One autopsy case of an elderly traffic accident victim with Tetralogy of Fallot

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Abstract : The case of a 61-year-old male traffic accident victem with Tetralogy of Fallot (TOF) is reported. The autopsy revealed massive hemorrhages in the subcutaneous tissue, muscle, and subarachnoidal space. Furthermore, multiple fractures of ribs, sternum and thoracic vertebrae were observed. Histopathological examination revealed changes characteristic of trauma, such as acute lung congestion, acute renal cortical necrosis, and embolization in the lungs and kidney. These autopsy and histological observations indicated that traumatic shock was cause of his death. Moreover, histologically, we observed changes due to his congenital heart disease, such as right ventricular hypertrophy, heart failure cells in the lungs, sclerosis of the liver, and hyaline degeneration in the kidney. Furthermore, ischemic changes, shrinkage or loss of neurons, were seen in hippocampus, and swelling of astrocytes in both cortex and hippocampus were also observed. These observations lead us to speculate that a hypoxic episode may have caused his accidental death while driving. J. Med. Invest. 46 : 115-119, 1999

Key words **:** *tetralogy of Fallot, forensic pathology, cause of death, traffic accidental death, immunohistochemistry*

INTRODUCTION

Tetralogy of Fallot (TOF) is a cyanotic congenital heart disease with pulmonary outflow tract stenosis (PS), ventricular septal defect (VSD), displacement of the aorta, and right ventricular hypertrophy (RVH). Generally, TOF patients without severe anoxic episodes can survive into the first decade or early into the second decade of life, few patients live on into adult life, and patients who survive into their fifth and sixth decades are very rare (1, 2).

We autopsied a victim of a traffic accident, a 61-year-old male, and found that he might have been suffering from TOF for long time. Here, we reported the results of histopathological examination of this case. In this report we present the histopathological findings that revealed changes due to traumatic shock and TOF. Furthermore, the cause of his death is briefly discussed.

Received for publication December 7, 1998 ; accepted January 4, 1999.

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CASE REPORT

A 61-year-old male lost control while driving and his car fell down a precipice, 8 m in height. He died 2 hours after the accident without regaining consciousness.

His medical history included the Tetralogy of Fallot (TOF), and hypertension and arrhythmia under treatment for more than 10 years. He also had occasional syncopal episodes.

Autopsy findings and microscopic examination

The patient was 166.8cm in height and weighed 57.5kg. The autopsy showed multiple abrasions, contusions and lacerations of the head, chest, and shoulder. Multiple bilateral ribs and the sternum, and 10 th and 11 th thoracic vertebrae were fractured.

There were contusions and hemorrhages in the frontal lobes and diffuse subarachnoidal hemorrhages. A microscopic examination revealed multiple bone-marrow and fat embolization in the lungs and kidneys (Fig. 1), which probably came from fractured ribs.

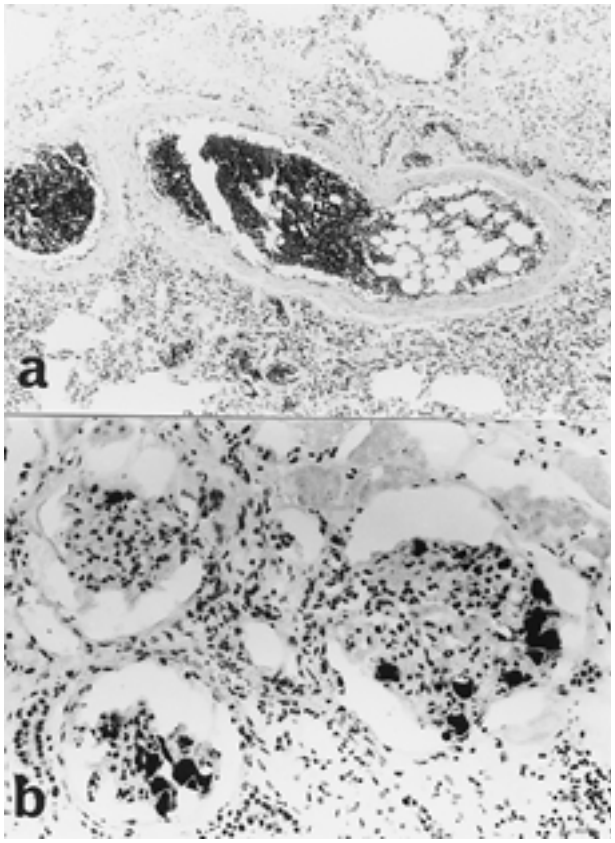
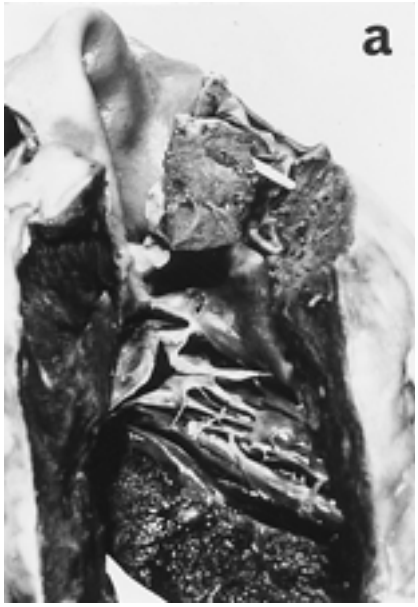


