**Treatment of the Survivors from the Nagoya Air Crash**

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**Abstract**

Six patients were taken to our hospital alive just after the air crash at Nagoya Airport in 1994. On admission, all patients suffered from profound shock which rapidly progressed. Serum albumin and hemoglobin levels, and platelet count decreased on admission or soon after. The four patients who died could not recover from the shock and associated rapid deterioration of neurological signs. However, two patients were successfully treated with massive transfusion of packed red blood cells, fresh frozen plasma, fresh blood, and/or platelet concentrate and survived to discharge. We recommend addition of albumin to the transfusate in such cases.

**Key words:** airplane accident, multiple trauma, hemorrhagic shock, diffuse brain injury, blood transfusion, albumin

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**Introduction**

Victims of severe traffic and airplane accidents frequently present with multiple trauma, shock, and hypoxemia. Adequate clinical data about the volume and type of fluid infusion and transfusion, which is essential for recovery from shock, has yet to be compiled. Here, we report our experience in treating survivors from the Nagoya air crash.

**Case Summary**

At 20:15 on April 26, 1994, an Airbus A300-600R crashed on landing at Nagoya Airport. The airplane lost the minimum velocity required for flight and crashed from a height of 240 m. Just before the accident, the speed of the plane was estimated to be 240 km/hr and the head of the plane was inclined at 30 degrees up. There were 271 passengers and crew, of whom 16 were alive at the airport after the crash. Almost all these survivors had escaped the fire which flared up after the crash. Six of these patients were taken to our hospital alive (Table 1), and one was dead on arrival at our hospital. Only two of these six survived their injuries. Five of the other nine victims who were transported to other hospitals were saved.

Emergency treatment consisted of rapid infusion of lactated Ringer’s solution through a venous route. Roentgenography and computed tomography (CT) were performed, then the patients were transported to the intensive care facility. Rapid transfusion of blood components consisting of packed red blood cells and fresh frozen plasma (FFP) was performed. Dopamine hydrochloride, sodium bicarbonate (7%, 250 ml), ulinastatin (300,000 U), or calcium chloride (400 mg) were also given to prevent hypotension, acidosis, disseminated intravascular coagulation (DIC), and hypocalcemia which might result from the massive transfusion. However, pupillary dilation or apnea occurred in four patients about 2 hours after admission and 3 hours after the accident.

**Case 1:** A 58-year-old male. Chest roentgenography on admission showed mild mediastinal dilatation, and repeat examination 1 hour 20 minutes later showed hematothorax on the left side (Fig. 1). More than 1300 ml of blood was continuously aspirated from the thoracic cavity drainage, so hypotension (systolic blood pressure 50–60 mmHg) persisted. Head CT showed a small contusion in the left frontal lobe. Pupillary dilatation was verified at 23:10 and the patient died at 24:00 on the same day.

**Case 2:** A 28-year-old female. Chest roentgenography on admission showed mild mediastinal dilatation, and repeat examination 1 hour 20 minutes later showed hematothorax on the left side (Fig. 1). More than 1300 ml of blood was continuously aspirated from the thoracic cavity drainage, so hypotension (systolic blood pressure 50–60 mmHg) persisted. Head CT showed a small contusion in the left frontal lobe. Pupillary dilatation was verified at 23:10 and the patient died at 24:00 on the same day.
Table 1 Summary of cases

