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MEDICO-LEGAL ASPECTS OF EMBOLISM WITH A CASE ILLUSTRATION

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Summary

An adult male sustained a number of stab injuries and other injuries including a fatal stab injury to the neck. There was evidence of air embolism which was considered to be a major factor causing death. The discussion is in four parts. Part I is confined to the post mortem examination. Part II relates to the medico-legal aspects of the case. Part III is a general discussion on embolism and its medico-legal significance whilst Part IV is on the medico-legal aspects of air embolism.

Keywords: Air embolism, air, amniotic fluid, bullet, fat, thrombus.

INTRODUCTION

An embolus is defined as "a detached intravascular solid, liquid or gaseous mass that is carried by the blood to a site distant from its point of origin", or more simply, "an abnormal mass of undissolved material which is transported from one part of the circulation to another". The classification can be based on its composition i.e. - (a) thrombus and clot, (b) gas -air/nitrogen, (c) fat, (d) tumour, (e) bullets, (f) amniotic fluid, (g) bone marrow, (h) others (e.g. parasites).

Emboli can be present in the lungs i.e. "pulmonary embolism" or in the tissues supplied by the "systemic circulation" i.e. systemic embolism. In any forensic discussion on embolism consideration must be given not only to the composition of the embolus but also to the circulatory system which is predominantly affected. Paradoxical embolism may also occur.

PART I: CASE REPORT

A post mortem examination was carried out on the body of G.J., an adult male of Afro-Caribbean origin at a public mortuary in the United Kingdom.

External Examination

a) Clothing

The deceased was dressed in a navy blue jumper with a zip in the front, a pair of khaki trousers held in position by a belt, underpants, a pair of white socks and a pair of white trainer shoes. The jumper, trousers and trainers were stained with blood and the soles of the trainers were also stained with blood. There were three metal rings on the right middle finger, the distal two having a stone embedded in each of them whilst there were two metal rings on the right finger, both having stones embedded in them. From the "distal" ring one stone was missing.

b) External Findings

The height of the deceased was 1.52 metre and the estimated weight was about 95.4 kg. The eyes were natural. There were no subconjunctival petechiae. The ears and mouth were normal and healthy. He had his own teeth in a good state of preservation.

Rigor mortis was established and full. Post mortem lividity was indistinct. The anal orifice was normal in appearance. There were no fissures or scars.

External Injuries

(i) Neck

a) A stab wound 37mm long was obliquely placed on the neck, 75mm to the right of the midline, 25mm above the middle third of the right collar bone. This was 1525mm (about 60 inches) from the right heel. The upper and inner end was incised whilst the opposite end was wide and shelving. The stab was directed downwards and to the left for a depth of about 135mm (about 5.5 inches) (Fig. 1).

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(ii) Trunk

b) **A** stab wound 11mm long was present on the lower left back, 85mm to the left of the midline and 1160mm from the left heel. The right margin was shelving with'a triangular area of skin sliced off at this margin. This was 16mm deep and had not entered the chest or abdominal cavities.

c) **A** stab wound 22mm long was placed on the back of the left shoulder, 195mm to the left of the midline and 80mm from the top of the left shoulder. Both medial and lateral ends appeared incised, 33mm deep and directed almost horizontally towards the right.

d) **A** stab wound 21mm long was placed on the right side of chest, 113mm to the right of the midline and 1225mm from the right heel. Both ends were incised. The stab was 49mm deep directed almost horizontally to the left entering the chest cavity through the 6th intercostal space and cutting the upper border of the 7th rib.

e) **A** stab wound 28mm long was placed on the right side of the lower chest, 115mm below injury (d) and 125mm to the right of the midline, entering the abdominal cavity through the 10th intercostal space for a depth of 110mm. This was directed backwards to the left and slightly upwards. The front end was incised and the opposite end was lacerated.

(iii) Upper Limbs

f) An incised wound 30mm long was placed on the radial aspect of the right forearm, 50mm above the level of the wrist.

g) 35mm from injury (f) on the back of the right forearm was a stab wound 25mm long, placed 55mm above the level of the wrist. The lower end was incised whilst the opposite end was fishtailed. This stab wound was 50mm deep and directed upwards (Fig. 2).

