PHENSIC ADDICTION

by

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Phensic tablets have not contained phenacetin since 1966 (1976). Phenacetin was first introduced in 1887 but it was not until 1953 that suspicion was aroused that it might cause renal damage (Lawrence, 1973). There is only circumstantial evidence for this, but it has become so strong that in the United States of America it is compulsory for a warning label to be displayed on all preparations sold to the public. In Sweden it has to be sold on prescription only, and in Great Britain "The Medicines (Phenacetin Prohibition) Order 1974" prohibited the sale of phenacetin-containing compounds except on prescription, because, if used regularly, it could cause renal damage (Prescribers Journal, 1974). Currently, the tablet of Phensic contains aspirin 380 mgm, salicylamide 30 mgm, caffeine 16 mgm (Martindale, 1972).

A CASE REPORT

A 37-year-old married woman, who worked in a bakery, presented with a history of severe headaches and inexhaustible energy. She was happily married with one daughter, aged 5 years. After careful questioning she reluctantly admitted that she had been taking an average of 14 tablets of Phensic per day for 6 years. She looked healthy, appeared happy but agitated. The heart was normal, pulse 82 and regular, and the blood pressure was 110/70mm/Hg. All other systems including optic discs were normal. She was taking Ovulan 50.

Investigations

Blood count, thyroid function, sedimentation rate, plasma protein levels and urine analyses were normal. Serum bilirubin 54.4. umol/1 (normal range 1-17) and liver enzymes including alkaline phosphatase 886 IU/1 (normal range 115-320), asparate transaminase 307 IU/1 (normal range 5-40), alamine transaminase 605 IU/1 (normal range 5-50), gamma glutmyl transpepitase 140 IU/1 (normal range 5-50)10-45), cholinesterase 2220 IU/1 (normal range 2000-500) were greatly elevated. The blood urea was 12 mmol/1 (normal range 2.3-6.6).

(Dr. J. N. Brown) "There is no intra-renal calcification. The kidneys are normal in size and outline. There is no opacification of the renal papillae throughout, due to contrast in the distal tubule. This (pyelotubular reflux) can be seen in normal people, but it is unusual to be seen to this extent and particularly in the film without compression. It is felt that the appearances described are likely to be a result of her phenacetin intake, and probably reflects some change in the renal papillae prior to the necrosis of a fully developed case."

Progress

The patient was warned of the damage done to kidneys and liver, and of the dangers if the addiction was allowed to continue. She was given diazepam 5 mgm three times a day for the expected withdrawal symptoms and fluazepam 30 mgm for the insomnia. She was seen regularly so that the withdrawal period was closely scrutinised, and she was extremely co-operative. She made excellent progress and one month later the liver function tests had returned to normal and the blood urea was 4.6 mmol/1. On the advice of the consultant radiologist an intravenous pyelogram was carried out after six months and he reported that there was no change in the appearance of the renal papillae. The patient is still being reviewed after nine months and remains well and free from addiction.

COMMENT

Phenacetin was introduced in 1887 and until 1953 it was not thought to have any serious effects. Since then many observers have tended to incriminate it as causing renal papillary necrosis as well as certain blood dyscrasias. All the evidence so far tends to be circumstantial because in animal studies phenacetin has not produced the renal changes that have supposedly been found in man (Strimer and Morin, 1975. This difficulty in proving that phenacetin was toxic was due to the fact that some phenacetin was contained in a large number of analgesic preparations but now that it has been taken out of Phensic since 1966 the reason for this patient's liver and renal damage must be sought elsewhere. D'Arcy and Griffin (1972) state that it is clear that there is an association between papillary necrosis and heavy consumption of preparations containing not only phenacetin but also salicylates. Salicylates cause renal irritations and cells, casts and albumin can appear in the urine. Prescott (1968) has reviewed salicylate toxicity and has stated that impaired renal function, obliguria and anuria with renal tubular necrosis may occur with overdosage of salicylates. Nanra and Kincaid-Smith (1970) reported that nearly half the rats fed with aspirins and aspirin-containing compounds developed papillary necrosis in 20 weeks.

There is strong circumstantial evidence to suggest that this patient suffered renal damage by prolonged ingestion of aspirin, and when this was discontinued the blood urea reverted to normal although the radiological picture has not yet returned. Her withhdrawal symptoms could be assumed to be due to the cessation of the caffeine intake. It is difficult to know whether the liver damage was due to salicylate or caffeine, but it returned to normal very quickly after stopping the Phensic. The further precautions to be taken in this case would be to ensure that this patient should not take any analgesic compound as further serious and indeed fatal results could ensue.

This case is reported to draw attention to the dangers of prolonged ingestion of a simple compound "Phensic", containing salicylates and caffeine.

ACKNOWLEDGMENTS

I wish to thank Miss Margaret Hammond, Professor P. F. D'Arcy, Professor P. C. Elmes, Dr. T. C. Dale, Dr. J. N. Brown and Dr. R. A. Neely for their help in the preparation of this paper.

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