CASE 2: A 51-year-old male with no previous history of any medical condition was admitted to hospital after being knocked down by a vehicle while riding a motor cycle. He died six hours after admission.

A post-mortem examination was conducted on the following day on the refrigerated body. The body was that of a thinly built adult male, weighing 54 kg. There were multiple abrasions, contusions and lacerations on the head, face, trunk and lower limbs. He had skull fractures associated with sub-arachnoid and subdural haemorrhages, contre-coup cerebral contusions, a large peri-renal haematoma, burst fracture of the third cervical vertebra and multiple fractures of the right lower limb. Other internal organs including the heart were free from any traumatic injuries. The lungs were oedematous.

Furthermore, very interestingly examination of the heart revealed macroscopic and microscopic pathological features in the myocardium and RCA although they didn’t play a role in the cause of death.

The heart weighed 320 gm, myocardium showed sub-endocardial whitish discoloration due to fibrosis in the left ventricular wall. The mitral valve leaflets were enlarged, thickened and were ballooning upwards. The chordae tendinae of the mitral valve leaflets were thickened and shortened (Figure 2a). The left ventricular wall including the inter-ventricular septum was of uniform thickness and measured 14 mm. The right ventricular wall measured 3 mm. The LCA originated from the centre of the left sinus of Valsalva and the ostium was circular shaped. The anterior descending and circumflex branches showed 10-20% narrowing due to atherosclerotic changes. The RCA originated from the right margin of the left sinus of Valsalva 0.3cm above the valve commissure and had an acute take-off from the aorta with a slit-like ostium (Figure 2b). The RCA was seen to emerge between the aorta and pulmonary artery and coursed along the right atrio-ventricular groove. The RCA lumen was 20-30% narrowed due to atherosclerotic changes. Toxicological examination of blood and urine showed no alcohol or drugs. The cause of death was determined as multiple injuries following the road accident.

The samples taken from the inter-ventricular septum and free wall of the left ventricle were subjected to the same staining procedure described in Case 1.
The microscopic features were very much similar to those observed in case 1 which showed three groups of histopathological features. The myocytes were of different sizes with some showing hypertrophy and myocyte disarray (Figure 3a). The microvasculature showed thickening of the media with protrusion of the intima causing obstruction of the lumina of the small vessels (Figure 3b). There was also intra-myocardial, perivascular and septal fibrosis (Figures 3c and 3d).

Figs. 3: a) Histology of the left ventricular myocardium showing myocytes of different sizes in disarray (H&E, x200), b) showing myocytes of different sizes and thickening of the intramural coronary arteries (H&E, x200), c) showing intra-myocardial, perivascular and septal fibrosis (Masson’s trichrome, x100), and e) showing intramural coronary artery wall thickening with hump like projections, perivascular and focal fibrosis (Masson’s trichrome, x100).