Diagnostic Dilemma with Interval Death in a Road-Traffic Accident Victim

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Background: The cause of death is difficult to interpret in a road-traffic accident (RTA) victim, because multiple injuries make it difficult to decide on the most fatal lesion, particularly when death is delayed by prompt medical intervention; and secondary haemorrhage, renal failure, fat embolism, systemic infections, myocardial or cerebral infarction – all comes under the potential differential threats.

Case Report: A 20 year-old RTA victim was hospitalized in a comatose state and died after surviving 21 days on intermittent positive pressure ventilation. Post-mortem examination of different organs revealed pneumonic consolidation in lung, fatty changes in liver and acute tubular necrosis in kidney; in addition to haemorrhage and congestion in these organs and brain.

Conclusion: Acute contusions of various internal organs, compounded by hospital-acquired infection and medical interventions turn the overall diagnostic scenario messy in a resuscitated RTA victim. In such condition notification about all lethal organic defects, instead of mentioning mere 'multiorgan failure', is the best way to sign out an 'autopsy report.

►*Implication for health policy/practice/research/medical education:* Diagnostic Dilemma with Interval Death in a Road-Traffic Accident Victim

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1. Introduction:

Each year around five lacs road-traffic accidents (RTA) happen on Indian roads, in which approximately 1.4 lacs people lost their lives in recent years and more than five lacs people suffered various forms of injuries (1, 2). In India RTA is projected to become the fifth highest leading cause of death by 2030, outranking diabetes mellitus, HIV/AIDS, certain cancers and tuberculosis, up from its ninth position in 2004 (1). In an RTA victim, it is easy to decide the cause of death, when injuries are grossly obvious. But, in case multiple injuries are suffered, it is extremely difficult to decide on the most serious and grave defect. In such cases the term 'multiple injuries' are often used, listing several of the lethal injuries. If prompt medical intervention is instituted and the patient dies after a significant period of hospitalization, then an array of complex and

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varying pathological changes supervene in different vital organs over the acute changes inflicted by the accident; making it difficult to diagnose the proper cause of death. In such cases death is usually caused by secondary haemorrhage, renal failure, septicaemia, myocardial or cerebral infarction and other sequela (3).

Herein we report an unusual case of variable and overlapping multi-organ changes in a 20 year old RTA victim, who survived 21 days on ventilation after the accident.

2. Case Report:

A 20 year-old man, while returning from work, riding his personal bicycle, collided head-on with an on-rushing heavy truck. He was immediately brought to the 'Emergency OPD' of nearby North Bengal Medical College and Hospital by the passers-by. On presentation, the patient was comatose with obvious evidence of head-injury and numerous bruises-abrasions-lacerations spread all over the trunk. His vitals were unstable and pO2 was declining. He was admitted instantly to the Intensive Care Unit, provided with hemodynamic support and put on intermittent positive pressure ventilation (IPPV). After 21 days of IPPV support and few negligible episodes of remissions, the patient finally expired. His body was autopsied by forensic surgeons and his brain, kidneys, lungs, liver and spleen were en masse sent to the Department of Pathology for histopathological (H/P) evaluation in order to diagnose the proper cause of death.

Grossly, the brain, lungs, kidneys and spleen appeared congested with blood clot in subarachnoid space (Figure 1A, 2A, 3A), but the liver appeared normal. The cut surface of the lungs appeared congested with areas of consolidation. On sectioning the kidneys appeared flabby with medulla appeared as a red band, in contrast to the more widened and pale cortex (Figure 3A).

Microscopically, there was congestion of the cerebral and cerebellar blood vessels along with subarachnoid haemorrhage and congestion; cerebellar purkinje cell layer was hardly demonstrable (Figure 1B and C). Both lungs featured congestion, exudation, superimposed pneumonic consolidation with