Fig. 1. Bone marrow embolisms in the alveolar lumen (a; hematoxylin-eosin, x200) and fat embolisms in the renal glomeruli (b ; Sudden III, x200).

The heart was hypertrophic and 675 g in weight (normal weight; 349±67.2g) (3). The right ventricular (RV) wall was also hypertrophic and was 30 mm thick (normal thickness; 4.2±1.52mm) (3). His pulmonary outflow tract was narrowed by the hypertrophic RV wall. The VSD was found just below the pulmonary valves, and the aorta was overriding above the VSD. These malformations of the heart were considered as signs of TOF (Fig. 2). The left ventricular (LV) wall was hypertrophic and its thickness was 18 mm (14.6±2.81 mm) (3). Microscopically, hypertrophy and disarray of myofibers, and enlarged interstitial fibrous tissues were found in RV (Fig. 3a).

Lungs weighing 510 g for the left, and 470 g for the right, were congestive. Histological examination revealed pulmonary congestion and fibrous thickening of the alveolar walls. Heart failure cells, hemosiderin-laden phagocytes, were found in the alveolar lumen (Fig. 3b).



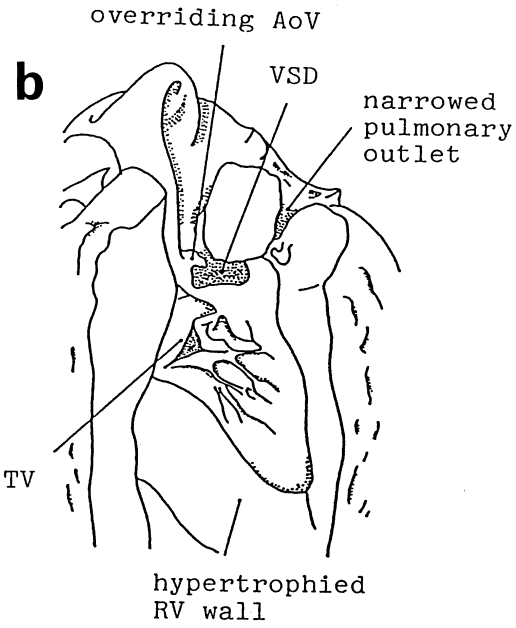


Fig. 2. Outer view (a) and illustration (b) of the right ventricle.

The liver weight was 1000g. Hepatocytes in center of the liver lobules were replaced by fibrous tissue, and the centrilobular area was enlarged, forming a fibrous bridge among the lobules (Fig. 3c).