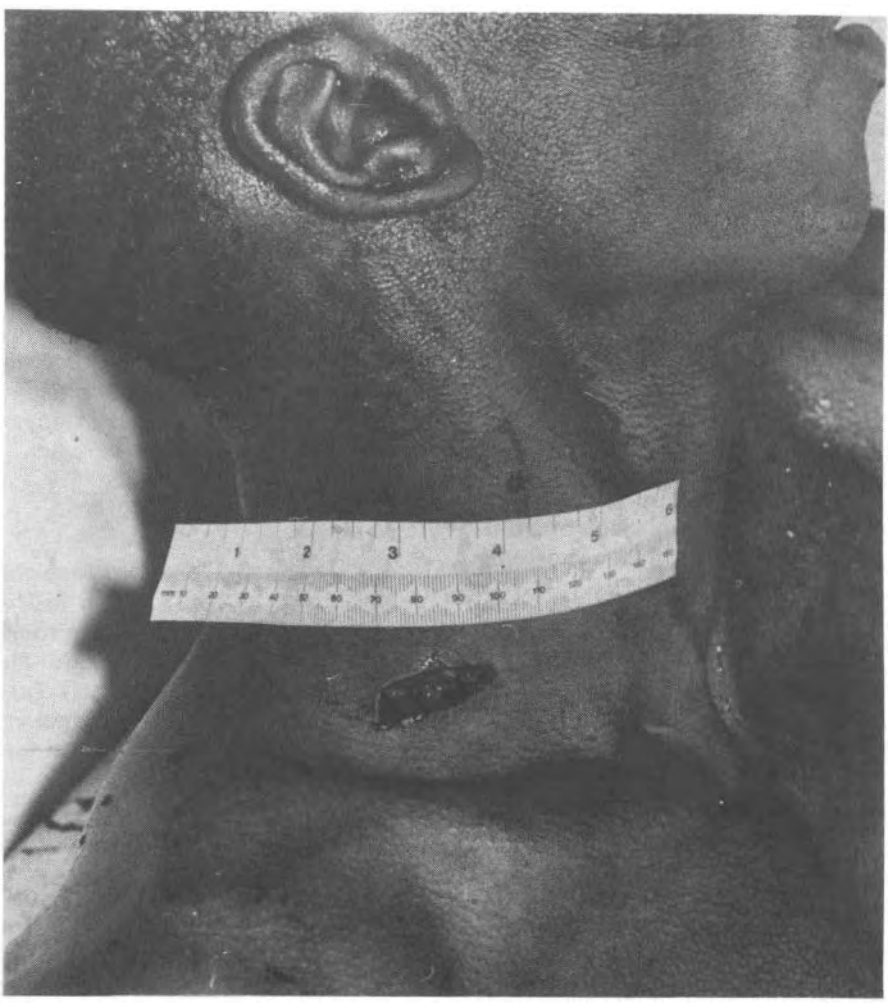

FIG. 1: Stab injury on right side of neck

h) Small abrasions raising a flap of cuticle on the dorsum of the distal interphalangeal joint of left ring, middle and index fingers were present.

(iv) Lower Limbs

i) An abrasion 15mm x 10mm was present on the outer aspect of the left knee cap extending as an interrupted abrasion 110mm x 10mm. It was vertically placed with rucking of the cuticle in an upward direction.

Internal Examination

There was no bruising of the scalp. There was infiltration of blood around external injury(a).

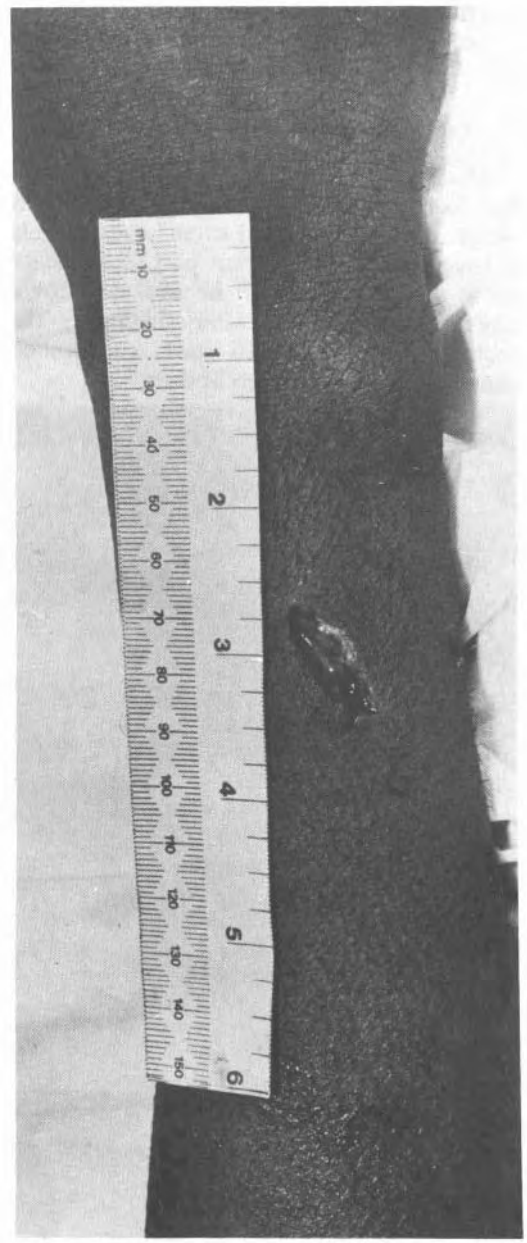

FIG. 2: A "defence" injury.

Skeletal System

There was no bony injury other than the cut to the 7th right rib.

Central Nervous System

The vessels at the base of the brain were healthy and free of any congenital abnormality. The vessels on the surface of the brain were healthy. There was no evidence of "air bubbles" in the vessels on the surface of the brain. The brain and meninges were healthy. The sinuses were clear. "Air bubbles" can occur as an artifact in the cerebral veins.

Cardiovascular System

The heart weighed 320gms and was contracted. The myocardium was healthy as were the valves, coronary arteries and aorta. Corresponding to external injury (a), the right external carotid artery was divided as was the superior vena cava. There were air bubbles in the pulmonary artery and its two branches (Fig. 3) in addition to frothy blood in the right ventricle and pulmonary artery.

