TWO CASES OF FAT EMBOLISM

BY

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These two following cases of fat embolism are reported for the following reasons:

1. They both occurred within the space of one month.

2. The second case shows fat embolism out of all proportion to the magnitude of the bony structures involved.

Case No. 1

Pte. G., a healthy young paratrooper aged 29 years, sustained a subtrochanteric fracture of his right femur whilst parachuting at night. He was taken immediately to hospital where, under general anaesthesia, his leg was immobilized by skin traction on Thomas splint. The following morning his condition was most satisfactory and he showed no signs of shock and was fully conscious and comfortable. It was arranged for him to be transferred to the Cambridge Hospital that afternoon. Fifteen hours following the injury his transfer was made. He was given 1/2 grain morphine. The journey was thirty miles and was made by ambulance, taking two hours.

On arriving at his destination he was drowsy and had pin-point pupils. It was thought at this time this was due to morphine. Two hours later (eighteen hours after injury) he showed signs of cerebral irritation coupled with cyanosis. Following X-rays of skull and lumbar puncture (negative results) the diagnosis of fat embolism was made. He was given continuous oxygen through B.L.B. mask and penicillin. Twenty hours following injury his condition worsened and he was in deep coma, no focal CNS signs; râles at bases of lungs. Temperature 99°; pulse 130, regular; respiration normal. Thirty-four hours following injury his condition suddenly deteriorated again. He had gradually developed a pyrexia of 103°. His pulse became irregular and he developed signs of circulatory collapse. Death took place at forty-eight hours after injury.
Autopsy Findings

The body is that of a well-developed, well-nourished, young adult male, measuring 69 inches in length and weighing approximately 160 pounds. Full rigor is present in the jaw, upper and lower limbs. There is a slight abrasion in the skin overlying the bridge of the nose. The left pupil measures 5.5 mm.; the right one measures 6.0 mm. in diameter. The right lower limb is in external rotation, and is markedly crepitant to movement. The girth of the right thigh at its midpoint is considerably increased, and the lateral aspect shows a sizeable dark blue discoloration.

Each pleural space contains approximately 150 ml. of serosanguinous fluid. The abdomen is free of adhesions and of extraneous fluid.

Cardiovascular System

Heart.—The heart shows a scant number of epicardial petechial haemorrhages, posteriorly at the atrio-ventricular groove.

Aorta.—The aorta shows a few atheromatous plaques throughout.

Respiratory System

Lungs.—The left lung weighs 818 gm. The surface is smooth and shiny throughout, and shows many disseminated petechial haemorrhages. The entire lower lobe is liver-like in consistency, dark purple, and non-crepitant to digital compression. The upper lobe shows small foci of atelectasis, but it is otherwise normally crepitant. On section, the free edge of the lower lobe everts, and copious serosanguinous fluid runs from the free uniformly dark purple surface.

There are no grossly demonstrable emboli.

The right lung weighs 908 gm. It resembles its fellow in all respects, except that the involvement extends into the upper lobe to a moderate degree.

Hematopoietic System

Spleen.—The spleen is not remarkable.

Lymph Nodes.—The lymph nodes are not remarkable.

Gastrointestinal System

The gastro-intestinal tract is examined from the level of the epiglottis to the anus. It is not remarkable except for many mucosal petechial haemorrhages in the sigmoid colon.

Liver.—The liver weighs 1,800 gm. It is dark brown, and on section displays a slight nutmeg pattern.

Central Nervous System

Brain.—There are no abnormalities in either the scalp or calvarium. The dura is pearly-grey, and tense. The brain is markedly hyperæmic and the gyri are flattened. A marked pressure conus is noted in the inferior surface of the cerebellum. Further microscopic examination of the brain reveals nothing worthy of note.
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Miscellaneous

Right Lower Limb.—The right thigh at its midpoint measures 24.5 inches in circumference; the left thigh at the same level measures 20.5 inches in circumference. On dissection of this region of the right thigh, the femur is found to be involved in a comminuted fracture, the ends of the bone overriding for a distance of approximately 3 inches. The adjacent muscle is disrupted by massive hæmorrhage, and torn by the sharp bone ends.

Microscopic Examination

Heart.—A fresh sub-epicardial hæmorrhage is noted. The nuclei in the myocardium vary considerably in size and in staining quality. There are rare small foci of myocardial coagulation necrosis. There is no cellular inflammatory exudate.

Lung.—One section displays well-aerated lung, showing congestion of the vasculature, minute focal recent hæmorrhages, and a diffuse, very light, intra-alveolar cellular infiltrate comprised of macrophages, an occasional neutrophile, and a rare eosinophile.

Another section shows large focal patches of cellular consolidation, generalized vascular congestion, and frequent focal recent hæmorrhages. In the more darkly-staining areas the cells are principally neutrophiles, and an occasional macrophage. In the more lightly-stained cellular zones the infiltrate is either a sparse number of neutrophiles in a protein precipitate or the infiltrate is phagocytic, with many of these showing clear cytoplasmic vacuoles.

A third section displays the same picture as seen in the second slide, except that the entire lung is consolidated.

