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CARDIOGENIC SHOCK WITH IMIPRAMINE

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SUMMARY

A 35 years old physically healthy individual, being treated for depression with imipramine and electroplexy, developed cardiogenic shock which was managed successfully with inotropic support. The case is presented to highlight a rare and potentially fatal side effect of tricyclic anti-depressants.

The concentration of orally administered tricyclic antidepressants in heart muscle is 40-200 times higher than in plasma. Hence, tricyclic antidepressants have a number of cardiovascular effects of clinical significance including palpitations, tachycardia, orthostatic hypotension, arrhythmias and electrocardiographic abnormalities in the form of prolonged PR and QT intervals, depressed ST segment and flattened T waves. Precipitation of congestive heart failure and sudden death have been reported as rare complications (Hollister, 1978). Nevertheless, mortality is greater than in patients treated with electroconvulsive therapy, especially in elderly males not treated with adequate doses (Avery and Winokur, 1976). Tricyclic antidepressants in low doses have an antiarrhythmic action on the heart, but higher doses may become arrhythmogenic (Vale and Meredith, 1984). We report a case who developed cardiogenic shock following imipramine.

Case Report

A 35 year old unmarried Sikh male was hospitalised with the complaints of weakness, bodyache, feeling of sadness, disinterest in work, poor appetite, loss of weight and disturbed sleep with early morning awakening of gradual onset over the past 3 months.

There was past history of depression treated with antidepressant drugs and electroconvulsive thearpy (ECT) about one year back. He was the youngest of 6 sibs, from a low socioeconomic rural family. His father was an alcoholic indulging in wife-beating frequently. One of his brothers, who had been treated for depression in mental hospital, is an alcoholic and abuses cannabis. As a child, patient was neglected and unhappy, physically weak and frequently ill. He studied upto class two. He got enrolled voluntarily in military and apparently did well in his job.

Physical examination revealed no abnormality. Psychiatric evaluation showed depressed facies, psychomotor retardation, low tone speech, mood of anxiety and depression with fleeting suicidal rumination and feeling of worthlessness in a clear sensorium. Relevant investigations including haemogram, urine analysis, ECG, X-Ray skull and fundoscopy were normal.

The patient was diagnosed to have a recurrence of his depressive illness and was treated with Tab. Imipramine hydrochloride 75 mg/day in divided doses, gradually increasing to 150 mg/day, Tab. Chlordiazepoxide 30 mg/day in divided doses, alongwith psychotherapy and other supportive measures. Due to suicidal rumination he

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was also given six bi-weekly electroconvulsive therapies. He responded gradually, depression became less, suicidal ideas disappeared, sleep normalised, appetite improved and he gained weight.

After about 30 days of starting antidepressants and 36 hours after his last ECT, the patient suddenly complained of weakness and giddiness without pain in chest, nausea or vomiting. On examination he was afebrile with tachycardia (140/min, regular, low volume), tachypnea (resp rate 26/min), hypotension (BP 80/60mm of Hg) and cold clammy extremities. Systemic examination revealed no abnormalities. Electrocardiogram showed normal sinus rhythm, rate 140/min., generalized low voltage complexes and staircase ascent of ST segments. Over the next few hours he developed features of cardiogenic shock with impaired mentation, cold periphery, tachycardia, persistant hypotension and oligoanuria. Serial ECGs did not reveal any changes of IHD nor was there any rise of serum transminase levels. Haemogram, urine analysis, liver function tests, blood sugar, blood urea, serum creatinine and serum electrolytes were all within normal limits.

He was managed with inotropic support with dopamine drip and was required to be maintained on the same for six days before his cardiovascular status stabilized, urine output was restored and ECG had normalised.

Discussion

The therapeutic dose of imipramine is 100-300 mg/day in divided doses. Our patient was recieving 150 mg/day in divided doses, administered under direct supervision of nursing staff, and so accidental/-deliberate ingestion of excessive medication was ruled out. Pre-existing underlying ischemic heart disease and arrhythmias were adequately ruled out. The clinical profile, serial ECGs and laboratory parameters also did not suggest any cardiac illness. Therefore, the possibility of myocardial failure due to direct depressant effect of tricyclic antidepressant a rare cause was considered likely. Concurrent administration of ECT increases the intracellular levels of imipramine which may result in quinidine like myocardial depression. The chances of such depressive action of imipramine on heart increases with increasing number of ECTs. But in our patient the myocardial failure occured about 36 hours after the last ECT. Therefore, the cardiogenic shock is unlikely to be due to the combined effect of ECT and imipramine.

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