Respiratory System

The trachea was divided, corresponding to external injury (a) and contained some frothy blood. The lungs were pale and showed patchy collapse with evidence of inhaled blood. The left chest cavity was free of blood whilst the right chest cavity contained about 1.5 litres of fluid and clotted blood. There was no injury to either lung.

Gastro-Intestinal System

The oesophagus was healthy. The stomach contained about 250ml. of fluid with some altered blood. The intestines were healthy but the mesentery was infiltrated with blood and extended into the right perirenal region. There was a through and through stab on the right lobe of the liver. It was 23mm long, incised at one end and fishtailed at the opposite end. The gall bladder contained bile. The bile duct was patent and the pancreas was healthy.

Genito-Urinary System

There was peri-renal infiltration of blood in relation to the right kidney (as mentioned earlier) with an 18mm long stab on the lateral border of the kidney and entering a depth of 10mm. Both kidneys were pale but otherwise healthy. The bladder contained a few drops of urine. The prostate was healthy. The stab injury to the liver and kidney corresponded to external injury (e).

The spleen was wrinkled. The thyroid and adrenals were healthy.

A total of fourteen exhibits were handed over to the exhibits officer.

Special Investigations

Microscopic examination of the brain, heart, lungs, liver, kidney, spleen, pancreas and prostate showed no evidence of natural disease. The lungs showed evidence of red blood cells in alveolar spaces confirming the naked eye impression of inhalation of blood.

The cause of death was given as:

1a. MULTIPLE STAB INJURIES.

N.B. In medico-legal work it is advisable to give an anatomical cause of death.

PART II: MEDICO-LEGAL ASPECTS OF THE CASE

Anatomical Consideration

The deceased had a number of external injuries - incised wounds (cuts), stab wounds and abrasions on the neck, trunk, upper and lower limbs associated with a number of internal injuries. All the injuries were consistent with being sustained at the same incident.

The principal injury that was responsible for death was injury (a) (Fig.1) associated with the internal injuries. This stab had divided the right external carotid artery. superior vena cava and the trachea. Stab injury (e) had entered the abdominal cavity causing a through and through stab of the liver terminating in a stab injury of the right kidney. There was evidence of infiltration of blood into the mesenteric tissues. There was no significant amount of free blood in the abdominal cavity.

The other injuries would be consistent with having been sustained in the course of a struggle and are unlikely to have been major contributory factors to his death.

Patho-Physiological and Medico-Legal Considerations

There was evidence of blood loss at the scene. In addition, there were about 1.5 litres of fluid and clotted blood in the right chest cavity. Primarily it would be reasonable to conclude that haemorrhage caused death.

There was also evidence of inhaled blood and some blood in the stomach. This aspiration was of limited extent and unlikely to have played a significant part in his death but it does indicate that he was alive for a short period of time after sustaining the neck injury and had not succumbed immediately to an air embolism.

