PAPERS AND ORIGINALS

Non-accidental poisoning: an extended syndrome of child abuse

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Summary

Six cases of persistent non-accidental poisoning of children by their parents are reported. Certain features may draw attention to the diagnosis, particularly bizarre symptoms and signs with no apparent pathological explanation, and toxicological analysis should be carried out to obtain rapid confirmation of the diagnosis. The underlying disorder may include marital conflict, over-involvement between parent and child, or drug abuse in the parents. A suggested plan of action for managing this problem is outlined.

Introduction

Studies of the battered-child syndrome have concentrated on physical violence, nutritional neglect, and emotional deprivation to which children, usually under the age of 3 years, have been subjected by their patients. The syndrome may, however, be extended to include a broader spectrum of abuse, ranging from child murder and physical injuries sustained by older children to the less obviously parentally inflicted child morbidity and mortality resulting from non-accidental poisoning. In their original description of the syndrome, Kempe *et al*[1] noted: "In an occasional case the parent may also have assaulted the child by administering an overdose of a drug." Weston[2] suggested that when child abuse is suspected at necropsy appropriate toxicological investigations should be undertaken to exclude poisoning, and Lansky[3] described a case in which it was thought that the mother had attempted to attack the father by poisoning their child. In the past three years six cases of non-accidental poisoning of children have come to the attention of the Hospital for Sick Children (HSC) and the poisons unit at Guy's Hospital. This manifestation of child abuse may be commoner than previously supposed, and these cases are reported here to emphasise the need to consider and exclude this diagnosis when confronted with otherwise unexplained symptoms in a child.

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Case 1

A breast-fed two-month-old girl who had had an exchange transfusion for rhesus haemolytic disease was admitted to hospital with a history given by her mother, a trained children's nurse, of anorexia, stridor, and dehydration. A respiratory infection was diagnosed and the serum electrolytes were estimated in view of the history. Serum sodium was 174 mmol(mEq)/l; other serum electrolytes and blood urea were normal. Urine sodium was 320 mmol/l. After rapid recovery and return to normal of her serum and urine sodium concentrations she was discharged for outpatient follow-up. Two further admissions within two months with similar symptoms and biochemical abnormalities prompted extensive investigation of her sodium and water handling, which were found to be normal. Mother's breast milk was found to contain extraordinarily high sodium concentrations, and she volunteered that she often had attacks of "salt sweats." Her sweat electrolytes, however, were normal. After transfer to a low-salt, cows' milk feed the child remained well initially but was then re-admitted with an identical episode, and, after yet another "attack" in hospital, she was transferred to the HSC.

At the HSC regular investigations were instituted, including daily measurement of plasma electrolytes, osmolality, and urea and 12-hour measurements of urinary electrolytes and osmolality. These and all other investigations, including plasma calcium, urine creatinine, albumin and lysozyme clearances, intravenous pyelography, and a water deprivation test, showed no abnormality. On the ninth day mother was persuaded to become resident in the baby's cubicle, and next morning she announced that her baby was developing an

attack. Physical examination showed nothing abnormal. Over the next 24 hours the child gained 500 g and her plasma sodium increased from 135 to 144 mmol/l, urine osmolality from 200 to 760 mmol-(mOsm)/kg, and urine sodium excretion from 2 to 21 mmol/kg/24 h. Mother then had to return home unexpectedly and the child's biochemical values quickly reverted to normal. A negative sodium balance of 160 mmol was calculated (estimated total body sodium 240 mmol). It was concluded that mother had added about 6 g of salt (less than one teaspoonful) to the child's feeds.

During interviews mother was told that her baby had been receiving extra salt in her feeds but she did not volunteer that she had been responsible. Further inquiry showed that she was socially isolated, lacked family support, and, after the death of her adoptive father, had had depressive episodes necessitating inpatient treatment. Although not overtly depressed she agreed to seek further psychiatric help at home, and the child was transferred to the care of the local paediatrician as an inpatient while mother attended a psychiatrist. The child was later discharged home and seen weekly at the hospital.

