Leaders

Confession of ignorance of causation in coroners' necropsies – a common problem?

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Introduction

The failure of a postmortem examination to demonstrate a cause of death in a significant proportion of deaths of young soldiers was documented many years ago.¹ It has been apparent that pathologists may experience difficulty from coroners or colleagues when acknowledging that a cause of death remains unascertained after detailed postmortem examination and may be tempted to ascribe a false cause of death.²

Recent newspaper reports of inquests suggest that, perhaps because of the analogy drawn with sudden infant death syndrome—sudden unexpected death syndrome³—, such difficulty may be decreasing. No such difficulty has been experienced in what is now the Wales Institute of Forensic Medicine for many years, the concept of “the obscure autopsy” having been well established by Knight.⁴ This leader describes that experience and the philosophy underlying usage of the terms “unascertained” or “indeterminate” as applied to the cause of death.

Methods

All postmortem reports issued between 1984 and 1993 by the Wales Institute of Forensic Medicine were examined to determine the pathologist's final opinion as to the cause of death. In those cases where this was “unascertained”, “indeterminate” or, indeed, where no cause of death was offered, more detailed analysis was carried out. To avoid the semantic difficulty posed by cases of sudden infant death syndrome, only deaths in subjects over two years of age were included. Details of age, sex, medical history, drug therapy, postmortem findings, and results of toxicological analysis (where performed) were studied. Causes of death and verdicts recorded at inquest were obtained from the coroners involved.

Cases were separated into two groups depending upon whether there had been police investigation in addition to enquiry by the coroner's office (suspicious cases) or not (routine cases).

Results

ROUTINE CASES

Analysis of the routine cases from 1984 to 1993 revealed 48 deaths where the cause was not determined at postmortem examination out of a total of 5840 (less than 1%). There were 25 males and 23 females and cases were found in all decades of life save the first.

Six (13%) subjects showed advanced decomposition, two of these being discovered in water. Six other subjects—in two of which there was a history of epilepsy—were discovered in water, giving a total of eight (17%).

A clinical history or pathological evidence of natural disease was present in 21 (44%) subjects: idiopathic epilepsy in six, coronary artery atherosclerosis in six, asthma in two, diabetes mellitus in two, and single cases of diabetes mellitus with asthma and epilepsy, tuberous sclerosis with asthma, superior mesenteric artery atherosclerosis, orbital myositis, and left ventricular hypertrophy. Six of those subjects with a history of epilepsy were on medication: in four the blood concentrations were considered sub-therapeutic, in one subject the blood concentration was within the therapeutic range and in another decomposition was so advanced that a suitable sample could not be obtained.

All of the subjects with coronary artery atherosclerosis had a significant degree of luminal obstruction, but none showed acute changes within the atheromatous plaque or myocardium and other features—recovery of the decomposed body from water, a history of alcohol abuse, a plastic bag enveloping the head, a clinical history of hypothermia, or circumstantial evidence of the possibility of overdose of digoxin—made it impossible for the pathologist to determine the precise role of atherosclerosis in the death. Two of the subjects with diabetes mellitus were suspected of having hypoglycaemic coma and one ketoacidotic coma; in the latter, where there was also a history of epilepsy, there was evidence that biochemical analyses similar to those obtained from postmortem samples had been recorded in life while the subject had been so well as to refuse admission, without fatal consequence. None of the cases with a history of asthma showed pathological evidence of mucus plugging of airways or other respiratory complications.

Alcohol was detected in 13 (27%) subjects; in four the level was above 80 mg/dl (range 90–271 mg/dl). Nine (19%) subjects had a history of alcohol abuse; alcohol was detected in five but in none was the level above 40 mg/dl. Fatty change of the liver was noted in six of the subjects with a history of alcohol abuse.

Drugs were detected in 14 (29%) subjects: all had been prescribed but in four (involving digoxin, dextromoramide, lofepramine, and

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oxpentifylline, respectively) the possibility of an overdose was raised in the history. None of the concentrations was within a fatal range but in the case involving lofepramine the concentration was 10 times greater than the upper limit of the therapeutic range; it was not clear what contribution, if any, this played in the death, given the presence of mesenteric ischaemia. In two subjects with a history of drug addiction, no drug was detected; because of hepatitis C infection in one of these subjects only external examination and sampling of blood were performed.

There was no analysis for alcohol or drugs in 20 (42%) and 18 (37%) subjects, respectively.