The bronchi and bronchioles are filled with neutrophiles. The epithelium is desquamated and the structure is identified only by the muscle in the wall.

Tracheobronchial Lymph Node.—The node shows generalized vascular engorgement. The sinusoids are filled with macrophages, lymphocytes, eosinophiles, and a few neutrophiles. Many of the phagocytes show a foamy cytoplasm; some show large, solitary, clear vacuoles. In the medullary portion of the node there is considerable anthracotic deposit.

Colon.—The colon shows vascular congestion and severe œdema of the lamina propria.

Liver.—The liver shows a severe centrallobular passive congestion.

Spleen.—The spleen shows generalized, severe passive congestion. It is not otherwise remarkable.

Thyroid.—The thyroid gland shows acini lined by a low epithelium, and filled with a deeply-stained colloid.

Fat Stain Report

Prostate.—Definite fat embolism.

Trachea.—Definite fat embolism.

Suprarenal.—Copious lipoid, but not definite fat embolism seen.
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Brain. — A small area of recent softening shows diffuse positive Sudan III staining, suggesting a leakage of embolic fat from a ruptured vessel.

Spinal Cord. — Fat embolism not seen.

Thyroid. — Definite fat embolism.

Spleen. — Abundant fat in splenic sinusoids, with an occasional globule in a vessel.

Kidney. — Fat in vessels, glomerular tufts, tubular epithelium and limina of tubules.

Stomach. — Abundant fat irregularly distributed throughout sections.

Lung. — Copious fat in vessels and alveoli.

Myocardium. — Some diffuse lipoid staining, but no true embolic fat seen.

It was not possible to frame any suspicion of fat from the H. and E. sections.

Discussion

The positive findings in this case include:

1. Fractured (simple comminuted) right femur.
2. Cerebral congestion and oedema, moderately severe.
4. Bilateral pulmonary oedema, severe.
5. Bilateral pulmonary congestion, severe.
6. Petechial haemorrhages, pulmonary, diffuse.
7. Tracheobronchitis, mucous, moderately severe.
8. Petechial haemorrhages, heart and sigmoid colon, mild.
9. Widespread fat embolism (trachea, lungs, thyroid, spleen, kidneys, stomach, prostate).

Comments

The immediate cause of death in this case must rest with severe pulmonary embarrassment due to massive pulmonary fat embolism, oedema and congestion. The pulmonic lesions led to anoxia of all organs, but to the most serious detriment of the cerebral centres. The fat emboli no doubt were instrumental in causing rightsided cardiac failure manifested by widespread passive congestion of viscera. The brain, of course, showed the effect of this, in the oedema and hyperæmia found therein. The latter in effect compounded the cerebral anoxic sequelæ and no doubt aided failure of cardio-respiratory and thermic centres.

Cause of Death

1. Anoxia, severe, secondary to acute focal pneumonitis, oedema, and congestion caused by fat embolism.
2. Simple complete fracture, right femur.

Case No. 2

Pte. R., a healthy young soldier aged 22 years, was on an assault course. He was crossing a river by a rope-walk and he fell. As he fell some ten feet he landed in shallow water and a thunderflash exploded under his right foot. He was not concussed. The explosion blew the toe of boot away and damaged the
right foot. Half an hour later he was given a $\frac{1}{10}$ grain of morphine and removed by ambulance to hospital. He arrived at the hospital three-quarters of an hour after the injury was incurred. On admission a tourniquet (applied immediately following the injury) was found to be only causing venous congestion and was removed. It was found that he had sustained compound fractures and partial amputation of all his toes of his right foot and he had simple fractures of the metatarsals III and IV. His condition was one of a slight degree of shock, but he was quite lucid and gave a clear account of the accident. He was immediately given A.T.S. and started on penicillin therapy.

Five hours following accident his condition had improved and it was decided to take him to the theatre for wound inspection and treatment; he was given atropine 1/100 grain. A half an hour later, before he had left the ward, he became drowsy, and within a period of ten minutes became fully unconscious. He was slightly cyanosed, pulse regular and normal (72), B.P. 150/90; chest, abdomen and CNS were NAD. X-ray of skull and lumbar puncture proved normal, and a diagnosis of fat embolism was made. Continuous oxygen therapy (B.L.B. mask) was started. His condition gradually worsened and seven hours following injury he gradually developed a pyrexia (101°). Blood sugar level was 200 mgms. per cent.

Next day (twenty-two hours following injury) he was still in deep coma and his respirations had become stertorous. Numerous râles could be seen in both lungs. Temperature was now 102° and B.P. 155/95. He had pin-point pupils not reacting to light. No other focal signs in his CNS. Blood sugar level was now 130 mgms. Urine (catheter specimen) was examined, but no lipuria could be demonstrated. Twenty-three hours following injury he had an attack of Jacksonian epilepsy, commencing on the right side of his face and spreading to the right arm and leg. Small petechiae appeared on the chest wall. His condition gradually worsened and the patient died thirty-one hours following injury.