After two further admissions because of abscesses, attributed by mother to heel pricks and intramuscular injections, the child was admitted moribund at the age of 1 year. Serum electrolytes were: sodium 200 mmol/l, chloride 170 mmol(mEq)/l, and potassium 4·6 mmol(mEq)/l; blood urea was 7 mmol/l (42 mg/100 ml). Necropsy showed a massive gastric ulcer 7 by 3 cm having the appearance of a Curling's ulcer, the rest of the mucosa being "hypertrophied" with areas of extreme reddening. Histology showed an absence of gastric mucosa, which was replaced by superficial blood vessels, slough, and inflammatory cells. The lungs were oedematous and exuded thin frothy pus, histological changes being consistent with the inhalation of foreign material.

Case 2

The second child of a 27-year-old woman was under supervision at the HSC because of the sudden death of his brother at the age of 2 years with hypernatraemia and a sagittal sinus thrombosis after a prolonged illness with non-specific symptoms. At the age of 4 years he began to have attacks of sweating, dizziness, and pallor and of falling to the ground limp but rousable. Mother, an orderly in a geriatric unit, insisted that the attacks were hypoglycaemic and that they responded to oral glucose; however, no abnormality of glucose metabolism was found. Urine chromatography showed excess sucrose, and mother was suspected of adding sucrose to the child's urine.

By the age of 7 years mother's longstanding over-protectiveness had reduced the child to a state of dependence and withdrawal. Admission to hospital was precipitated by his arriving at the clinic curled up in a push-chair, in which he had been staying day and night. Mother insisted on being resident in the ward with him, seeming to foster his total dependence on her, carrying him around on her hip, performing all his toiletry, and sleeping by his bedside, for five weeks. She reluctantly accepted psychiatric interviews. After the child's discharge the attacks recurred and evidence was obtained of electrolyte disturbance, with, on different occasions, plasma sodium 112 mmol/l, plasma potassium 3·1 mmol/l, and flattened T waves on the electrocardiogram. Surreptitious administration of diuretics was suspected, and urine specimens were found to contain frusemide and chlorthalidone on separate occasions.

The child was admitted to the psychiatric unit at the HSC under a place of safety order. His state of withdrawal improved but he became preoccupied with being poisoned and attacked, especially during mother's visits, although he was distressed by her enforced separation. As the parents refused further psychiatric help for themselves the child was taken into local authority care.

Case 3

This patient was the third child of a 21-year-old woman, who had sought but later declined termination of the pregnancy. He was born by caesarean section after premature onset of labour and developed respiratory distress. Mother did not see him until he was 2 weeks old. When he was a year old father left the family after a cooking accident in which the patient's sister's hand was burnt. Mother responded by taking an overdose of chlordiazepoxide, and for the next six months received psychiatric care as an inpatient.

At the age of 2 years the child was admitted to hospital ataxic and confused. The admitting doctor's suggestion of an overdose of adult tablets was rejected when the child was readmitted with shaking and drowsiness, for which phenytoin was prescribed. Episodes of drowsiness and ataxia continued to occur but further investigations at the HSC showed nothing abnormal, even on an occasion when he was brought back unconscious from a walk with mother.

Mother developed multiple sclerosis, requiring several hospital admissions, during which the child was looked after by grandmother and suffered no further attacks. The attacks recurred after mother's discharge, when her treatment included amylobarbitone. When the child was aged 6 years his attacks became frequent and severe. He was admitted four times in one month, recovering spontaneously on each occasion. Barbiturate poisoning was considered, and plasma analysis at the Poisons Unit showed the presence of amylobarbitone and quinalbarbitone, constituents of Tuinal, which mother had been prescribed after grandmother's recent death.

The child was referred back to the HSC where an episode of, nystagmus, ataxia, and unconsciousness occurred after mother had bathed him, and again plasma analysis showed evidence of Tuinal ingestion. It was concluded that mother had been giving the child barbiturates, probably from the onset of his symptoms four years previously. Before she could be confronted with this she was admitted to hospital for an episode of multiple sclerosis with hysterical overlay. When she recovered she tried to discharge the child from hospital. A place of safety order was obtained. Mother then acknowledged that she must have given the child barbiturates and asked for psychiatric help. After several months in the inpatient psychiatric unit the child returned home under the supervision of social services.