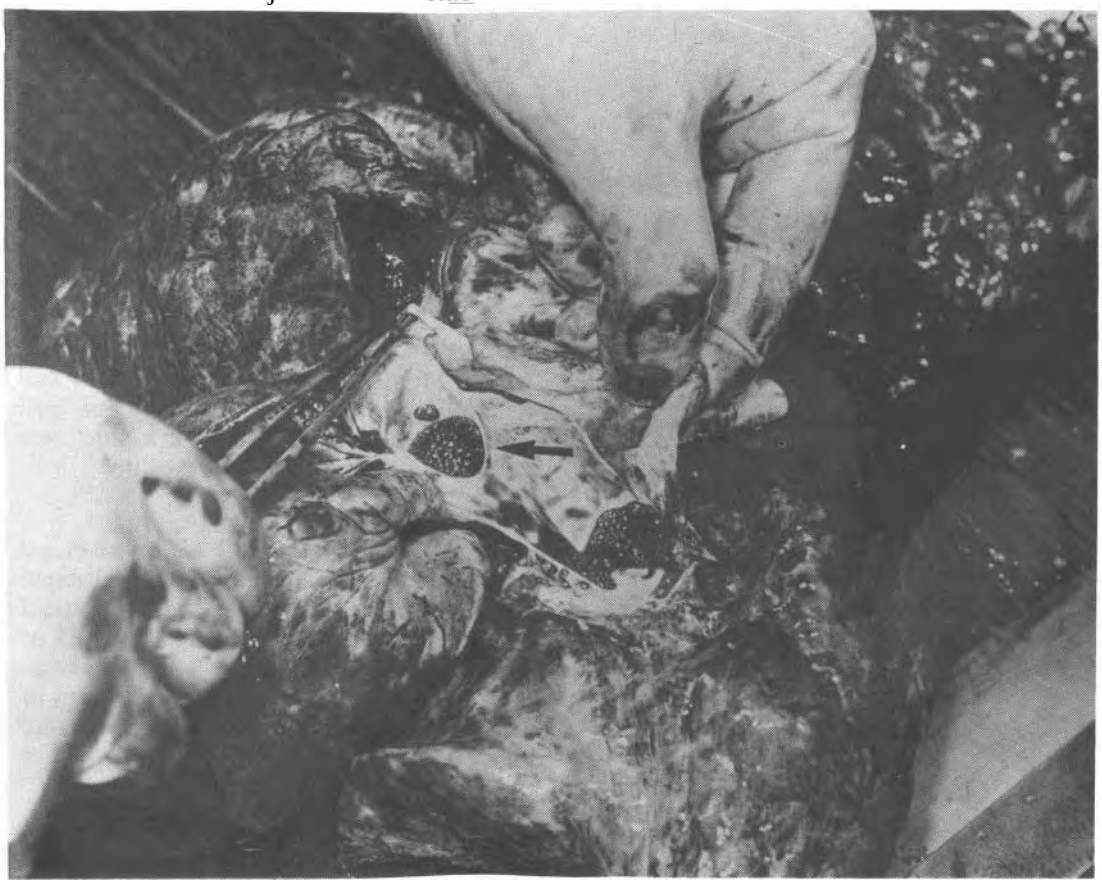

FIG. 3: Bubbles of air (black arrow) showing air embolism.

Fig. **3** clearly indicates embolism. This was an important patho-physiological mechanism that led to his death. These conclusions were confirmed at the trial where the evidence was that the deceased received the stab injury to the neck (injury a) when the deceased was seated in the car. After a short period of time, a second stabbing incident occurred a few feet away from the initial spot whereby he sustained injury (e). Thus the extra physical effort is likely to have caused aspiration of air through the superior vena cava resulting in air embolism. It is also likely that the stab injury through the liver and kidney was received after receipt of the neck injury - at the second spot of stabbing.

Collapse and death due to air embolism is usually sudden in cases of injury to the major veins of the neck, if the vein does not collapse and there is a positive pressure in the jugular veins. In this case as the vein was completely divided it is likely that the proximal end of the vein collapsed. Furthermore, as the victim was seated, the "positive" pressure of the abdominal organs on the chest is likely to negate the negative pressure in the chest and hence no suction of air occured. At the same time, haemorrhage from the divided artery and vein occurred. (There was evidence of "arterial blood spurts" on the windscreen of the car). However, when the victim was out of the car air is likely to have been sucked into lesser circulation via the superior vena cava, resulting in collapse and death. Death resulted from the combined effects of haemorrhage and air embolism.

The presence of only a small quantity of blood in the abdominal cavity and perirenal tissues indicates that this injury was sustained after the neck injury.

At the conclusion of the trial, the accused was found guilty of murder and duly sentenced.

Accident - Suicide - Homicide

Stabbing is the most common method of homicide in the United Kingdom. Self inflicted or accidental stabbing is much less frequently encountered, particularly in the neck region. Occasionally differentiation between homicidal and self inflicted cases may cause difficulties.

Accidental stab wounds from knives are rare. King' reported such a case. Penetrating injuries may occasionally be encountered in accidents involving glass, ie. falling through plate glass or mirrors. Stabbing of the neck is from most accounts an unusual mode of suicide.[2,3]. Most victims are male. Differentiation between various categories of stabbing is not difficult but on occasions difficulties in interpretation may arise. The wounds are in accessible regions of the body, however unusual may be the site(s).[4]

In homicidal stabbing wounds are frequently multiple, usually are also present on other parts of the body and are accompanied by "defence" wounds. Self-inflicted wounds frequently have accompanying tentative wounds / stabs.[3] In a study by Vanezis and West[3] 50% of 29 suicidal cases had such tentative wounds.

Assessment of the scene is vital in arriving at a conclusion and such a procedure is to be strongly recommended.

PART III: GENERAL DISCUSSION ON EMBOLISM

As indicated earlier, embolism. is defined as "an abnormal mass of undissolved material which is transported from one part of the circulation to another".[5]

(a) Thrombo-Embolism

Pulmonary Embolism

Virtually *99%* of all emboli arise from thombi. The usual source of these emboli are the deep veins of the legs in over 95% of cases. The incidence at autopsy has varied from 1% in the general population of hospital patients to 30% in patients dying after severe burns, trauma and fractures. It is the sole or major contributing cause of death in about 10% of adults dying acutely in general hospitals.[6] Pulmonary thromboembolism is not unknown among the younger age groups and in the past 20 — 25 years there has been an increased incidence among young women, which may be linked to the use of oral contraceptives.' The condition may be a fatal complication of pregnancy and can occur in either the antenatal or postnatal period.[8] Although deep veins of calf muscles are the commonest source, it is not unusual for a thrombus in pelvic veins or the prostatic venus plexus to form emboli." Usually a 20 — 45cm long clot from the femoral or popliteal vein becomes detached, enters the right side ot the heart and passes on to the pulmonary artery or its branches.

Three major influences that predispose to thrombosis are:

1. Injury to the vascular endothelium
2. Alterations in normal blood flow
3. Alterations in the blood (hypercoagulability).

Clinically, pulmonary embolism can be classed as:

(i) massive, resulting in sudden death or severe shock
(ii) silent, with little or no clinical symptoms
(iii) embolism resulting in pulmonary infarction
(iv) recurrent embolism giving rise to cor pulmonale.

