Common Errors in the Management of Poisoning

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Self-poisoning is a very common cause of admission to hospital. Its commonness and the medical triviality of the majority of cases can lead to a casual approach to management. In order that this paper may be a realistic critique of current practice I have based it on a number of studies in which case notes of patients admitted to hospital in Birmingham and the West Midlands were examined. In no case was review of a patient’s notes refused, and the co-operation of all consultants involved is gratefully acknowledged.

History

Patients who have taken an overdose are usually admitted at the time of crisis. There is no time for the story to be adjusted into a bland and unrevealing fiction. The unvarnished truth can often be obtained but if the opportunity is missed the information may be irretrievably lost.

To assess the adequacy of history recording two sets of case notes were examined (Table 1). These consisted of a series of 51 seriously poisoned patients admitted to the West Midlands during 1976 and who subsequently died, and an unselected series of 105 self-poisoned patients admitted to a District General Hospital.

Table 1. Documentation.

|                | Serious Poisoning (n = 51) | Mild Poisoning (n = 105) |
|----------------|---------------------------|--------------------------|
| Type of drug   | %                         | %                        |
| Informant      | 100                       | 100                      |
| Quantity       | 76                        | 72                       |
| Time taken     | 74                        | 78                       |
| Previous psychiatric history or previous history of overdose | 63 | 72 |

The nature of the substance taken was recorded in all cases and was the basis for inclusion in the series. Other important information such as informant, the quantity of drug taken, the time that it was taken, previous history of overdose and previous psychiatric history was noted in around 75 per cent of cases. This was equally true of patients who were seriously poisoned and of patients who were trivially poisoned and able to give an accurate history.

Investigation

The drugs implicated in a case of poisoning, as indicated by the history, often bear little relationship to what is found in the urine of poisoned patients. In order to quantitate this claim a drug history was obtained from 151 consecutive self-poisoned patients. It was shown that in only 45 per cent of patients did the history tally exactly, on a qualitative basis, with what was found by exhaustive laboratory investigation of the patients’ urine (Table 2). Most of the discrepancy was due to exaggeration of the event but a significant group understated the number of substances taken. As a consequence, the physician might be expected to request the laboratory to confirm the diagnosis of poisoning. In the series of 51 patients subsequently dying in hospital 43 survived for at least six hours and there was ample opportunity to institute investigation. In only 35 per cent of this subgroup was there any laboratory confirmation of the presenting history of poisoning. Confirmation was obtained in 84 per cent of cases by the forensic pathologists. Perhaps it would not matter if confirmation of the drug history by laboratory investigation did not influence further management of the patient.

In a consecutive series of 81 seriously poisoned patients, that is patients who required active or supportive treatment, admitted to the West Midlands Regional Poisoning Treatment Centre, an accurate knowledge of the toxicological results led to a major change in management in 10 per cent compared with that which
was instituted on the basis of the history alone (Table 3). These changes in management included institution of cardiac monitoring in patients who turned out to have been poisoned by tricyclic antidepressants, forced alkaline diuresis in patients in whom no history of salicylate poisoning was available, cysteamine where no history of paracetamol poisoning was obtained and the use of Fuller's Earth in paraquat poisoning which was denied by the patient.

Table 3. Laboratory findings which contradicted clinical history in severely poisoned patients. (81 patients investigated.)

| Unexpected Finding                              | Number |
|-------------------------------------------------|--------|
| Salicylate present in significant amounts        | 2      |
| Paracetamol present in significant amounts       | 1      |
| Paraquat present                                 | 1      |
| Tricyclic antidepressant present but barbiturate claimed | 2 |
| Phenobarbitone claimed but short-acting barbiturate found | 2 |

General Treatment

The criteria for performing a stomach washout is clearly laid down by a number of authorities[1]. All seems to be quite clear but the application of these criteria lead to very different implementation rates for the procedure. For example, in the West Midlands it is 52 per cent but in Newcastle 78 per cent[2]. This may be partly due to different estimates of the unreliability of the history offered by the patient but there is no excuse for carrying out a stomach washout when there is little reason to think that the patient has taken anything other than a benzodiazepine drug. A stomach washout should not be performed as a punitive measure, as there is no evidence that it acts as a deterrent. The technique is well documented[1] and is usually regarded as safe, but complications of this procedure contributed to death in 8 of the 22 patients who were washed out in the series of 51 patients dying in hospital in the West Midlands.

It is generally said that a stomach washout following corrosive poisoning is contra-indicated[3] because of the risk of perforation of the stomach. Paraquat is a strong corrosive and stomach washout is invariably recommended in this type of poisoning. Though there have been cases of aspiration of stomach contents into the lungs, very few cases of perforation have been reported. By and large, there seems to be less risk in washing out corrosive substances than in leaving these substances to destroy the stomach wall.

