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# Autopsy Pathology of Hypertensive Cardiovascular Disease: Case Reports

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## ABSTRACT

An Autopsy is performed on a dead body primarily to determine the cause of death. Two autopsies of death from hypertensive cardiovascular disease were performed at Abia State University Teaching Hospital, Aba, Nigeria. The first autopsy was on a 51 year old man diagnosed about 12 years before his death with hypertension. Autopsy report after his death revealed pan-valvular dilatation, benign nephrosclerosis, chronic pyelonephritis, hepatomegaly with chronic passive congestion of the liver, splenomegaly, pleural effusion, pulmonary edema, ascites and moderate cerebral edema. The cause of death was ruled as congestive cardiac failure and hypertensive cardiovascular disease. The second autopsy was on a 60 year old woman who died after a 5 day history of headache and a day history of loss of consciousness. Autopsy report after her death revealed massive intracerebral and intraventricular haemorrhage, pontine haemorrhage, laryngotracheobronchitis, pulmonary edema, and congested liver and spleen. The cause of death was ruled as hypertensive cardiovascular disease with massive intracranial haemorrhage. A thorough autopsy of deaths from hypertensive cardiovascular disease to reveal other associated pathologies was recommended.

***Keywords:*** Autopsy, Hypertensive cardiovascular disease, Intracranial haemorrhage, Congestive cardiac failure, Hypertension

## INTRODUCTION

An autopsy (also known as a post-mortem examination or necropsy) is the examination of the body of a dead person and is performed primarily to determine the cause of death, to identify or characterize the extent of disease states that the person may have had, or to determine whether a particular medical or surgical treatment has been effective. [1] Autopsies are performed by pathologists - medical doctors who have received specialty training in the diagnosis of diseases by the examination of body fluids and tissues. In academic institutions, autopsies sometimes are also requested for teaching and research purposes. Forensic autopsies have legal implications and are performed to determine if death was an accident, homicide, suicide, or a natural event. A medical examiner can order an autopsy without the consent of the next-of-kin. Deaths that are investigated by the medical examiner or coroner include all suspicious deaths, and, depending upon the jurisdiction, may include deaths of persons not being treated by a physician for a known medical condition, deaths of those who have been under medical care for less than 24 hours, or deaths that occurred during operations or other medical procedures. [2] In all other cases, consent must be obtained from the next-of-kin before an autopsy is performed, even at academic institutions or hospitals. The next-of-kin also has the right to limit the scope of the autopsy (for

example, excluding the brain from evaluation or limiting the procedure to examination of the abdomen).

The extent of an autopsy can vary from the examination of a single organ such as the heart or brain, to a very extensive examination. Examination of the chest, abdomen, and brain is probably considered by most pathologists as the standard scope of the autopsy. The autopsy begins with a complete external examination. The weight and height of the body are recorded, and identifying marks such as scars and tattoos also are recorded. The internal examination begins with the creation of a Y or U- shaped incision from both shoulders joining over the sternum and continuing down to the pubic bone. The skin and underlying tissues are then separated to expose the rib cage and abdominal cavity. The front of the rib cage is removed to expose the neck and chest organs. This opening allows the trachea (windpipe), thyroid gland, parathyroid glands, esophagus, heart, thoracic aorta and lungs to be removed. Following removal of the neck and chest organs, the abdominal organs are cut (dissected) free. These include the intestines, liver, gallbladder and bile duct system, pancreas, spleen, adrenal glands, kidneys, ureters, urinary bladder, abdominal aorta, and reproductive organs. [3]

To remove the brain, an incision is made in the back of the skull from one ear to the other. The scalp is cut and separated from the underlying skull and pulled forward. The top of the skull is removed using a vibrating saw. The entire brain is then gently lifted out of the cranial vault. The spinal cord may also be taken by removing the anterior or posterior portion of the spinal column. The organs are first examined by the pathologist to note any changes visible with the naked eye. Examples of diseases that may produce changes readily recognizable in the organs include atherosclerosis, cirrhosis of the liver, and coronary artery disease in the heart. After the organs are removed from the body, they usually are separated from each other and further dissected to reveal any abnormalities, such as tumors. Small samples are typically taken from all organs to be made into slide preparations for examination under a microscope. [4] At the end of an autopsy, the incisions made in the body are sewn closed. The organs may be returned to the body or may be retained for teaching, research, and diagnostic purposes. Performance of an autopsy does not interfere with an open casket funeral service, as none of the incisions made in order to accomplish the autopsy are apparent after embalming and dressing of the body by the mortician.