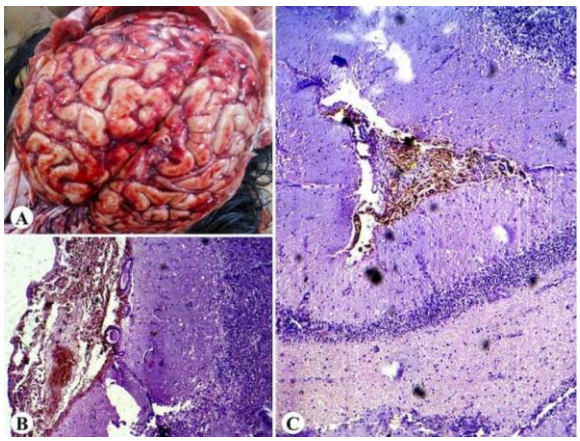

Fig. 1. Gross appearance of congested cerebral hemispheres [A]; Subarachnoid haemorrhage [B] with congested intracerebellar vessels and barely visible cerebellar purkinje cell layer [C] [H&E stain, 40x].

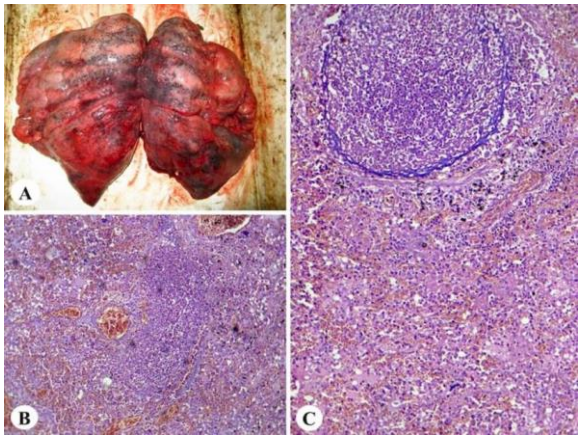

Fig. 2. Bilateral congested lungs [A]; Pneumonic consolidation, haemorrhage, exudation with dense inflammatory infiltrate, congested blood vessels [B] [H&E stain, 40x]; and occasional lymphoid follicle formation [C] [H&E stain, 100x].

dense lymphoplasmahistiocytic infiltration and occasional lymphoid follicle formation, pertaining to the ventilator associated pneumonia (VAP) (Figure 2B and C). His kidneys revealed, oedematous enlargement of glomeruli, extensive coagulative tubular necrosis, detachment of tubular cells from their tubular basement membrane (TBM), tubular dilatation with flattening of tubular epithelium, intraluminal accumulation of proteinaceous cell debris along with few intact epithelial cells and hyaline casts. Interstitial haemorrhage with accumulation of hemosiderin-laden macrophages, oedema, mild mononuclear cell infiltrate along with

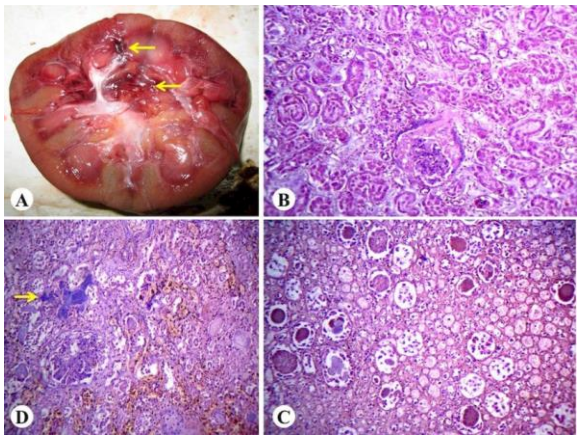

Fig. 3. Bisected kidney with widened cortex and areas of congestion [arrows] [A]; Extensive coagulative necrosis with detached tubular epithelial cells from their basement membrane [B] [H&E stain, 100x], intratubular proteinaceous debris and casts [C] [H&E stain, 40x] along with massive interstitial hemosiderin deposits and bacterial colonization [arrow] [D] [H&E stain 40x].

bacterial colonization was also prominent. All these features were consistent with acute tubular necrosis (ATN) leading to acute renal failure (ARF) and superimposed infection (Figure 3B, C and D). His liver showed, disorganized lobular architecture with diffuse macrovesicular steatosis, particularly surrounding the central veins (Figure 4), indicative of early changes of non-alcoholic fatty liver disease (NAFLD) in adjunct to ventilator-related porto-hepatic circulatory compromise.