Case 4

A 7-year-old boy had been attending his local paediatrician because of episodes of unsteadiness and falling dating from a febrile illness two years previously. The only abnormality was a moderate excess of fast activity in his electroencephalogram (EEG). He was admitted unconscious to the HSC. During play at school he had fallen and banged his forehead, and later in the morning he had become unsteady and drowsy and mother was called to take him home. In the afternoon he had become progressively more drowsy. On admission he was unresponsive to painful stimuli and generally hypotonic with upgoing plantar responses. A forehead bruise was thought insufficient to account for his unconsciousness. Skull *x*-ray appearances and an EMI scan were normal. Twelve hours after admission he was fully conscious and examination showed no abnormality apart from poor balance. EEG showed excess fast activity, suggesting drug intoxication.

Over the next 48 hours the patient had two further episodes of drowsiness and unsteadiness. For five days a watch was kept to ensure that no drugs were administered. The EEG returned to normal and he had no attacks. Supervision was intentionally relaxed for an afternoon when mother was visiting, and by evening he was again drowsy and ataxic. EEG showed recurrence of fast activity, and blood and urine were found to contain methaqualone (a constituent of Mandrax). Mother was told that her son had been receiving Mandrax tablets, which she initially denied, later agreeing that she must have administered them. She then precipitated her own admission to hospital for a postponed hysterectomy. Psychiatric interviews subsequently showed that she had been severely depressed with retardation, depersonalisation, and suicidal impulses involving herself and her son and that she had been taking Mandrax. She agreed to inpatient psychiatric treatment and the child was discharged to his aunt's care.

Case 5

A girl aged 15 months was admitted to hospital with a history of fever and cyanosis. Mother, an unstable personality with a history of overdoses, was aged 22; father was 64. A stepbrother aged 3 years had recently been taken into care. The girl was listless and her throat inflamed. She developed stridor, was treated with ampicillin, and was discharged fully recovered. Ten hours later she was readmitted because mother reported "breathing difficulties." She was drowsy, with stridor and constricted pupils. Opiate poisoning was suspected, although mother denied giving any medicines. Dihydrocodeine was identified in her urine, the police were informed, and the drug was found at home. The child remained unwell for a week. Inquiries showed that when she returned home after her first admission mother and father quarrelled and one of them administered the dihydrocodeine in Guinness. She was secured under a place of safety order.

Case 6

A girl aged 2 years was brought to hospital unconscious and collapsed. The night before she had had a sweating attack and, later, twitching of the arms. Eighteen months previously she had been admitted in hypoglycaemic coma, phenformin being identified in the stomach aspirate. Six months later she was again hypoglycaemic and three months after that had an epileptic fit. Father was diabetic and mother epileptic. A sister had died after falling from a window, and a brother had died in hypoglycaemic coma.

On examination no apex beat could be detected. Despite initially successful resuscitation, including intravenous glucose, the child died.

Laboratory analysis in suspected cases

When a physician suspects that drugs have been administered illicitly blood and urine should be collected at the earliest opportunity for laboratory analysis. The laboratory should be consulted by telephone for guidance on the appropriate specimens and transport arrangements. When anticonvulsant, hypnotic, or psychotropic drugs are suspected, 50 ml of urine (unpreserved), 10 ml lithium heparin blood, and stomach contents are appropriate. Larger samples may be required for investigation of other drugs; a continuing urine collection is advised.

For screening, the urine sample is divided into five aliquots for various colour tests and chromatographic techniques, as reported elsewhere.[4] Table I lists some common drugs that may be detected in each fraction. The method is not totally comprehensive, as several compounds—for example, monoamine oxidase inhibitors—may not be detectable even after an overdose. The blood sample is usually reserved for quantitative measurements of drugs detected by the screening tests.

Any information about the child's presentation or drugs available in the household may help the laboratory in designing suitable analyses. The diuretic and phenformin poisonings in our series were detected by specific tests as a result of a clinical suspicion of the type of drug involved.

Discussion

DIAGNOSIS

Regrettably the first requirement for diagnosis is a low threshold of suspicion. Bizarre symptoms and signs with no apparent pathological explanation should lead to the consideration of pharmacological causes. In the case of the child with barbiturate poisoning ingestion of adult tablets was suggested from the physical signs at the time of first presentation. In the cases of salt and diuretic poisoning, otherwise inexplicable electrolyte values provided the first indication of the cause of the illnesses. A full drug history of all members of the child's household may show the availability of appropriate drugs. A history of parental drug abuse or overdose should heighten suspicion.