Hypertensive cardiovascular disease is a long-term condition that develops over many years in people who have high blood pressure. Chronic high blood pressure (higher than 120/80 mmHg) causes hypertensive heart disease. [5] As people get older and continue to have high blood pressure, their risk of heart disease increases. Heart failure occurs most often in people older than 65. [5] Chronic high blood pressure puts a strain on the heart and makes it harder for it to pump blood. The heart muscle can get thick and weak, possibly leading to heart failure. The walls of the blood vessels can also thicken because of high blood pressure, and this becomes more dangerous when cholesterol collects inside the blood vessels. High blood pressure makes men twice as likely and women three times more likely to get heart failure. [6] However, people who manage their high blood pressure can greatly reduce their risk of heart failure. High blood pressure that is not controlled for years causes hypertensive heart disease. Because there are no symptoms with high blood pressure, many people don’t know they have it. Symptoms of hypertensive cardiovascular disease often show up after the heart has already been damaged. These symptoms include chest pain, shortness of breath, palpitations, dizziness, fainting, stroke and sudden cardiac death. [7] This study reviews two autopsy reports of death from hypertensive cardiovascular disease at Abia State University Teaching Hospital, Nigeria.

## MATERIALS AND METHODS

This study was a laboratory study in which a detailed autopsy was performed on two dead bodies at Abia State University Teaching Hospital, Aba, Nigeria. This includes external description, examination of internal organs and histological analysis. Clinical notes of the patients before their death were also reviewed.

## RESULTS

**Case One: Hypertensive Cardiovascular Disease**

**Clinical Notes:** A 51 year old known hypertensive man, diagnosed about 12 years ago in a peripheral hospital, who had not been regular on prescribed antihypertensive drugs and checkup schedules, presented to the cardiology outpatient clinic of the Abia State University Teaching Hospital, Nigeria on the 5th of March, 2019 with complaints of generalized body weakness and leg swelling of 6 months duration. He had difficulty with breathing, moderate exercise intolerance, and mild paroxysmal nocturnal dyspnoea. On physical examination he was dyspnic, mildly pale and cyanosed with tachypnea and pitting pedal edema up to the knee. There was no peripheral lymphadenopathy in any of the lymph node groups. Chest examination revealed tachypnea (respiratory rate of 30cpm) and bronchovesicular breath sound. The pulse was fast (106 bpm), regular and moderate volume. His blood pressure was 170/100mmHg. S3 heart sound was also heard. There was demonstrable ascites and hepatosplenomegaly. Laboratory investigations done showed urea of 196.52mg/dl, creatinine of 479mg/dl, Glomerular filtration rate (GFR) of 49mls/min/1.73m$^2$, Na of 148mg/dl, K of 3.7mg/dl and proteinuria of 1+ on urinalysis. The full blood count showed haemoglobin of 9g/dl. Ultrasound confirmed hepatomegaly and ascites. An assessment of biventricular failure secondary to hypertensive heart disease was made. He was admitted and placed on antihypertensive drugs with diuretics. His condition however deteriorated and he developed cardiac arrest on the 7th day of admission after all effort to resuscitate him failed and he was confirmed dead at 9.24pm on the 12th of March, 2019.

**Autopsy Findings**

**External description:** The body of a middle aged man of African descent. He was pale with mild peripheral and central cyanosis, and pitting pedal edema up to the knee. There was no jaundice and no peripheral lymph node group enlargement.

**Thorax, abdominal and pelvic organs in situ:** Test for pneumothorax was negative. The thoracic, abdominal and pelvic organs were in normal anatomic positions. There was pleural effusion of about 300mls of serous fluid and about 450mls of serous ascitic fluid.

**Respiratory System:** The Tracheobronchial tree was patent down to the bronchioles. The mucosa of the trachea was hyperaemic. The right and left lungs were heavy and weighed 400g and 450g respectively. The visceral pleural was shiny. Both lungs were subcrepitant. The cut surfaces of both lungs showed exudation of free frothy fluid and bits from all the lobes floated in water.

