less readily considered. Furthermore, the fact that the fundal appearances of CMV retinitis so resemble malignant retinal deposits, particularly in a primed situation, as in this case, is likely to confuse. Reported cases of CMV retinitis mostly concern renal transplant recipients. The ophthalmic appearances in this case, we now realise, are very similar to published descriptions.³ The CAT-scan appearances, which have not been previously described, are of interest.

Interpretation of CSF cytocentrifuge preparations can be very difficult. In retrospect we realise that the cells we saw were reactive lymphocytes and not malignant cells. CMV may cause encephalitis.⁴ Possibly our treatment, which would have produced further immunosuppression, was causally related to the proliferation of the lesions. Corticosteroids enhance CMV infection in mice.⁵ Local radiotherapy clearly failed to halt the progression of disease. There is no effective treatment known for CMV disease, but had the true diagnosis been considered in this patient possibly management would have been different.

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¹ Smith, M E, *Archives of Ophthalmology*, 1964, **72**, 44.

² McElwain, T J, *et al*, *British Journal of Cancer*, 1977, **36**, 276.

³ de Venecia, G, *et al*, *Archives of Ophthalmology*, 1971, **86**, 44.

⁴ Dorfman, L J, *Neurology*, 1973, **23**, 136.

⁵ Henson, D, *et al*, *American Journal of Pathology*, 1967, **51**, 1001.

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Repeated self-poisoning with paracetamol

Although repetitive self-poisoning is common, the case described here is probably unique.

Case report

From November 1972 to September 1978 a 26-year-old single man was admitted with 82 episodes of self-poisoning, 31 of which involved paracetamol.

Eighteen episodes of paracetamol poisoning in one patient

Treatment	Ingestion-treatment interval (h)	Admission plasma paracetamol (mg/l)	Ingestion-admission interval (h)	4-h plasma paracetamol* (mg/l)	Maximum aspartate or alanine aminotransferase (U/l)	Maximum bilirubin (μmol/l)	Maximum prothrombin time ratio
Supportive		133	3-8	126	19	14	1-2
Supportive		104	16	308	>3000	54	1-9
Supportive		153	12	376	1152	37	1-8
Supportive		149	18	421	4800	145	2-9
Supportive		146	18	461	9120	247	4-0
Cysteamine	6-2	228	5	266	196	22	1-4
Cysteamine	7-5	258	6-5	356	91	15	1-2
Cysteamine	8-8	258	7	406	179	24	1-4
Cysteamine	8-0	191	6-5	402	49	17	1-3
Cysteamine	11-8	211	9-5	397	474	17	1-4
Methionine	6-5	218	4-5	227	43	12	1-4
Cysteine	8-2	245	6	351	24	14	1-3
N-acetylcysteine	10-5	200	8-5	350	860	75	1-6
N-acetylcysteine	10-0	229	8-2	407	270	27	1-4
N-acetylcysteine	?	242	?	?	114	36	1-4
N-acetylcysteine	6-0	249	4-8	267	47	14	1-6
N-acetylcysteine	15-5	133	13-5	288	9075	146	3-2
N-acetylcysteine	20-8	145	19-5	431	10000	252	>3-4

*Extrapolated values: see reference 2.

Conversion: SI to traditional units—Bilirubin: 1 μmol/l = 0.058 mg/100 ml.

Other drugs included barbiturates, codeine and derivatives, benzodiazepines, and tricyclic antidepressants, which he obtained without apparent difficulty despite his known exceptional history of drug overdosage. He had a disturbed, unhappy upbringing and had been expelled from both the Danish Merchant Navy and the British Army. The usual psychiatric diagnosis was psychopathic personality disorder. His self-destructive behaviour was not averted by multiple psychiatric admissions except during an 11-month period in Carstairs State Mental Hospital in 1976-7. It was considered that he could not properly be detained there longer under the Mental Health Act.

The first paracetamol overdose was taken in July 1974. Poisoning was usually severe with plasma paracetamol concentrations far above the "treatment" line¹ and recovery of up to 20 g of drug in the urine despite late admissions and incomplete collections. He knew that treatment after 10-12 hours was useless and often deliberately misled us over the times of ingestion. Details of treatment and laboratory investigations for 18 admissions are shown in the table. He was treated with intravenous cysteamine, L-methionine, L-cysteine, and N-acetylcysteine¹⁻³ on 13 admissions and never suffered severe liver damage (aminotransferases >1000 U/l) when these agents were started within 12 hours of ingestion. Severe liver damage occurred at least seven times, however, when he presented late, and transient hepatic encephalopathy developed once. Repeated tests for hepatitis B surface antigen and antibody were negative, and a recent barium swallow and intravenous pyelogram were normal.

Percutaneous liver biopsies were performed after the 16th and 31st overdoses of paracetamol. The first specimen showed some fibrosis without inflammatory reaction, but the second, carried out during recovery from an episode of severe liver damage, showed considerable disturbance of architecture, fibrous extension of portal triads, and fibrous septa with a suggestion of regeneration and progression towards cirrhosis. There was also marked cholestasis.

Comment

There seems little doubt that multiple episodes of severe liver damage were prevented by early treatment with protective agents. Nevertheless, severe damage occurred several times. The latest liver biopsy specimen suggested developing cirrhosis, which, despite intermittent heavy alcohol consumption, could probably be attributed to paracetamol hepatotoxicity. He remains a burdensome enigma to the medical, psychiatric, and nursing staff (from whom he has perhaps received care and attention he has not otherwise known). Opinions are divided concerning his future management, but the likelihood of suicide is high. His latest admission followed the discovery of Weedol (paraquat) and he now has extensive pulmonary damage.

¹ Prescott, L F, *et al*, *Lancet*, 1976, **2**, 109.

² Prescott, L F, *et al*, *Lancet*, 1977, **2**, 432.

³ Prescott, L F, *Health Bulletin*, 1978, **36**, 204.

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