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/Sex</th>
<th>Fractured or traumatized site</th>
<th>Head injury</th>
<th>Infusion</th>
<th>Date (April)</th>
<th>Blood pressure (mmHg)</th>
<th>Pulse rate (/min)</th>
<th>pH</th>
<th>PaCO₂ (mmHg)</th>
<th>PaO₂ (mmHg)</th>
<th>Base excess</th>
<th>Total protein (g/dl)</th>
<th>Albumin (g/dl)</th>
<th>Hemoglobin (g/dl)</th>
<th>Platelet (× 10^9/μl)</th>
<th>Outcome</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>58/M</td>
<td>hemotorax (1300 ml)</td>
<td>contusion</td>
<td>Ringer</td>
<td>25</td>
<td>60/38</td>
<td>89</td>
<td>7.206</td>
<td>28.8</td>
<td>67.2</td>
<td>-15.1</td>
<td>6.34</td>
<td>3.43</td>
<td>15.1</td>
<td>24.4</td>
<td>dead</td>
</tr>
<tr>
<td>2</td>
<td>28/F</td>
<td>Bil femur, bil humerus, rt ulna, L-2 mediatunium</td>
<td>skull fracture</td>
<td>RBC</td>
<td>26</td>
<td>91/34</td>
<td>153</td>
<td>7.129</td>
<td>51.5</td>
<td>153.6</td>
<td>-10.3</td>
<td>9.2</td>
<td>19</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>22/F</td>
<td>Lt femur, Lt radius, L-1</td>
<td>laceration of scalp</td>
<td>FFP (U)</td>
<td>26</td>
<td>129/55</td>
<td>106</td>
<td>7.468</td>
<td>29.5</td>
<td>191.3</td>
<td>-13.3</td>
<td>6.13</td>
<td>6.8</td>
<td>16.8</td>
<td></td>
<td>paraplegia</td>
</tr>
<tr>
<td>4</td>
<td>23/F</td>
<td>Lt humerus, Lt tibia, rt talus</td>
<td>traumatic</td>
<td>RBC</td>
<td>26</td>
<td>86/39</td>
<td>131</td>
<td>7.223</td>
<td>43.2</td>
<td>288</td>
<td>-9</td>
<td>10.8</td>
<td>4.6</td>
<td>13.8</td>
<td></td>
<td>dead</td>
</tr>
<tr>
<td>5</td>
<td>3/M</td>
<td>Rt femur, rt humerus, spleen, lung</td>
<td>subdural hematoma</td>
<td>RBC</td>
<td>26</td>
<td>130/12</td>
<td>157</td>
<td>4.88</td>
<td>15.7</td>
<td>5.79</td>
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<td>full recovery</td>
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<td></td>
<td></td>
<td>operation (hemorrhage 138 ml)</td>
<td>RBC</td>
<td>23</td>
<td>137/62</td>
<td>154</td>
<td>7.197</td>
<td>48.8</td>
<td>15.7</td>
<td>9</td>
<td>10.8</td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>(fresh blood 2 U)</td>
<td>RBC</td>
<td>23</td>
<td>137/62</td>
<td>154</td>
<td>7.197</td>
<td>48.8</td>
<td>15.7</td>
<td>9</td>
<td>10.8</td>
<td>28.8</td>
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<td></td>
<td></td>
<td></td>
<td>operation (hemorrhage 1200 ml)</td>
<td>RBC</td>
<td>14</td>
<td>140/51</td>
<td>140</td>
<td>7.118</td>
<td>51</td>
<td>90.4</td>
<td>-13.7</td>
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<td></td>
<td></td>
<td></td>
<td>(platelet 10 U)</td>
<td>RBC</td>
<td>14</td>
<td>140/51</td>
<td>140</td>
<td>7.118</td>
<td>51</td>
<td>90.4</td>
<td>-13.7</td>
<td>7.4</td>
<td>22.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>32/M</td>
<td>Lt femur</td>
<td></td>
<td>RBC</td>
<td>26</td>
<td>122/70</td>
<td>142</td>
<td>7.458</td>
<td>33.4</td>
<td>248.9</td>
<td>1.1</td>
<td>10.8</td>
<td>5.2</td>
<td></td>
<td></td>
<td>dead</td>
</tr>
</tbody>
</table>

same day showed the progression of the mediastinal dilatation and hypotension deteriorated thereafter. Head CT showed a skull fracture on the left side. Pupillary dilatation occurred at 21:40 on April 26, and the patient died at 4:33 on April 28.