Clinico-pathological correlation in these I'our categories is not at all straightforward, thus on occasion a massive embolism can be silent with the patient feeling faint or slightly breathless.[9] In fatal cases the mechanism could be either mechanical, or caused by platelet release of 5-hydroxytryptamine or thromboxane, resulting in spasm in the pulmonary vasculature.

Small septic pulmonary emboli from suppurative thrombophlebites may result in septic infarcts.

Fate of Pulmonary Emboli

When death is instantaneous, coiled thrombi are usually found lying packed in the main pulmonary artery or occluding both right and left main pulmonary arteries. If the patient survives, the vasa vasorum dilates adjacent to the point of lodgement. After about 12 hours a layer of fibrin can be seen to cover the embolus. Within 24 hours there is oedema of the media, dissolution of the elastic laminae adjacent to the dilated vasa vasorum with polymorphonuclear leucocytic infiltration of the vessel at the point of attachment. It is likely that the embolus retracts in sire. This allows blood flow around the embolus and thus may cause further embolisation peripherally. The embolus is covered with endothelium-like cells at 24 hours. After about a week, clefts lined by endothelium appear and fibrous tissue invade the thrombus firstly at its point of attachment. By about two to three weeks the thrombus is firmly adherent to the arterial wall and has undergone recanalisation with fibrous tissue surrounding new vascular channels."

Systemic Embolism

The emboli usually arise in the heart, pulmonary veins, mitral and aortic valves in infective endocarditis, left atrium in mitral stenosis and the left ventricle in myocardial infarction and some forms of cardiomyopathies.

The effects of systemic emboli are that of obstruction, a block distal to the obstruction with thrombosis and clotting beyond the obstruction. Bland emboli produce no other effects other than ischaemia or infarction whilst septic emboli give rise to secondary foci of suppuration.

Medico-Legal Considerations

Trauma with subsequent immobility such as bed rest are potent factors leading to deep vein thrombosis of the calf with subsequent pulmonary embolism. Pulmonary embolism classically occurs 10 - 14 days after injury but the range varies from 2 - 3 days up to several months."

Since pulmonary embolism is an extremely common condition and is a well recognised complication of trauma in medico-legal terms, it is vital to show that the commencement of the leg vein thrombus occurred since the injury. The criteria for "dating" depends on the histological examination of the interface between the thrombus and the vein wall in the deep vessels of the legs. In taking histological blocks, special care must be taken to preserve sections of vessel with the thrombus still in place. The histological criteria have been described by Zahni.[11] In several road accident deaths, variable legal results have emerged from virtually the same medical situations of calf vein thrombosis with fatal pulmonary embolisation.

(b) Fat Embolism

INTRODUCTION

The subject of far embolism was first described in 1862 by Zenker.[12] Fat emboli may be discovered in the lungs (pulmonary fat embolism) or in the tissues supplied by the "major" circulation (systemic fat embolism). There are two different aspects of fat embolism - the pathological finding and the clinical syndrome.

In patients who die shortly after injury, microscopic droplets of fat are found obstructing small vessels, particularly in the lungs but also sometimes in other organs. Lung embolism is physiologically found in nearly all patients whose injury include fracture of long bones. On the other hand, the clinical syndrome is observed in only certain patients with hone fractures.

The pathological findings are of forensic iriterest and in this context bone marrow embolism (i.e. the presence of recognisable particulate bone marrow in the vessels) is inseparable from fat embolism. This is commonly seen in cases of multiple fractures and multiple fracture to ribs following vigorous external cardiac massage.

ANATOMICAL CONSIDERATIONS

Pulmonary Fat Embolism

In 1928 Lehman and McNattin showed that fat embolism occured in the lungs in a variety of circumstances unrelated to injury or fracture of bones, and apparently did not contribute to the fatal outcome. They found fat embolism in the lungs in 50% of deaths from natural causes. The finding was entirely incidental and without any pathological or clinical significance." The reported incidence varies according to the source. Mason reported an incidence of 20%.[14]

The most common and most important cause of fat embolism is trauma to bone. It has been recognised after orthopaedic surgical procedures, extensive burns, inflammatory lesions in bones and adipose tissue, alcoholic fatty liver and decompression sickness.

Systemic Fat Embolism

The clinical syndrome of fat embolism may be defined as a complex alteration of homeostasis presenting clinically as acute respiratory insufficiency. The syndrome is evident in 0.5 - 2% of long bone fractures and increases to 5% - 10% in multiple fractures associated with pelvic injuries. This clinical state usually develops following a latent period after injury of some 24 hours." If the embolus passes through the pulmonary bed it reaches the brain, kidney, skin and other organs. This could result in coma within a few hours followed by death in 1 - 2 days.

As stated above, the symptoms usually appear 24 - 48 hours after injury and manifest as fever, restlessness, respiratory distress, cyanosis and cerebral symptoms (anxiety, restlesness, confusion and coma). Petechiae often appear on the chest. There is tachycardia. Occassionally, haemoptysis may occur and this may indicate heavy pulmonary involvement. Cerebral damage dominates the picture and is the principal cause of death.

