Problems in an Unconscious Patient Who Had Taken Lysergic Acid Diethylamide

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The psychological effects of lysergic acid diethylamide (L.S.D.) were discovered in 1943 by Hofmann. Nausea is common and vomiting rare (Hoffer, 1965) after small doses, and overdosage in animals results in death due to respiratory failure. The site or sites of action of L.S.D. are not known, though it produces sympathomimetic effects.

CASE REPORT

A 17-year-old boy was admitted unconscious to hospital at 9.30 a.m. on 25 September 1967. On examination he was cyanosed; respirations were gasping and infrequent, with diffuse crepitations throughout both lungs. His pupils were dilated and fixed, B.P. 120/75, pulse 80, arm reflexes present and equal, leg reflexes absent, and there were no plantar responses. There was no reaction to painful stimulus. Glycosuria + + + +.

The history was of his having taken L.S.D., diazepam, and ?barbiturates. He was given aminophylline and frusemide intravenously, and a drip set up. At 11.30 a.m. his pulmonary ventilation remained clinically inadequate, and when endotracheal intubation was resisted 50 mg. of suxamethonium chloride was given. He immediately went into cardiac arrest (11.45 a.m.), but after external cardiac massage, intubation, and intravenous bicarbonate his heart started to beat spontaneously at 11.52 a.m. He was put on a respirator, but at 12.45 p.m. he sat up, pulled out the endotracheal tube, for one minute respired 18 times unaided, and then returned, comatose, to infrequent gasping respirations. During further intubation he vomited undigested food, and laryngoscopic examination revealed extensive inhalation of vomit. Hydrocortisone bronchial lavage was performed and intravenous hydrocortisone and penicillin were started. At 2.45 p.m. his arterial P_{O_2} was 42 mm. Hg and P_{CO_2} 50 mm. Hg. At 3.30 p.m. gastric lavage was performed and much undigested food obtained. After this he maintained an adequate minute-volume unaided. There was still no response to painful stimuli, and plantar responses were absent. At 4.30 p.m. he suddenly became fully conscious, asked where he was, and vomited; blood sugar was 362 mg./100 ml. At 4.35 p.m. he returned to deep coma.

Chest x-ray examination showed bilateral pulmonary oedema, compatible with Mendelson's syndrome, and collapse of the right lower lobe. At 5.45 p.m. bronchoscopy was performed, much solid matter being aspirated from the right lower lobe bronchus. At 10 p.m. his plantar responses were flexor. Between the bronchoscopy and this time he had three further episodes of lucidity and vomited during one of them. At 10.30 p.m. his B.P. was 85, he was curarized and put on the respirator. Methedrine (methylanphetamine) was added to the drip, maintaining his systolic B.P. between 110 and 75 until morning.

On examination the next day his temperature was 100.2° F. (37.9° C.), pulse 100, B.P. 110/65, and crepitations were heard throughout both lungs. Chest x-ray film showed no change. After being taken off the respirator he became markedly cyanosed when not inspiring O_2 -enriched air. The intravenous therapy and hydrocortisone were stopped on his return to a continuous drowsy consciousness. On 29 September penicillin was stopped and tetracycline given because of a growth of tetracycline-sensitive coliforms in the sputum; arterial P_{O_2} 54 mm. Hg, P_{CO_2} 44 mm. Hg. On 2 October the P_{O_2} was 98 mm. and P_{CO_2} was 38 mm. By 5 October, after intensive physiotherapy, the right lung signs had disappeared, and on the 9th the chest x-ray picture was clear.

Urea and electrolytes were within normal limits throughout. No methaemoglobin was detected spectroscopically (28 September).

The initial W.B.C. count was 25,000/cu. mm., E.S.R., 3 mm./hour, the blood film showing marked neutrophilia, with myelocytes and metamyelocytes. On 26 September the W.B.C. was 5,900/cu. mm., E.S.R. 10 mm./hour, and no primitive cells were seen.

He had taken about 350 µg. of L.S.D. at 2 p.m. on 24 September and had lunch at 2.30 p.m., the L.S.D. not apparently having any effect on his perception. At 6 p.m. he had taken 40 mg. of diazepam and "a large quantity" of methadone hydrochloride linctus. He went to bed at 2.30 a.m. on the 25th, having smoked hashish at intervals throughout the day. The last thing he remembered was at about 6.30 p.m., and the next thing he remembered was "waking up" on the morning of the 26th.

This boy had taken many drugs in various combinations during the previous three years—L.S.D. 40 times and up to 1,000 µg. in one dose—with no ill effect. He was discharged from hospital, quite well, on 10 October.

DISCUSSION

Firstly, what was the cause of the coma and bizarre changes of consciousness? All the drugs taken on 24 September could in adequate dosage produce coma, but the time between ingestion and presentation and the small doses concerned incriminate L.S.D., which has been widely reported to produce a variety of changes in quality of consciousness. A not dissimilar case was reported by Milman (1967). A non-psychotic 5-year-old girl accidentally ingested 100 µg. of L.S.D. and for four hours afterwards was alternately screaming and silent, and when silent was motionless and unresponsive. It is supposed that in the above case the cocktail of drugs resulted in a prolonged state of deep coma relieved briefly and periodically by the "break-through" stimulatory effect of L.S.D.

The initial respiratory failure is difficult to account for. Respiratory failure due to L.S.D. in animals occurs only with comparatively high doses. The patient had taken methadone many times in the past, so tolerance can be presumed. During the period of coma it was difficult to decide on the management of the patient's respiratory function in view of the sudden changes of consciousness and hence respiratory ability. The possibility of inhalation of vomit before admission might have contributed to the initial intense cyanosis and chest signs. The relation of the suxamethonium to the cardiac arrest is probably coincidental with chronic anoxia and the simultaneous vagal reflexes resulting from a difficult intubation.

The sympathomimetic intestinal effect of L.S.D. presumably caused retention of food in the stomach for 22 hours, and peripheral adrenaline release the temporary neutrophilia, leukaemoid reaction, and high blood sugar with glycosuria.

This case is reported to draw attention once again to the idiosyncratic psychological and physical ways in which L.S.D. intoxication may present.

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