**Case 3:** A 22-year-old female. The patient was almost conscious at admission, but she had paralysis of both legs due to a fracture of the first lumbar vertebra (Fig. 3). Large amounts of blood and fluid infusions were necessary for the treatment of gradually progressive hypotension. She had no brain injury and was discharged 3 months later with complete paraplegia.

**Case 4:** A 23-year-old female. Pupillary dilatation occurred during the CT examination, which revealed traumatic subarachnoid hemorrhage (Fig. 4). The patient never recovered and died at 12:25 on April 27.

**Case 5:** A 3-year-old male. After admission, the patient showed progressive anemia, hypotension, and acidosis. Head CT showed only a small subdural hematoma beside the falx (Fig. 5). Peritoneal tap and abdominal echography verified an intraabdominal hemorrhage. A laparotomy was performed to remove the lacerated spleen. Although his general condition improved thereafter, anemia and hypotension

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**Fig. 1** Case 1. Chest roentgenogram on admission showing mild mediastinal dilatation (left), and repeat roentgenogram 1 hour 20 minutes later showing hematothorax on the left side (center). Head computed tomography scan showing small contusion in the left frontal lobe (right).

**Fig. 2** Case 2. Chest roentgenogram on admission showing mild mediastinal dilatation and pulmonary contusion on both sides (left), although echocardiography showed no cardiac tamponade. Repeat roentgenogram on the same day showing progression of the dilatation (center). Head computed tomography scan showing skull fracture in the left convexity (right).

*Neurol Med Chir (Tokyo) 38, July, 1998*
continued and a second operation was performed to remove 1200 ml blood retained in the retroperitoneal cavity. Transfusion of platelet concentrate and fresh blood was given.

Fig. 3 Case 3. Roentgenogram showing a fracture of the first lumbar vertebra causing paralysis of both legs.

Fig. 4 Case 4. Head computed tomography scans revealing traumatic subarachnoid hemorrhage (arrow).

Fig. 5 Case 5. Head computed tomography scan showing a small subdural hematoma beside the falx (asterisk) and a subcutaneous hematoma (arrowheads).

Fig. 6 Case 6. Computed tomography scans showing normal findings, but this did not exclude brain stem injury.
Respirator control was necessary for 2 weeks due to a lung contusion. A pre-DIC state also persisted for more than 2 weeks as shown by the following: Prothrombin time 67.6% (normal range 80-100%), activated partial prothrombin time 100% (normal range 80-120%), thrombo test 80.1% (normal range 70-130%), fibrinogen 207 mg/dl (normal range 200-400 mg/dl), D-dimer 5.421 µg/ml (normal range less than 0.15 µg/ml), alpha2-plasmin inhibitor-plasmin complex 1.4 µg/ml (normal range less than 0.8 µg/ml), thrombin-antithrombin complex 60 ng/ml (normal range less than 3 ng/ml) on April 27; fibrin degradation products (FDP) 18 µg/ml (normal range less than 10 µg/ml), D-dimer 8.1 µg/ml on April 28; FDP 22 µg/ml on May 1; FDP 36 µg/ml, D-dimer 17 µg/ml on May 9; and FDP 13 µg/ml, D-dimer 13.1 µg/ml on May 16.

Fortunately, the patient recovered completely and was discharged 3 months after the accident. **Case 6: A 32-year-old male.** Although the patient could speak on admission, his spontaneous respiration ceased during the CT examination and his pupils dilated soon after. CT of the brain showed normal findings, but this did not rule out brain stem injury (Fig. 6). He died at 22:35 on April 26.