**Cardiovascular System:** The pericardium was smooth and shiny and was covered by moderate pericardial fat. The heart was grossly enlarged and weighed 450g. The tricuspid, pulmonary, mitral and aortic valves measured 13.5cm, 11cm, 8.5cm, and 8cm respectively. The right ventricular free wall measured 0.4cm, while the left ventricular free wall measured 1.8cm. One of the papillary muscles on the left side had an external diameter of 1.6cm. The atrial walls were free of patches or plaques. The coronary arteries and vessels were patent and had normal anatomical distribution. The thoracic and the abdominal aorta showed atherosclerosis Grade 5. The cerebral blood vessels also had atherosclerosis.

**Gastrointestinal System:** The tongue was furred and had prominent papillae. The esophageal mucosa was hyperaemic. The

stomach contained about 250mls of semisolid food particles. The gastric mucosa was hyperaemic with normal rugae pattern. The small and large intestines were grossly normal. The rectum and anus were unremarkable. The liver was enlarged and weighed 1650g. The Gleason capsule was smooth and cut surfaces showed a variegated mottled appearance, reflecting hemorrhage and necrosis in the centrilobular regions (‘nut meg’ appearance). The gall bladder appeared normal in size and contained about 15mls of bile that drained freely at the ampulla of Vater.

**Lymphoreticular System:** The thymus was vestigial. The tonsils and neck glands were grossly normal. The Spleen was enlarged, and weighed 300g. The cut sections were dark brown in appearance with prominent red follicles and showed rolled out cut edges.

**Endocrine System:** The thyroid gland was bilobed and grossly normal. The cut sections showed unremarkable colloid material in circumscribed areas. The pancreas weighed 150g and was grossly normal as well as its cut section. The adrenal glands were grossly unremarkable. Cut sections showed golden yellow surfaces.

**Genitourinary System:** The kidneys were grossly normal in size. The left kidney weighed 160g and the right kidney weighed 150g. The renal capsules stripped with relative difficulty and showed fine granular subcapsular surfaces. Both surfaces also showed cortical scars of varying sizes. Cut surfaces showed marked corticomedullary differentiation. The calyces, pelvis and ureter were unremarkable. The ureters were patent also. The urinary bladder mucosa was unremarkable. The prostate and seminal vesicles were grossly normal. Both testes were present in the scrotum and were of normal size and shape. The external genitalia were unremarkable.

**Musculoskeletal System:** The muscle bulks were largely preserved. No fractures of the long bones were noted.

**Central Nervous System:** The cranium was intact with no fracture in the skull. The meninges were shiny with prominent arachnoid granulation. The brain was heavy and weighed 1600g. There was no contusion or softening, and the sulci were narrowed and gyri flattened. There was no significant tonsillar herniation. The cranial vessels had a normal anatomical distribution with no aneurismal dilatation and no significant atheroma. Serial coronal sections of the cerebral hemispheres reveal well differentiated white and gray mater. The cerebellum and brainstem appear unremarkable.

**Histological Findings**

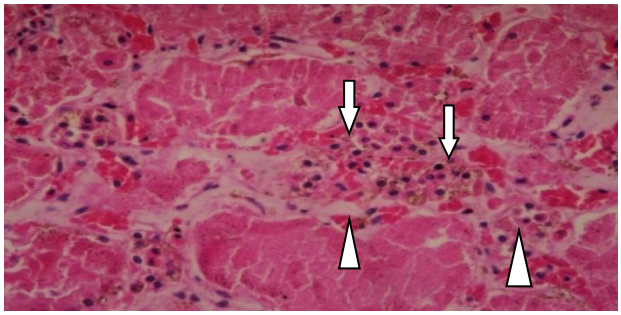
**Lungs:** Histologic section of the lungs showed alveoli lined by pneumocytes containing amorphous eosinophilic material. Some of the alveoli contain inflammatory cells predominantly polymorphonuclear inflammatory cells. Also seen were haemosiderin ladened macrophages (heart failure cells) as well as congested blood vessels.

**Liver:** Histologic section of the liver showed focal areas of necrosis with intervening viable periportal areas, containing benign plates of hepatocytes with congested blood vessels.

**Spleen:** Sections showed fibrocollagenous capsule with trabecular ramifying into the substance of the spleen. The splenic matrixes were populated by mixture of lymphocytes, macrophages, fibroblast and red cells.