Features characteristic of the non-accidental injury syndrome[5] may be present. The prenatal history may contain evidence of maternal ambivalence about the pregnancy, as in the case of the child receiving barbiturates. Neonatal separation may have occurred, as in this instance because of respiratory distress, or, as in the case of the salt poisoning, because of haemolytic disease. There may be a history of unstable family relationships or of parental psychiatric disturbance. In the cases of hypoglycaemic and diuretic poisoning a sibling had died under unusual circumstances.

Examination of the temporal relationships between the episodes and presence of the parent was responsible for the diagnosis in a case of chloral poisoning reported by Lansky[3]; in our case of barbiturate poisoning the child was symptom-free when mother was away from home. In both instances and in the cases of salt and methaqualone poisoning "attacks" while mother was in hospital with the child provided further evidence of her responsibility. Alternatively symptoms may reappear when the child returns home.

These features, considered to have been helpful in reaching the diagnosis, are correlated in table II with the cases in which they were noted.

MANAGEMENT

When the diagnosis is suspected the child should be admitted to hospital and an investigation planned, as follows:

Full history including all previous episodes of illness
Family history:
(1) Unusual illness or hospital admission of siblings
(2) Psychiatric illness of parents
(3) All drugs available
Careful recording of all physical findings on examination
Immediate collection of blood for biochemical analysis and blood and urine for toxicological analysis
Electroencephalography
Planned collection and recording of further data
Record of parents' visits and activities with child
Involvement of general practitioner, health visitor, hospital and local social workers

TABLE I—*Drugs that may be found in each aliquot of urine*

Spot tests	Acid + neutral	Basic + neutral	Benzodiazepines	Amphetamines + miscellaneous drugs
Salicylates	Barbiturates	Tricyclic antidepressants	Nitrazepam	Amphetamine
Paracetamol	Phenytoin	Most antihistamines	Chlordiazepoxide	Fenfluramine
Chloral	Glutethimide	Phenothiazines	Diazepam	Methylamphetamine
Etchlorvynol	Methaqualone	Most narcotics	Medazepam	Pargyline
Imipramine group	Meprobamate	Methaqualone	Oxazepam	Mephentermine
Phenothiazines	Phenazone	Meprobamate	Lorazepam	Chlorphentermine
		Chlormethiazole edisylate	Flurazepam	Phenmetrazine
				Chlormethiazole edisylate
				Diethylpropion
				Methyprylone

TABLE II—*Features in seven cases of poisoning that helped in reaching the diagnosis*

	Salt	Diuretic	Tuinal	Mandrax	Dihydro-codeine	Phen-formin	Chloral*
Inexplicable symptoms, signs, or biochemical values	+	+	+	+	+	+	+
Neurological presentation (fits, faints, ataxia)		+	+	+	+	+	+
Drug known to be available to parent			+			+	+
Parental drug overdose or abuse			+	+	+		
Psychosocial stress in family	+	+	+	+	+		+
Suspicious illness, injury, or death in sibling		+	+			+	
Episodic illness	+	+	+	+		+	+
Episodes related to parent's visit	+		+	+			+
Recurrence after discharge home	+	+	+		+	+	
Drug detected in urine, blood, or gastric contents		+	+	+	+	+	

* Case reported by Lansky.[3]

The child's medical history may contain evidence of previously unrecognised episodes. The social history, obtained when possible by a social worker, may throw light on the home environment. Carefully kept inpatient records are essential, not only because of possible legal proceedings but also to convince all parties that there is no other explanation for the illness. Drugs administered while the child was at home may be detected in specimens obtained on admission, and further specimens should be collected at once if symptoms recur. An EEG may be helpful when there are neurological symptoms or signs and may need to be repeated urgently. A full record of the parents' visits should be correlated with episodes of illness and detection of drug administration.