**Discussion**

On admission, all six patients suffered from shock, which apparently progressed very rapidly. Although sufficient fluid infusion and transfusions were given immediately to replace the estimated blood loss calculated from the injuries, the volume of thoracic and abdominal hemorrhage and the shock index, three patients (Cases 1, 2, and 4) never recovered and died of profound shock. We think that laceration of the aorta may have occurred in Cases 1 and 2,6) and there was an unknown hemorrhagic site in addition to the fractured extremities in Case 4. Acidosis was worsened even after application of a respirator in Cases 1 and 4. In contrast, the blood pressure of the two survivors (Cases 3 and 5) was maintained above 100 mmHg and their blood gas data improved after admission. Intraabdominal hemorrhage from the lacerated spleen in Case 5 was successfully treated with two abdominal operations.

Blood albumin levels decreased on admission or soon after, so infusion of albumin as well as concentrated red blood cells and FFP is recommended. Serum albumin is estimated to increase by 0.6 g/dl after infusion of 25 g of albumin (100 ml of 25% albumin). However, 1 U of FFP contains only 2.6 g of albumin.

Treatment of traumatic brain injury absolutely requires maintenance of the systemic blood pressure and cerebral perfusion pressure.2-3,6-14) The rapid deterioration of neurological signs in Cases 1 and 2 was thought to be caused by the failure to maintain cerebral perfusion pressure. Deteriorating metabolic acidosis might have accelerated the brain damage in Case 4. Occult laceration of the brain stem might explain the abrupt spontaneous disappearance of respiration followed by pupillary dilatation in Case 6.16)

Platelet count also decreased rapidly on admission or soon after. Transfusion of fresh blood or platelet concentrate is required to compensate for platelet loss due to hemorrhage.1,15) Furthermore, coagulopathy was caused by hemodilution and release of tissue thromboplastin from the injured sites in Case 5, so the transfusion of fresh blood, FFP, and uinastatin is necessary to reinforce coagulation.

**References**

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Commentary

This paper provides invaluable information to neurosurgeons in Japan, since most of them will never have experience in treating traumatic shock following an air crash during their lifetime. The authors report that hemorrhage-induced hypovolemia is the major cause of traumatic shock in an air crash. They also provide evidence indicating that neurogenic shock must always be considered as a potential contributor. Traumatic shock may be caused by more than one pathophysiologic mechanism. High speed acceleration and/or deceleration of the head can produce injury to the central nervous system, such as laceration of the pontomedullary junction or cervical spinal cord trauma. The neurogenic shock caused by central nervous system trauma, if it occurs in isolation, is generally characterized by neurologic deficits, warm extremities, and preserved urine output. When hypovolemia is superimposed, however, the presence of injury to the central nervous system may be overlooked and the characteristics of neurogenic shock become unclear. Excessive fluid administration to patients with injury to the central nervous system may be detrimental. Volume status must be carefully monitored, therefore, if neurogenic shock is suspected to exist. Areas for research in traumatic shock include the use of hypertonic saline. The hypothesis is that hypertonic saline could provide adequate replacement for hemorrhage-induced hypovolemia while limiting injury-induced edema of the central nervous system.

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The authors have presented six well documented cases of multiple injuries caused by the tragic air crash at Nagoya Airport in 1994. Besides head injury, the patients who died suffered from severe multiple injuries (hemothorax, pulmonary contusion, spleen laceration, and multiple fractures of the extremities) associated with profound shock state. The present report is quite suggestive that one of the most important key factors in the treatment of severe head injury with multiple injuries is to overcome the shock state. JD Miller has demonstrated that an ICP exceeding 20 mmHg after head injury is an unfavorable prognostic sign. Age, initial Glasgow Coma Scale, cerebral perfusion pressure (CPP), CBF, and systemic factors such as hypotension are also important determinants of neurological outcome after severe head injury. Recently, MJ Rosner has shown that directing therapy (vasopressor treatment) to keep CPP over 70 mmHg significantly improves outcome in patients with severe head injury. Indeed, the present report clearly demonstrated the importance of maintaining blood pressure (CPP) as an initial treatment of severe head injury. The authors also emphasized the importance of massive transfusion of packed red blood cells, fresh frozen plasma, platelet concentrate, and albumin to treat the initial shock state.

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