In 37 (77%) subjects the coroner recorded an open verdict; a verdict of natural death was recorded in eight subjects with single verdicts of accidental death, suicide and unlawful killing. In those eight natural deaths there were histories of epilepsy in three, alcohol abuse in two, "chest pains" in two, and of diabetes mellitus in one. Unlawful killing was recorded on a body returned from abroad where the brain had been retained and was not available for examination in the UK. The cause of death was recorded by the coroner as diffuse axonal injury and intracerebral haemorrhage following receipt of a neuropathology report from abroad. This was one of six cases in which there was a disparity between the cause of death given by the pathologist and that recorded by the coroner. In the other five cases the causes of death recorded by the coroner were epilepsy in two and single cases of senility, plastic bag asphyxia and hypothermia, the last two attracting verdicts of suicide and accidental death, respectively.

SUSPICIOUS CASES

Analysis of the suspicious cases from 1984 to 1993 revealed 47 deaths where the cause was not determined at postmortem examination. The total number of suspicious deaths during this period is not known exactly but is estimated to be approximately 1000: "unascertained deaths" account for just under 5%. There were 28 males and 19 females and cases were found in all decades of life.

Decomposition of the body was present in 22 (47%) subjects. Fourteen (30%) of the bodies were found in or at the edge of water and nine of these were decomposed. In another case the body was so damaged by fire as to preclude proper examination. One body was exhumed after initial postmortem examination elsewhere but, given the retention of material from that first examination, changes of early putrefaction were no hindrance.

Natural disease was present in 11 (23%) subjects: nine cases of coronary artery atherosclerosis and one each of multiple sclerosis and motor neurone disease. Only one of the cases of coronary artery atherosclerosis showed evidence of an acute complication of the atherosclerotic plaque but even in that case it was not possible to comment on its significance, the body being removed from water. Of the remaining eight subjects, three were removed from water, two were victims of

assaults which could not be excluded from having caused death, one was found with a plastic bag around the head, and hypothermia or drugs could not be excluded in two.

In contrast to the routine cases, in all suspicious cases there was toxicological analysis for alcohol and drugs, unless samples could not be obtained because of decomposition.

Alcohol was detected in nine (19%) subjects, the blood level being above 80 mg/dl (range 80–338 mg/dl) in eight. Six subjects had a history of alcohol abuse and alcohol was detected in four (range 80–260 mg/dl).

Drugs were detected in seven (15%) subjects: cannabis, flunitrazepam, heroin, morphine, temazepam, diazepam, and amphetamines; more than one drug was present in two of these subjects. In all of these subjects the blood concentrations were below toxic or fatal ranges, although three subjects were said to have been drug misusers (in one case where a low level of diazepam was found it was suspected that the subject may have suffered an epileptiform seizure secondary to diazepam withdrawal). Three additional subjects were said to have misused drugs but in two decomposition was so advanced that suitable samples could not be obtained. The third was hepatitis B surface antigen positive: only external examination to exclude injury was performed.

In three subjects there was pathological evidence of head injury. One body was so badly decomposed that it could not be determined whether the injuries were antemortem or postmortem; a plastic bag around the head and a rope around the neck were additional findings. In the suspected diazepam withdrawal mentioned above the injuries were not considered by the pathologist to be fatal but the coroner recorded head injury as the cause of death. The third case is discussed later.

The cause of death recorded by the coroner differed from that given by the pathologist in seven subjects: immersion in two, drowning in two, and single cases of head injury, plastic bag asphyxia and asphyxia due to multiple sclerosis.

An open verdict was recorded in 26 cases; there were four verdicts of natural death, two of accidental death and a single verdict of misadventure. Criminal charges were proved in five cases (manslaughter in three, murder in two): inquests were not resumed. The verdict and cause of death at inquest could not be traced in nine cases.

Discussion

Common to most of these deaths is the inability of the postmortem examination to reveal sufficient objective pathological evidence to determine a cause of death. One explanation of this inability is immediately obvious: the degree of decomposition or destruction of the body is such that the remains are insufficient. That marked decomposition is itself recognised as a bar to successful examination may be demonstrated by the high proportion of decomposed bodies among the cases attracting police investigation. However, when these are removed from the total a substantial number

remains where it may be said that the cause of death is apparent from the history and the absence of 'positive' objective pathological evidence is typical (if not classic), reflecting either a well recognised true absence of findings—idiopathic epilepsy,⁵ immersion,⁶ chronic alcoholism,⁷ enveloping of the head in a plastic bag⁸ which is removed at the scene before the pathologist views the body—or a 'masking' of diagnostic findings by postmortem change—hypoglycaemia or hyperkalaemia,⁹ fresh water drowning.⁶ In such cases the pathologist may feel that the cause of death indicated by that history should be given but such a decision is unwise, given that the history may be incomplete or false, or both.¹⁰

It may be that the history is consistent with several possible scenarios, a choice between which must be overtly arbitrary: a person with a history of idiopathic epilepsy found under water in a bath. Where several verdicts at inquest are then possible (with possibly dramatically different consequences regarding life insurance) an arbitrary decision by a pathologist from the absence of findings that the underlying cause of death was idiopathic epilepsy (even though the absence of findings cannot eliminate accidental or intentional fresh water drowning or, indeed, suffocation and placing of the dead body in the bath) is again unwise and an arrogation of the function of the coroner.