SOURCES OF FAT EMBOLI

It has been the classical and natural view that ingress of local fat into the blood stream at the site of injury is the source of emboli. It also has been suggested that fat originates elsewhere than in the bone marrow or from injured tissues and plasma fat is the suggested source. Another theory is that altered conditions in the blood following injury may cause flocculations or agglutinations of chylomicrons to produce fat in the form seen as emboli. Yet another view is that fat may be mobilised from tissue as a consequence of a rise in serum lipase after injury.[16]

Originally it was believed that fat released from damaged adipose tissue (or bone marrow) was forced or aspirated into the central ends of lacerated veins. That this does not always happen is indicated by the occasional finding of organised masses of myeloid tissue in the pulmonary vessels of persons who have died after fractures. In some instances the amount of fat found in the pulmonary vascular bed is far in excess of what might plausibly be derived from the site of injury.

It has been suggested that the phenomenon of post-traumatic fat embolism was the result of a change in the droplet size of endogenous plasma lipids.

Pulmonary fat embolism occurring independent of mechanical violence may be encountered in association with such diverse conditions as diabetes mellitus, extensive cutaneous burns and decompression sickness."

DIAGNOSIS

The diagnostic criteria of fat embolism are not clearly defined. Fat embolism can be classified into three major categories: subclinical, clinically manifest but non-fatal, and fatal forms.

The gross findings at autopsy in systemic fat embolism are not specific. They consist chiefly of petechiae and minute disseminated haemorrhages in the brain and kidneys. The diagnosis rests on the identification in the lumina of blood vessels of globules of neutral fat that can be visualised by fat stains.

Whenever fat embolism results from injury to bone, the presence of bone marrow particles and bony spicules adds validity to the diagnosis.[18]

ASSOCIATION WITH BONE MARROW EMBOLISM

There is adequate clinicopathological and experimental evidence that significant pulmonary fat embolism is due almost exclusively to bony injury and that the emboli comes from the injured part. The same applies to the rapid appearance of emboli during orthopaedic procedures of a relatively localised nature or those involving rib traction. The local origin of fat is most strongly indicated by the very frequent concurrence of pulmonary bone marrow emboli, the origin of which cannot be in doubt. Bone marrow emboli were found in 81% of cases showing grade 2 or more fat embolism.[15]

MEDICO-LEGAL SIGNIFICANCE

Evidence indicates that pulmonary fat embolism and bone marrow embolism are associated with fracture (trauma to bone) and the appearance of fat and bone in the lung is dependent upon a combination of a and the time for which that circulation continued after injury.

The practical application of this concept is in determining the duration of survival/time of death after an accident where there are multiple fatalities (many members of the same family). Thus in civil litigation associated with life insurance policies and in the disposal of the estate these findings are important.[14] This opinion is expressed on the assumption that no external cardiac massage has been attempted. Resuscitation is frequently accompanied by rib fractures and if an effective circulation has been established the finding of pulmonary fat emboli should be considered as an artifact.

Amniotic Fluid Embolism

Introduction

This is a rare condition and has become a recognised hazard of labour since its clinical significance was stressed by Steiner and Lushbaugh in 1941.[19] It is a catastrophe that takes place during or just after labour and is characterised by profound shock.

If the patient survives the initial episode a severe haemorrhagic diathesis develops with serious fibrinogen depletion, probably associated with disseminated intravascular coagulation defect. The mortality is in the order of 80%.[9]

Diagnosis

It is essential that the diagnosis should be borne in mind during the examination. The order conditions that will have to be considered are - air embolism, Mendelson's syndrome, Gram negative septicaemia, pre-eclampsia, eclampsia, hypertension and disseminated intravascular coagulation. Macroscopic examination will not be sufficient and blocks of all the organs should be taken, particularly from all the lobes of the lungs to establish the diagnosis of amniotic fluid embolism.

Microscopy

The histological components of the amniotic fluid emboli are:

Epithelia1 squames
Lanugo hairs
Fatty material - vernix caseosa
Mucin and meconium
Bile pigments from the meconium.

These are best demonstrated by the method described by Attwood.[20]

Mechanism of Embolism

It is thought that the fluid enters the maternal circulation via venous channels in the uterus following strong uterine contractions. It is associated with rupture of the membranes and placental or uterine traumatic injury while the foetal head is impacted in the pelvic outlet. In this situation uterine contractions are thought to force amniotic fluid into the systemic uterine veins." Attwood by his staining technique was able to demonstrate that amniotic fluid embolism was often very extensive and support the hypothesis that it was the physical obstruction of the pulmonary circulation which caused death. This view is not universally accepted and many believe an anaphylactic reaction to be of major importance.'

(c) Bullet Embolism

Introduction

Embolism of a bullet is a rare occurrence. It should be suspected whenever a penetrating bullet wound occurs with failure to discover the bullet at post-mortem, particularly if there is no exit wound. It can occur with pellets, the result of discharge from a shot gun. Pellets from air rifles too, can embolise.

The first report of a bullet embolus is attibuted to Thomas Davis in 1934 and a review of the English language literature to January 1988 yielded 153 cases.[22]

Most bullet emboli follow the direction of the blood flow although 15% of "venous" bullets cause embolisation in a retrograde manner.[23]

Medico-Legal Considerations

The recovery of a bullet is vital in any medico-legal post-mortem examination associated with a shooting incident. This will help to identify the firearm that was responsible for the discharge of this bullet. This is accomplished by test firing of the suspect gun and comparing the marks on this bullet with the specimen bullet. Thus when the specimen bullet is recovered from the victim care must be taken not to scratch or mark the bullet.