Ultimately, all these pathological features converged to a group of complex life-threatening disorders. These included 1) Cerebrovascular accident (CVA), which can lead to respiratory centre depression; 2) Pulmonary contusion with VAP, which can cause septicaemia; and 3) *ATN* is leading ARF. The hepatic pathology was not so lethal, but had definite impact on the overall pathological picture. Among these three defects, it was difficult to determine which one was most serious and mortal. So in the end all three aforementioned disorders were rendered as the cause of death, and thereby the pointless broad term of 'multiorgan failure' was avoided.

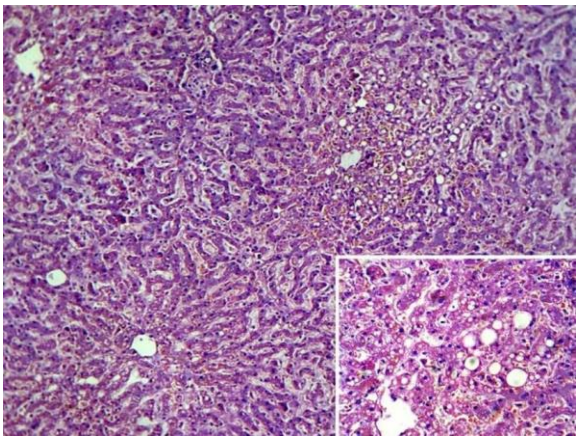

Fig. 4. Disorganized normal hepatic architecture with macrovesicular steatosis mostly around the central vein [H&E stain, 100x], [inset] [H&E stain, 400x].

3. Discussion:

The total count of RTA is on a rise and is projected to increase global deaths by 40% between 2002 and 2030 (1, 2). For this reason Mondal *et al* (4) rightfully suggested RTA as 'Tsunami on road'.

When an RTA victim succumbs to death instantaneously or soon afterwards, the cause is evident on gross autopsy in the form of organ damage, massive haemorrhage, and airway occlusion from a blood clot or traumatic asphyxia. But if multiple injuries of equivalent lethality are sustained, it becomes difficult to determine the exact cause of death. Moreover, in such an instance if the victim is resuscitated with immediate measures and he dies after a period of hospitalization, the reason behind death often becomes obscured by changes in different organs inflicted by various medico-mechanical interventions. Delayed deaths in these circumstances are usually ensued by continued bleeding, secondary haemorrhage or renal failure from hypotension, excessive muscle injury, septicemia, myocardial or cerebral ischemia (5). In such complicated cases all the lethal injuries are listed as potential causes of death (3). Likewise, in the discussed patient all three fatal defects were listed as the cause of death, in apparent decreasing order of compounding effects imparted by them.

In accident victims, extensive tissue injury and necrosis leads to widespread activation

of acute inflammatory pathway (6). These inflammatory mediators upregulate procoagulant mechanisms, platelet reactivity and downregulate physiological anticoagulants, fibrinolysis; which ultimately result in increased thrombotic tendency in RTA patients, commonly affecting the cerebral or myocardial vasculature (7). In this regard Hausmann *et al* (8) postulated that cerebellar purkinje cells are m susceptible to hypoxic damage than other cellular components in brain, and become shrunken on hypoxia. Recapitulating the similar phenomenon in the current patient, the intracerebellar vessels were thrombosed and cerebellar purkinje cells were hardly discernible. The subarachnoid haemorrhage found on autopsy was most likely sustained at the time of the accident. That might have contributed to his comatose state, but definitely had the last contribution to his delayed death.

VAP, caused by direct bacterial colonization in lower respiratory tract, occurs in patients receiving mechanical ventilation for longer than 48 hours. VAP occurs in 22.8% patients receiving mechanical ventilation and accounts for 86% cases of nosocomial pneumonia (9). Disseminated infections to heart valves, brain, kidneys are common complications of nosocomial pneumonia (10). The current patient received continuous IPPV support for 21 days. His lungs were solidified with diffuse pneumonic consolidation superimposed on congestion. His kidneys also displayed features of disseminated infection with bacterial colonization.

When muscle is severely crushed or rendered ischaemic as a result of trauma, rhabdomyolysis ensues and myoglobinuria stemming from it imparts severe toxic renal tubular injury. It manifests as cytoplasmic vacuolisation, necrosis and detachment of tubular cells from TBM; intraluminal cellular debris and casts, flattened tubular epithelium etc. Acute tubular necrosis (ATN) frequently develops into ARF (11). In this reported case the victim's kidneys exhibited features of ATN, along with superimposed changes of disseminated infection.