To arrogate the function of a jury in a criminal trial is no less unwise and for a forensic pathologist may be detrimental to, if not totally destructive of, credibility and career. Where prosecution and conviction for murder are possible in the absence of a body,¹¹ then the absence of a cause of death after complete postmortem examination of a body should not be a bar to prosecution. It may present practical difficulty to the Crown Prosecution Service but facilitation of the legal process through simplification of a pathological problem is no part of the function of the forensic pathologist.

In one of our subjects, the situation as presented to the pathologist was that the deceased, said to have been a chronic alcoholic, had been found after a person had admitted having caused death by strangling with a sheet; the sheet had been removed during attempts at resuscitation. Postmortem examination revealed no evidence of injury to the neck, subconjunctival petechiae, which might be explicable as a result of attempted resuscitation,¹² and a concentration of alcohol within the blood of 256 mg/100 ml. The cause of death was given as 1a) Unascertained and comment made in the report that there was nothing in the findings that was inconsistent with the application of pressure to the neck by means of a soft ligature. The Crown Prosecution Service sought an opinion from another forensic pathologist who from the report of the first pathologist "... and the circumstances given ... by the Police Officers" gave the cause of death as 1a) Asphyxia due to 1b) Strangulation with a ligature. As the defendant pleaded guilty to manslaughter, a plea accepted by the prosecution, no difficulty was apparent but

where a case may be more contentious (and who is to know when a case may become contentious?) the more cautious approach of the first pathologist is preferred.

There are cases where the findings at postmortem examination are not so much absent as so plentiful as to allow several different interpretations. A person, said to have been a chronic alcoholic, was found dead in a brook at the base of a rocky slope. Postmortem examination revealed blunt head injury and a concentration of alcohol within the blood of 338 mg/100 ml but diatoms were not present in the water. No cause of death was given because of the impossibility of precise comment upon the significance in the death of any or all of these findings but several permutations were given at committal:

1a) Blunt head injury;

1a) Blunt head injury and immersion;

1a) Blunt head injury and alcohol intoxication;

1a) Blunt head injury, immersion and alcohol intoxication.

The precise cause of death was not an issue at the trial, a person being convicted, on retrial after discharge of the first jury, of manslaughter.

We consider the cause of death in such cases is best given as 1a) Indeterminate rather than 1a) Unascertained.

The above case illustrates the common problem of interpreting the significance of values obtained after toxicological analysis of samples for drugs and alcohol. With regard to alcohol, two situations are of particular interest. Firstly, in relation to acute alcohol poisoning, what is a fatal blood concentration?¹³ Many would regard a blood concentration greater than 300 mg/dl as adequate evidence of such a cause of death. However, in the chronic alcoholic blood concentrations higher than 300 mg/dl may have little effect¹⁴ and, when complicating factors such as head injury and immersion in water also exist, it is impossible to define the significance of the concentration of alcohol in the blood. Comment on its significance should not be made without knowledge of the deceased's drinking habits and even then caution is advised. Secondly, our finding that none of the nine routine cases with a history of alcohol abuse had a blood concentration above 40 mg/dl, fits well with the hypothesis that these deaths may be related to "withdrawal from alcohol". Several pathophysiological mechanisms have been postulated to explain the association between severe fatty change in the livers of alcoholics and sudden death¹⁵ but this is an area that would benefit from further study.

Such issues as 'best' evidence and appropriateness or admissibility of expert opinion based on 'hearsay' evidence may be focused in criminal trials or deaths in overtly suspicious circumstances but they are no less important—and may be more dangerous to the pathologist's self-esteem and credibility—in deaths where no suspicious circumstances are apparent. A person, said to have been terminally ill with motor neurone disease, was found dead at home; the case was referred to the coroner

because of a possibility of suicide. Postmortem examination (by a forensic pathologist sceptical of the necessity) revealed only wasting of the muscles of the hands and forearms; no cause of death was apparent. The next day, while toxicological analysis and histological studies were in preparation, another person entered a police station and admitted having assisted the deceased in taking an overdose and, when unconsciousness and death did not ensue, having placed a plastic bag around the head and held a pillow over the face. Further dissection of the face, toxicological analysis and microscopy revealed neither injury nor an obvious cause of death, although there was microscopic support for the diagnosis of motor neurone disease. The cause of death was given as 1a) Unascertained. At inquest, the pathologist accepted that the postmortem findings (or their absence) were in keeping with suffocation with a pillow but that those findings did not exclude the possibility of death being due to motor neurone disease. An open verdict was returned and a plea of guilty to attempted murder was accepted by the Crown Prosecution Service. Where there is no objective pathological evidence the pathologist must not be swayed in his or her decision-making by a statement, admission or confession where that statement, admission or confession may be open to question, or indeed withdrawn at a later date.

