SVT, but there can also be a variety of ventricular rhythms as seen in our patient. The cardiac effects of these drugs could also be due to their anticholinergic effects and hence the rationale of using physostigmine in poisoning due to these drugs. Recently the cardiovascular effects of these drugs have been attributed to their quinidine like myocardial de-

* *Address for correspondence:*
Department of Cardiology and Cardiac Surgery,
NICVD
Karachi-Pakistan

pressant effects [1]. Coma is also an accompaniment of tricyclic antidepressant poisoning, although usually of a shorter duration than seen in our patient as is also respiratory depression requiring intubation and ventilation. Various antiarrhythmics have been used in the treatment of tricyclic poisoning eg. Lignocaine, verapamil or Carbocromene [1,2,3]. Physostigmine has also been used successfully but can have unwanted side effects [2]. It has also been suggested that alkalinization of the blood to a pH of 7.50-7.55 using sodium bicarbonate 8.4% or hyperventilation is also helpful in overcoming the cardiovascular symptoms of poisoning because this binds more free drug to the plasma proteins [2,4]. Finally there is experimental evidence that cardiopulmonary bypass can be used successfully to deal with the cardiovascular and ECG effects of tricyclic antidepressant poisoning [5]. The CNS and respiratory symptoms usually require supportive treatment and other treatments such as gastric washout and activated charcoal instillation can be given. A detail of these treatments can be found in any toxicology textbook. Haemofiltration is not considered very successful because most of the drug in the system is bound to plasma proteins[2]. Cardiopulmonary bypass no doubt treated the potentially lethal cardiovascular symptoms of our patient but it seems that he eventually succumbed to cerebral injury incurred either at the time of asystole and resuscitation or then due to the poisoning itself.

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