Antidotes

Naloxone is used as an antagonist of morphine and heroin; it is also an effective antagonist of dextropropoxyphene, a constituent of Distalgesic, of diphenoxylate, a constituent of Lomotil and of pentazocine but, unfortunately, its use is ignored. In a series of 18 cases of Distalgesic poisoning in which there was severe depression of the level of consciousness, only six were given any opiate antagonist.

Another widely used antidote is physostigmine, a cholinesterase inhibitor, which is used in tricyclic and antihistamine overdosage to reverse coma. There is evidence that physostigmine does this but it also precipitates fits[4] and possibly cardiac arrhythmias. There is no evidence that physostigmine improves the morbidity in severely poisoned patients and some evidence that it increases mortality in experimental animals[5]. The only possible indication for its use is in the management of refractory cardiac arrhythmias when all else has failed.

Specific Drugs

Hypnotics

Patients severely poisoned with barbiturates or other hypnotics and who arrive in hospital without brain damage should recover without complications. Standard treatment is supportive and, if the drug is excreted unchanged in the urine in significant quantities, a forced alkaline diuresis is indicated. However, despite the claims for success of these methods[6], the mortality from barbiturate and hypnotic poisoning remains high and in the West Midlands in 1976 accounted for 15 of 51 hospital deaths from self-poisoning. The cause of death in 10 of the 15 patients was respiratory complications, which usually occurred many hours after the admission to hospital.

In the middle and late 1960s there was a consensus against the use of excessive quantities of fluid in patients who had taken short-acting barbiturates[6], but in long-acting barbiturate poisoning forced alkaline diuresis was advocated. However, these deeply comatose patients have depressed myocardiums and may develop increased pulmonary capillary leak and 'shocked' lungs. It is not surprising that pulmonary oedema and infection should be a major cause of death. Measures other than forced diuresis should be considered for the elimination of these drugs and hence reduction of the duration of coma. Haemoperfusion rapidly extracts the drug and reverses coma and is indicated in patients who are grade 4 unconscious and fail to improve with supportive treatment over a 12 hour period[7].

Aspirin Poisoning

The typical adult patient with aspirin poisoning is conscious, sweating, hyperventilating and complaining of tinnitus, though some patients, especially in the early stages of poisoning, may have significant blood levels but be asymptomatic. In severe poisoning it is not uncommon for patients to present with coma[8]. In a series of 25 deaths due to salicylate poisoning alone, 18 had a gross disturbance of the level of consciousness. Because severe aspirin poisoning is not recognised as a cause of coma, treatment may be delayed. Patients with severe aspirin poisoning are also prone to the respiratory complications
of forced alkaline diuresis. It is claimed that even in the
absence of shock and coma there is an increased
pulmonary capillary fluid leak leading to a lesion similar
to 'shocked' lung[9]. Diuretics such as frusemide may
improve pulmonary oedema but also compete with
aspirin for transport into the proximal renal tubular
lumen, thus delaying excretion. In these circumstances
the acid-base status must be assessed and corrected and
other methods of removing aspirin such as haemoper-
fusion, considered. The other major criticism of
management is the lack of urgency in instituting
treatment. The mode of death is often sudden and
deterioration is unpredictable; it would seem reasonable
that treatment should take place as quickly as possible.

Tricyclic Antidepressants

Tricyclic antidepressant drug poisoning is a common
problem accounting for approximately 12 per cent of all
self-poisonings admitted to hospital and about the same
proportion of deaths. A frequent cause of death is
refractory cardiac arrhythmia and it is customary to
maintain all cases on cardiac monitors. However, there is
a suggestion that only those who are severely ill at some
stage after ingestion (at least grade III comatose) develop
the fatal complications. There is also some evidence that
cardiac dysrhythmias are drug level dependent[10]. On
the other hand, death may occur long after significant
clinical improvement in conscious level has occurred. In
view of the number of cases of tricyclic poisoning ad-
mitted to hospital, selection of cases for cardiac
monitoring may be a problem. Perhaps there is a ten-
dency to monitor too many trivially poisoned patients but
not to monitor for a sufficient length of time those who
are seriously poisoned.

Conclusion

The most common and disturbing deficiency in the
management of poisoned patients is the lack of interest
and care betrayed by physicians and their juniors. In-
vestigation is frequently incomplete. There is particular
need for improvement in emergency procedures such as
stomach washout and the use of antidotes. Technique
and application of supportive treatment are generally
well understood and efficiently applied. Because of its
rarity, treatment of serious poisoning, particularly cases
due to aspirin and hypnotics, could be improved.

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