The kidneys were 92 g (148±30.2 g) for the left and 95 g (138±30.5 g) for the right (3), and appeared to be atrophic. Macroscopically, the cortex was pale and the medulla congested. Furthermore, histologically, the proximal tubules were necrotic indicating renal cortical necrosis. The small vessels and glomerular capillaries showed hyaline degeneration, and the size of surviving glomeruli were varied. Hypertrophic glomeruli had reached 250 μm

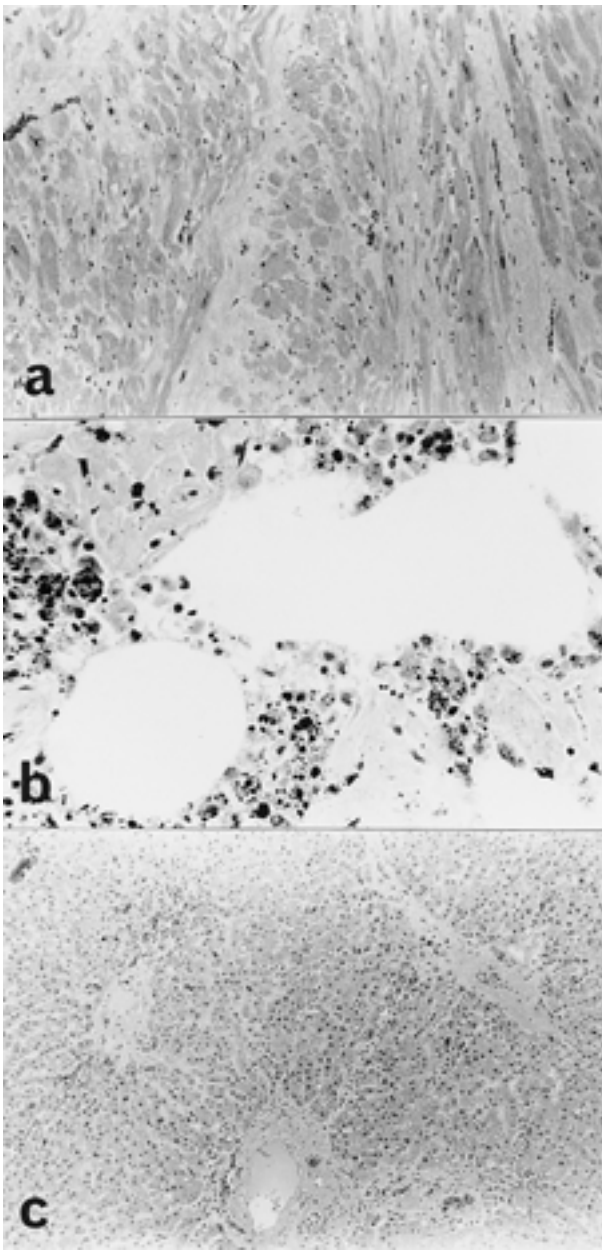


Fig. 3. Disarray of hypertrophic myofibers and interstitial fibrosis of the right ventricle (a ; hematoxylin-eosin, x100), heart failure cells in the alveolar lumens (b ; hematoxylin-eosin, x200), and cardiac sclerosis of liver (c ; hematoxylin-eosin, x100).

in diameter. Immunohistochemically by using the anti-human myoglobin antibody (Dako, Denmark), myoglobin was found in the distal tubules and Bowman's capsules

The brain weighed 1390 g, and no other remarkable changes were observed except for the cerebral contusion and the subarachnoidal hemorrhages as mentioned above. Microscopically, loss and shrinkage of neurons were observed in the hippocampus (Fig. 4a). Immunohistochemical staining using anti-glial fibrially acidic protein (Sigma, USA) and Retinus Communis agglutinin 1 (Sigma, USA) showed swelling of astrocytes in the frontal and occipital