**Autopsy Findings**

The body is that of a well-developed, well-nourished white male. Dependent lividity is marked and post-mortem rigidity is complete throughout. Occasional petechial hæmorrhages are noted over the face, chest and thighs. The nail beds display deep cyanosis. Several small abrasions are noted over the right thigh and leg. There is rather deep excoriation over the heel and instep of the right foot. Each toe of the right foot is involved in a ragged wound in which the soft tissues and splintered bone are visible.

**Head.**—Except for the previous mentioned petechial hemorrhages, the external examination of the head is not noteworthy. The examination of the brain displays moderate vascular congestion and a small linear subarachnoid hæmorrhage over the right post-central gyrus. There is no flattening of the gyri and a pressure cone is absent. Numerous coronal sections of the brain, brain stem, and cerebellum fail to show noteworthy findings. There are no apparent skull fractures.

**Thorax.**—There is a small amount of clear amber fluid in each pleural space.
Left Lung.—The left lung is smooth, shiny and generally of a gun-metal hue. Occasional clustered petechial haemorrhages are scattered over the surface. The lung is heavy and boggy, though some crepitus is appreciated upon digital compression. While the lung in general is filled out and somewhat tense, there are occasional focal, small, dark blue depressed areas suggesting atelectasis.

On section the lung appears wet and a moderate amount of serosanguinous fluid issues freely from the out surface. When viewed in obliquely reflected light, minute fat globules are seen to float upon this fluid. The tracheobronchial tree and pulmonary vasculature are not remarkable.

Right Lung.—The right lung resembles its fellow in all respects. Other organs appear normal.

Microscopic Appearance

Lung.—The vasculature is generally engorged with blood, and the alveolar capillaries often display small clear spaces, later identified as fat. The lung shows focal consolidation not always related to bronchioles. The consolidation is comprised of a varying admixture of protein-precipitated fluid, masses of neutrophiles and frequent masses of bacteria. There is a generalized light sprinkling of macrophages in the alveoli. Often these contain dark granular pigment, presumably anthracotic.

There are also foci of anthracosis-filled macrophages enmeshed by variously dense fibrous tissue accumulation.

Tracheobronchial Lymph Node.—The lymph node architecture is largely replaced by a faintly acidophilic structureless material ringed thinly by fibrous tissue. Just inside the fibrous tissue rim one sees occasional giant cells. The node otherwise shows intense vascular engorgement.

Heart.—The myocardium shows small focal degeneration attended by a neutrophilic cellular infiltrate.

A recent perivascular haemorrhage is seen in the epicardial fat.

Liver.—The liver shows centrilobular vascular congestion. Most other organs show slight congestion.

Fat Stain Report

Lung.—The lung shows considerable intra-alveolar and intravascular fat distributed focally.

Trachea.—Some intravascular and perivascular fat is seen in the tracheal adventitia.

Heart.—The heart shows focal fatty deposits in the myocardial fibres.

Liver.—Fat is seen within the centrilobular hepatic cells.

Adrenal.—Massive intracellular fat is seen in the adrenal cortical cells.

Kidney.—Fat is seen in the glomeruli and apparently within the lumen and cells of the proximal convoluted tubules.

Spleen.—The spleen shows no fat.

Brain and Brain Stem.—The brain and brain stem sections reveal no fat, nor is any abnormality found by H. and E. staining.
Two Cases of Fat Embolism

Significant findings in this case include the following:

1. Compound fractures and partial traumatic amputation of all toes, right foot.
2. Widespread traumatic excoriation of the skin (heel and instep), right foot.
3. Traumatic widely scattered small abrasions, right leg.
4. Petechial haemorrhages of face, chest, thighs, lungs, epicardium and pericardium.
5. Hydrothorax, mild, bilateral.
6. Passive congestion, moderately severe, pulmonary.
7. Pneumonitis, focal, acute.
8. Fat embolism, massive pulmonary.
10. Passive congestion, acute, liver.
11. Fat embolism, glomerular tufts, kidney.

Comments

This is the case of a young male who died of widespread fat embolism following compound fracture of all toes of the right foot. Actual fat embolism is demonstrated in many organs—mostly importantly in lungs and kidneys. In the former, a severe inflammatory reaction attends the fat deposit. In the kidney, the fat evokes no inflammatory response: the renal consequence would seemingly impose a pure mechanical blockage of glomerular loops. In connection with the patient’s coma, the normal macroscopic, microscopic and fat-staining appearances are interesting.

It is suggested that a considerable quantity (American Archives of Pathology—150 gm.) must enter the circulation for death to follow upon such an embolic phenomenon. In a case as this, the question arises as to the source of such a large quantity of fat. Certainly such a large amount did not arise in the marrow of the fractured phalanges; and it is difficult to see how it would gain access to the circulatory tree from the surrounding soft tissue. One is led to wonder if the fat might not arise at the site of injury, therefore, but that the injury sets off a metabolic derangement whereby fat from more distant body sources is in some way set free and allowed to enter the circulation.

The fat in myocardial fibres and in liver cells is thought to be fatty degeneration—on an anoxic basis. That in myocardium secondary to fat embolism; that in liver cells perhaps related to the passive congestion noted therein.

Cause of Death

1. Anoxia, severe, secondary to widespread fat embolism (particularly pulmonary).
2. Traumatic compound fractures, all toes, right foot.

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