When non-accidental poisoning has been proved the following plan of action may be adopted:

(1) Case conference
(2) Consideration of place of safety order
(3) Frank explanation of cause of illness to both parents
(4) Suggestion of source of drugs
(5) Recommendation for psychiatric investigation of whole family
(6) Place of safety order if voluntary separation from child is not accepted
(7) Later decision about long-term plans after court hearing or further case conference

Local social workers should be aware of the possible need for a place of safety order; this may be needed before diagnosis if the parents try to discharge the child before investigations are complete. When confronted with the diagnosis the parent may find it impossible to admit responsibility; this difficulty should be acknowledged but psychiatric referral should still be urged. The case histories show the necessity for the child to be separated from the parent either voluntarily or compulsorily until there is confidence that further episodes of poisoning will not occur.

PSYCHIATRIC ASPECTS

Specific psychiatric findings may account for this form of child abuse. Lansky and Erikson,[6] in describing the case of chloral poisoning, showed that marital conflict may lead to an attack by one spouse on a child perceived as unduly favoured by the other, the resulting illness of the child achieving a restoration of the marital relationship at the child's expense. Similar patterns of disturbed family relationships were found in several of our cases. Non-accidental poisoning may create a situation that enables parents to escape from their own physical or psychological illnesses or marital or social problems. Thus the mother of the child given barbiturates seemed to be projecting her own anxieties, and later her disease, on to her child. This was shown by remarks that she was reported to have made during the child's illness, telling others that her son had leukaemia, saying that she hoped that the child did not have her disease (multiple sclerosis) and remarking that the child himself expected to die before he was 10 and wanted to join his grandmother. By creating an actual illness by administering her drugs to the child and so projecting her poor prognosis on to the child she achieved denial of, and dissociation from, her anxieties concerning her illness.

At the same time the relationship between parent and child is characterised by longstanding over-involvement with each other, resulting in failure of differentiation emotionally into two individuals. Hence if the parent experiences conflicting emotions the child, felt as part of her, may become the object of intense love or hate. Such ambivalent feelings could be related to severe depression, as in the child poisoned by Mandrax, or unresolved grief reaction, as in the salt poisoning case. An alternation of blame and attack with overprotective care resulting from these feelings results in the paradox of the parent administering the drug and yet being concerned at the child's consequent symptoms. When the parent has a history of drug abuse or overdose, as in the cases of the children poisoned by barbiturate and Mandrax, child poisoning may be seen as a form of suicidal gesture allied to self-poisoning, the identification of the parent with the child allowing the parent to use the child's symptoms as a cry for help, representing a wish for parent and child to escape together from an unbearable situation.

The children in turn tend to protect the parent and may collude in the administration of the drug. Separation from the parent may result in great sadness and intense anger directed at those responsible for the separation. The abnormal parent-child relationship may be related to abnormal relationships of the abusive parent to their own parents, as was seen with the children poisoned by salt and barbiturates. The pattern of disturbed family relationships is completed by a marital partner who remains peripheral to the over-close relationship between the child and the abusive parent.

When confronted with the diagnosis the parent may find it impossible to accept responsibility. The mothers of the children poisoned by Mandrax and barbiturate both precipitated their own hospital admissions to escape the consequences of their child's diagnosis. Yet treatment of the underlying parental psychological disorder depends on the parents' acknowledgment of their responsibility for the poisoning, and hence of their abnormal relationship with the child. Restoration of normal family relationships may then make possible the child's reunion with the family.

We and other clinicians diagnosing this form of child abuse almost invariably found it difficult to be objective in assessing the significance of the known facts relating to these children. This emotional barrier has been recognised as a problem in the diagnosis of conventional forms of child abuse and must be overcome if such children are to be adequately protected. Once the validity of the diagnosis had been accepted in some cases subsequent cases were easier to assess objectively.

We thank the paediatricians who referred these children and the paediatricians and psychiatrists under whose care they and their parents were admitted for their encouragement and help in the preparation of this paper.

Requests for reprints should be sent to Dr Arnon Bentovim.

ADDENDUM—After this report had been prepared a 4-year-old girl was admitted to the HSC unconscious, having fits, and almost apnoeic. She had been on the "at-risk" register after a scald and black eyes at the age of 2 months, and her brother had recently been taken into care because of abuse. Plasma sodium was 188 mmol/l, and urinary sodium 330 mmol/l. The parents subsequently admitted having added salt to her diet for six months to prevent her being greedy and to induce vomiting. This case resembles those described by Pickel *et al*,[7] in which hypernatraemia resulted from water deprivation by parents for reasons such as prevention of enuresis.

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