Therefore, if bullet embolism is suspected, a total body x-ray is necessary in all instances of penetrating missile wounds, if an x-ray of the region fails to reveal the projectile in the expected region. Further, an x-ray examination should be carried out in all gunshot wounds.

(d) Miscellaneous Emboli

Emboiisation by other particulate materials have been recorded; particularly in relation to intra-venous drug abuse. viz. talc resulting in the development of non-caseating granulomata.

Tumour emboli is a recognised method of dissemination of malignant growths.

Microemboli from atheromatous plaques in the carotid arteries, from calcific valves during "open" heart surgery and parasitic embolisation produce clinical symptoms and signs.

Such cases are unlikely to be of medico-legal significance. However, with the development of selective invasive chemotherapeutic procedures, it is possible to inject a drug in a wrong vessel with disastrous results with possible medico-legal consequences.

PART IV: AIR EMBOLISM

PATHOGENESZS

Bubbles of air or gas within the circulation obstruct blood flow and damage tissues just as certainly as thrombotic masses. Air or gas may gain access to the circulation during delivery or abortion, therapeutic or criminal, when it is forced into ruptured uterine venous sinuses by powerful contractions of the uterus.

Air embolism may occur during perfomance of a pneumothorax when a large artery or vein is ruptured or entered accidentally. Similarly operations on the head and neck can result in fatal air embolism when a large vein is damaged or injured. Other therapeutic procedures, viz. mismanaged blood transfusions, during haemodialysis for renal failure, insufflation of uterine tubes and "bypass" cardiac surgery.

These bubbles of air act as physical masses. Many small bubbles may coalesce to produce frothy gasseous masses, sufficiently large to occlude a major vessel, usually in the lungs. Aggregates of larger size may become trapped in the chambers of the right heart and block the pulmonary artery.

Ingress of air at atmospheric pressure into peripheral veins is facilitated by two factors:

1. Fixation of venous walls to adjacent structures and tissues so that their lumens remain patent following their subtotal or complete transsection. Such perivenous fixation prevents collapse of the opened veins which would obstruct entry of atmospheric air. Instead what happens is that after slight initial bleeding, negative intravenous pressure permits entry of air into the patent channel.
2. The suction effect produced by thoracic respiratory excursions and cardiac action further abets ingress of air into the venous circulation.

After air has entered the venous lumen. it is transported promptly to the right atrium and ventricle where it immediately starts to exert its pernicious effects.

Disagreement exists as to the lethal malfunction which ensues. Forbes [24] states that death may be cause by mechanical interference with cardiac function, by a blockage of the pulmonary artery by forth with an arrest of the circulation in the lungs, or by an obstruction of the finer arterioles in the lungs by multiple emboli.

Some air is carried through the pulmonary arterioles and capillaries creating a minor degree of arterial air embolism. However, the lower haemodynamic pressure normally present in the lesser circulation and the rapidity with which death ensues prevents passage of significant quantities of air through the pulmonary circuit into the left atrium and ventricle for peripheral dissemination.

The rate at which air enters the systemic venous circulation is critical in fatal air embolism. As the rate of air entry increases,, survival time decreases. It has been estimated that between 100ml - 200ml of air is the minimum quantity required to kill a healthy adult, provided that air enters rapidly. If it enters slowly, larger volumes may not be fatal.

Death from air embolism, whether arising from attempted criminal abortion or therapeutic or diagnostic misadventure, usually occurs suddenly. The victim may complain of a "funny feeling" in the chest or manifest extreme dyspnoea prior to collapse. When delayed death from air embolism occurs the probability is that sufficient air did not enter the venous circulation to cause immediate death. However, when the victim moves about, the negative pressure or heart action or the combined effect of both result in sufficent air entering the venous circulation (as in the case under discussion).

If the patient survives the first 10 - 15 minutes, the danger is largely passed. Severe neurological deficits may completely disappear within hours or days, although mental disturbance may persist.

DIAGNOSIS

Large quantities of air probably in the region of 100ml are required to produce problems.[6]

As a preliminary measure Gordon et al[14] recommended radiological examinations carried out within a short period of death. They state that apart from their diagnostic value, such examinations provide a guide to the most advantageous dissection procedure to be followed. In the absence of overwhelming infection by gas forming organisms or of post mortem putrefaction with its accompanying gas formation, radiologic demonstrations of gas in the right heart chambers has specific diagnostic significance.

Gentle palpatation of the anterior chest wall of air embolism victims may elicit the characteristic crepitus of sub-cutaneous emphysema and incision of the crepitant areas permits escape of gas bubbles and frothy blood from dermal and sub-cutaneous vessels, furnishing additional evidence of the lethal mechanism.[25]

The calvarium is "opened" in the usual manner but care must be taken not to damage the cortical cerebral vessels. Interpreting the presence of air bubbles in the cerebral veins is difficult. These could be artefactual and not true embolism.'