This case highlights a logical paradox in everyday coroners' practice. Not only does an absence of objective pathological evidence of cause of death not exclude the possibility of homicide but it must also be borne in mind that, where there are pathological findings which may be considered sufficient to be a cause of death, the exclusion of another party having brought about the death by a means which leaves no evidence—such as suffocation with a pillow—is, again, impossible. It is then only the absence of an admission which allows the pathologist to remain confident in his or her ability to reach a conclusion as to cause of death, but it is little comfort to realise that this confidence may be false.

When those deaths where there is decomposition, or a multiplicity of findings whose precise relation to the death cannot be determined, or a history which leads the pathologist to expect to find nothing, are excluded there is a residuum—seven routine cases and three suspicious cases (age range 14–85 years) where the post mortem examination yielded no pathological findings. It may be argued that this demonstrates only an inadequate investigation and we would accept that it is only in the last two years of the period studied that—for example, examination of the cardiac conduction system has been adopted as routine in such cases. What investigations are required of a pathologist faced with a 'negative coroner's necropsy'?¹⁶ In this Institute there is a move toward a protocol comprising:

- (1) dissection of the face (in situ dissection of the neck structures by the pathologist is regarded as routine in all cases);
- (2) retention and detailed dissection of the

heart, including decalcification of calcified coronary arteries, determination of weights of isolated ventricles and examination of the cardiac conduction system;

- (3) inflation of a lung with fixative before dissection;

- (4) examination of the brain only after fixation in suspension;

- (5) examination of the fixed spinal cord;

- (6) analysis of vitreous humour for urea, sodium, chloride, and ketone bodies;

- (7) retention of samples of peripheral venous blood, urine and vitreous humour for analysis for alcohol and carbon monoxide, screening for common drugs of abuse or more specific analyses as indicated by the results of further enquiries by police or coroner's office;

- (8) institution of further enquiries regarding the medical history of the deceased and other family members, with particular regard to epilepsy, diabetes mellitus and palpitations/syncope.

When all such examinations, analyses and enquiries yield no evidence of a cause of death then there is no difficulty in stating that the cause of death is unascertained. Indeed some pathologists may find no problem in giving such a cause of death without such detailed investigation (that such a possibility exists may be inferred from the description of sudden infant death syndrome as a "too facile diagnosis"¹⁷) but they lay themselves open to a charge of insufficiency of enquiry.

Despite sympathy with the arguments advanced in support of sudden unexpected death syndrome,³ we see no reason to conceal ignorance behind such a term; the pathologist should state that the cause of death is unascertained and, if required, assist the coroner and relatives by explanation of current thinking as to possible mechanisms of death and the attendant difficulties of proof of—for example, the significance of minor anatomical abnormalities in the cardiac conduction system¹⁸ or the role of higher neural centres in control of the heart.¹⁹

The finding that in 12–14% of subjects the cause of death given by the coroner differed from that provided by the pathologist emphasises the fact that responsibility for the final decision as to cause of death and verdict at inquest rests solely with the coroner. What weight of evidence is required for that decision is a matter for the individual coroner guided by interpretation of the Coroners Act 1988, the Coroners Rules 1984 and case law from the Queen's Bench Division of the High Court.

We do not consider tenable arguments that the protocol outlined above is too labour intensive for a coroner's pathologist or that the coroner system is concerned only with the exclusion of criminality rather than accuracy of certification of cause of death. Where a cause of death remains unascertained, it is impossible for the pathologist to exclude a concealed criminal act and further investigation is the responsibility of the coroner. If such a philosophy is acceptable in one coroner's jurisdiction we find it difficult to understand why it might not be acceptable in another. Proper investiga-

tion (by the pathologist and other agencies) to permit accurate certification of cause of death must be regarded as important enough to outweigh any delay in the processing of the death attendant upon those investigations: why, otherwise, does the coroner system exist? If resources for proper investigation are insufficient then that insufficiency should be demonstrated and remedied, not concealed by facile and inaccurate compromise. Understanding of how and why people die will only be advanced if ignorance is acknowledged, thus identifying fields for further study.

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