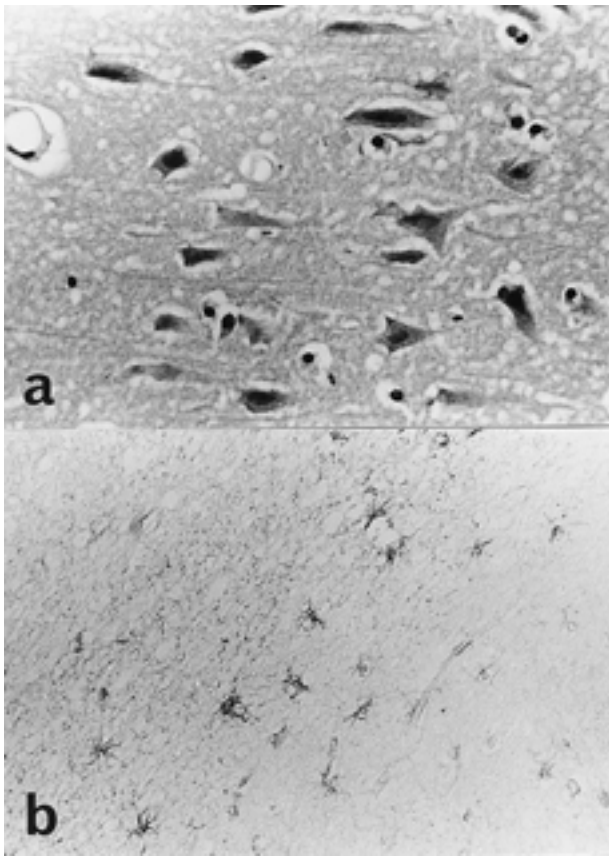


Fig. 4. Shrinkage of neurons in the hippocampus (a ; hematoxylin-eosin, x400), and the swelling of astrocytes in the cortex (b ; immunostainig. x200).

cortex and in the hippocampus (Fig. 4b), but no microglial proliferation.

DISCUSSION

The patient had many abrasions, discolorations, contusions, subcutaneous and muscular bleeding, and multiple fractures of bones, due to the traffic accident. In cases of multiple trauma, it is well known that lung edema and congestion and renal cortical necrosis are usually observed (4). Bone fractures are known to cause embolisms of the lungs and kidneys. In this case, both features and embolisms were observed. Moreover, myoglobin was found in the renal tubules immunohistochemically. Taken together, the cause of his death was considered to be traumatic shock.

The heart revealed the complex malformations of TOF (Fig. 2). The VSD was large (1.5 cm in diameter) but PS was mild. It seems conceivable that, because his right and left ventricular pressures were well balanced, he survived for 61 years without cardiac surgery. On the other hand, since the LV

and RV wall were hypertrophic, it was considered that both ventricular pressures had increased during the long survival, as a result of PS and VSD. The alveolar walls were thickened by fibrous tissues and hemosiderin-laden phagocytes, heart failure cells, were seen in the alveolar lumens (Fig. 3 b). Furthermore, small vessels in the lungs showed hyaline degeneration. These pathological changes are considered to be responsible for the chronic lung congestion due to his left ventricular hypertension (5, 6). The centrilobular area of his liver was enlarged because of hepatocytes necrosis and showed fibrous replacement. It is well understood that chronic, severe, hepatic congestion, an increasing right cardiac backward pressure, leads to these histopathological findings of cardiac sclerosis (7, 8). In the clinical and hemodynamic profile of 147 patients with TOF, the systemic hypertension was seen in 9.5% of TOF (9). The renal glomeruli and small vessels showed hyaline degeneration, and the juxtaglomerular apparatus was enlarged. These pathological changes were considered to be due to systemic hypertension (10, 11). Thus, it was considered that the changes seen in the lungs, liver and kidneys may have been secondary changes following his TOF condition.

However, the question as to why he met an accident remained to be explained. Both loss and shrinkage of neurons were seen in the hippocampus, and swelling of astrocytes occurred in the frontal and occipital cortex and the hippocampus (Fig. 4). These findings indicate that he might have a hypoxic episode a few hours before his death (12-14). Therefore, his traffic accident is presumed to have occured as the result of his hypoxic episode (syncopal attack). In TOF, it was reported that ventricular premature complexs (VPCs) and ventricular tachycardia (VT) are related to symptom of syncope (15, 16). Then, his suspected syncopal attack meight be attributable to recurrent VT.

In conclusion, a 61-year-old male, TOF patient, died 2 hours after a traffic accident. The autopsy and histopathological examination revealed two types of pathological changes. One was the changes occurred by trauma, and the other was due to his long history of TOF. Furthermore, based on the neuronal changes, it seems conceivable that a hypoxic episode may have caused his accidental death while driving.

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