When the neck, thorax and abdomen is opened but before the organs are eviscerated, the vessels in the neck (superior vena cava, internal jugular veins) and abdomen (inferior vena cava) are examined for the presence or absence of bubbles of air. If present these should be photographed. Again, if death is due to air embolism, examination of the right atrium and appendage, right ventricle, pulmonary conus in situ shows these structures "ballooned out". At this stage the parietal pericardium is incised and filled with water such that the pulmonary artery is below the water level, and when incised bubbles of air escape through the water (I prefer to have the photographer "shoot" as 1 cut into the pulmonary artery and demonstrate the presence of churned air bubbles in the pulmonary artery and its two main branches,. as seen in Fig: 3. The possibilty of post-mortem artefacts must be carefully excluded.

On rare occasions air may be demonstrated in the coronary vessels - air in the coronary veins indicates reflux from the right atrium via the coronary sinus and air in the coronary artery, the result of either transpulmonary migration of the gas into the left atrium or from passages of gas from right heart to left through a patent foramen ovale or another anomalous pathway.

CAISSON'S DISEASE

Caisson's disease or decompression sickness, a specialised form of gas embolism occurs in persons exposed to sudden changes in atmospheric pressure. Those at risk are scuba and deep sea divers and workers engaged in underwater tunnelling.

With shallow divers, compressed air may be used but with deeper levels, compressed mixtures of oxygen and helium are used. Whatever the gas, when it is breathed under high pressure increased amounts dissolve in the blood, tissue fluids and fat. If the individual. decompresses too rapidly, the gases come out of solution as minute bubbles. Oxygen is readily soluble but nitrogen and helium tend to persist to form gaseous emboli.within the blood vessels and tissues.

There are two types of decompression sicknesses, acute and chronic. The acute form is commonly known as "the bends" or "the chokes". The chronic form is referred to as Caisson's Disease.

A close parallel exists in the decompression sickness occurring in divers and that occurring in airmen.[26]

The hazard of rapid decompression with the occurrence of air (nitrogen/helium) embolism cannot be dissociated from the volume effects - barotrauma.

PULMONARY BAROTRAUMA

This occurs when the diver holds his breath or develops laryngeal spasms during ascent. As the atmospheric pressure falls, the lungs are distended to their full capacity. Blood is displaced from the pulmonary capillary bed with resulting hypoxia. Breaks in the continuity of the alveolar membrane occurs. Subpleural bullae form and may rupture into the pleural cavities. On reaching the surface the victim exhales, reopens the capillary bed and air may enter torn capillaries leading to systemic air embolism.

Unconsciousness and convulsions followed by paresis may occur. Air trapping is the most important factor in pulmonary barotrauma.[27]

SPINAL CORD EMBOLISM

Embolism to the spinal cord is considered to be rare. In 1967 [28] two cases of emboli to the spinal cord have been reported, one by atheromatous material and the other by fibres from the surface of a myxoma of the left auricle, both resulting in infarction of the cord.

PATHOLQGICAL *FINDINGS*

When death occurs within a few minutes, air is largely in the veins, there are no haemorrhages and the brain is normal. In a case surviving 55 hours cortical damage took the form of small perivascular foci of neuronal damage and of laminar necrosis.[29]

An accentuation of ischaemic damage along cortical arterial boundary zones after accidental air embolism in man was first described by Ameuille et al[30] and confirmed by Brion et al in 1974.[31] Thus the neuropathology of air embolism may virtually be indistinguishable from that due to a combination of reduced cerebral blood flow and hypoxaemia.

In hyperbaric decompression, patients who survive a short time have congested brain and spinal cord. There may be haemorrhages. After long survival, multiple small infarcts may be seen, particularly in the upper levels of the thoracic cord. The changes are essentially restricted to white matter, most commonly in the central position of each posterior column.[32] According to Haymaker[32] the lesions in the spinal cord are due to bubbles of nitrogen that appear in the blood stream after the drop in atmospheric pressures, together with embarrasment of the venous circulation of the cord by gas bubbles arising in the epidural and/or retroperitoneal fat.

If hypobaric decompression is rapid there may be symptoms, brain dam-age and even death. At autopsy, there are multiple foci of ischaemic damage in the grey matter of the cerebral hemispheres, the cerebellum and brain stem. There is relative sparing of the spinal cord.[33]

In addition to the changes to the brain and spinal cord, fatty change in the liver may be seen in fatal decompression disorders whether they occur after ascent from depth or following exposure to altitude.

MEDZCO-LEGAL ASPECTS

a. That a complete and detailed study of thosedeaths that occur as a result of rapid decompression is necessary is in no doubt. Its significance lies in the field of "preventative" medicine in connection with the armed forces and in industry, particularly with the development of "off shore" drilling in the construction of "off shore" platforms. The possibility of civil litigation exists.

b. In the "therapeutic misadventure?' situation there is a likelihood of civil action.

c. Criminal action for criminal abortion is rarely seen in the UK since the Abortion Act of 1967. The general rule is that death from venous air embolism occurs in situ where the incident occurred. Accordingly if the victim of fatal air embolism is found in some site where it is unlikely for an abortion to have been attempted, the victim has in all probability been transported to and deposited at this place after death transpired elsewhere. However, a case of fatal delayed air embolism following criminal abortion has been recorded.[10]

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