Chitturi *et al* (12) documented total parenteral nutrition and malnutrition as an important cause of NAFLD. As in the present case, NAFLD in early stage often presents with macrovesicular steatosis in a zone 3 distribution (12). It has also been evidenced that, continuous IPPV increases, the portal venous resistance and reduces portal venous flow by about 27-62% further complicating the hepatic pathology (13), which explains the distorted hepatic architecture in this discussed victim as well.

4. Conclusion:

Finally to conclude, in RTA victims determination of the cause of death is often problematic for the autopsy surgeons, particularly when death is postponed by resuscitative interventions. The compounding effects of trauma-induced changes, secondary infections and other metabolic derangements further complicate the job for autopsy pathologists. An ignorant compilation of all pathologies as simple 'multiorgan failure' makes the diagnosis flawy. Rather a careful consideration of every minute microscopic detail reveals several lethal and mortal defects and ideally all of these should be identified as 'causes of death'. Likewise, 'CVA' (caused by cerebrovascular thrombosis), 'pulmonary contusion with VAP' and 'ATN leading to ARF': all three were cited in the 'Autopsy Report' of the discussed case.

Proper education and awareness programmes of general public regarding electric safety will reduce the number of such cases.

Insulating/isolating high tension electric wires would insulate life.

5. References:

1. Government of India: Ministry of road transport and highways, Transport research wing's certified [Internet]. Upadhyay AK (IL): Road Accidents in India; 2011.

2. Government of India: Ministry of road transport and highways, Transport research wing's certified [Internet]. Upadhyay AK (IL): Road Accidents in India; 2012.

3. Saukko P, Knight B. Transportation injuries. In: Bureau S, editor. Knight's Forensic Pathology. 3rd ed. London: Arnold Publishers; 2004. pp. 295-6.

4. Mondal P, Sharma N, Kumar AK, Bhangale UD, Tyagi UD. A Silent Tsunami on Indian Road: A Comprehensive Analysis of Epidemiological Aspects of Road Traffic Accidents. Br J Med Med Res. 2011;1(1):14-23.
5. Sevitt S. Death after road traffic accidents. Med Sci Law. 1968;8:271-87.
6. Mitchell RN. Acute and Chronic Inflammation. In: Kumar V, Abbas AK, Fausto N, Aster JC, editors. Robbins and Cotran Pathologic Basis of Disease. 8th ed. Philadelphia: Elsevier Inc; 2010. pp. 45-68.
7. Hunt BJ, Greaves M. Acquired venous thrombosis. In: Hoffbrand AV, Catovsky D, Tuddenham EGD, Green AR, editors. Postgraduate Haematology. 6th edition. Chichester: Wiley-Blackwell Publishing Ltd; 2011. pp. 897-8.
8. Hausmann R, Seidl S, Betz P. Hypoxic changes in Purkinje cells of the human cerebellum. Int J Legal Med. 2007;121:175–83.
9. Augustyn B. Ventilator-Associated Pneumonia: Risk Factors and Prevention. Crit Care Nurs. 2007;27(4):32-9.
10.Husain AN. The Lung. In: Kumar V, Abbas AK, Fausto N, Aster JC, editors. Robbins and Cotran Pathologic Basis of Disease. 8th edition. Philadelphia: Elsevier Inc; 2010. pp. 710-9.
11.Racusen L, Kashgarian M. Ischemic and Toxic Acute Tubular Injury and Other Ischemic Renal Injury. In: Jennette JC, Olson JL, Schwartz MM, Silva FG, editors. Hepinstall's Pathology of the Kidney. 6th edition. Philadelphia: Lippincott Williams & Wilkins; 2007. pp. 1140-76.
12.Chitturi S, Farrell GC. Etiopathogenesis of non-alcoholic steatohepatitis. Semin Liver Dis. 2001;21:27-41.
13.Geiger K, Georgieff M, Lutz H. Side effects of positive pressure ventilation on hepatic function and splanchnic circulation. Int J Clin Monit Comput. 1986;3:103-6.