**Kidney:** Sections of the kidney showed numerous normal glomeruli. The renal vascular channels showed hyperplastic arteriolitis. Most of the renal tubules were lined by pale amorphous ghost outline of cells, most of which were sloughing off and forming eosinophilic cast in the lumen. Also there was thickening of the tubular basement membrane. Within the edematous interstitium were noted pale amorphous material with few infiltrates of haemosiderin ladened macrophages and lymphocyte inflammatory cells.

**Brain:** Histologic section of the brain showed moderate cerebral oedema.
**Final Diagnosis:** Hypertensive cardiovascular disease.
**Cause of Death:** (a) Congestive cardiac failure. (b) Hypertensive cardiovascular disease



Micrograph 1 (x 400): Histologic section of the kidney showing sloughing tubular epithelium forming intraluminal eosinophilic cast (arrow) and haemosiderin laddened macrophages (arrow head).

**Case Two: Hypertensive Cardiovascular Disease with Massive Intracranial Haemorrhage**

**Clinical Notes:** A 60 year old unconscious woman was referred from a peripheral clinic on account of 5 days history of headache and a day history of loss of consciousness and vomiting with faecal incontinence, to the accident and emergency Department of the Madonna Hospital, Umuahia, Nigeria. There was associated history of cough, mild chest pain but no dyspnoea or paroxysmal nocturnal dyspnoea. She was not a known diabetic. Physical examination revealed conjunctival pallor, jaundice, no dehydration and lymph node group swelling. There was pitting pedal edema. Chest examination revealed a respiratory rate of 30 cycles per minute, pulse rate of 107 beats per minute and blood pressure of 230/110 mmHg. Abdominal examination showed liver of 2 cm below the right costal margin. Examination of the central nervous system showed Glasgow coma scale (GCS) of 3. Laboratory investigations done showed a full blood count-packed cell volume of 31%, white blood cell count of 2,700/mm3, lymphocytes - 28.6%, neutrophils - 70%; Erythrocyte sedimentation rate - 80mm in the first 1 hour; Electrolyte, urea and creatinine: Na - 134mmol/l, K - 6.7mmol/l, $HCO_3$ - 20mmol/l, Cl - 105mmol/l, urea - 3.1mg/dl, and creatinine - 160 mg/dl. An assessment of cerebrovascular accident secondary to severe hypertension, complicated by aspiration pneumonitis was made. She was placed on intranasal $O_2$, intravenous fluid, antibiotics and other supportive management. The patient's condition however deteriorated within 6 hours of admission and suddenly seized breathing and efforts made to revive her proved abortive. She was confirmed dead on the 20th March 2019.

**Autopsy Findings**

**External Description:** The body was that of 60 year old woman of African descent. There was no pallor and no jaundice. There was pitting pedal edema up to the knee. There was no lymphadenopathy in any of the major lymph node groups.
Thorax, abdominal and pelvic organs in situ: There was no demonstrable pneumothorax. All the organs were in their respective normal anatomical positions. There was no abnormal fluid accumulation in the serous cavities.
**Respiratory System:** The tracheobronchial tree was patent up to the terminal bronchioles. The mucosa of trachea and bronchi were hyperaemic. The pleural surfaces were shiny and smooth. There were no adhesions. The left and right lungs weigh 500mg and 550mg respectively and had spongy consistency. Cut surfaces of the lungs were uniformly greyish brown, with frothy fluid exuding from the surface and bits from all the lobes floating in water.
**Cardiovascular System:** The heart appeared grossly normal in size and weighed 400g. The inner pericardial membrane was smooth and shiny and not attached to the visceral pericardium. The pericardial space contained 50mls of clear fluid. The tricuspid, pulmonary, mitral and aortic valves had circumferences of 12cm, 7.5cm, 10cm and 7cm respectively. The left ventricular free wall measured 1.5cm and

the right ventricular free wall measured 0.4cm. The papillary muscle external diameter measured 1.4cm. The coronary arteries showed normal distribution and about 45% occluded by atheromatous plaque. There were no congenital heart abnormalities. The great vessels had normal anatomic origin. Serial sections of the myocardium showed no evidence of old or recent ischaemic injuries. The abdominal aorta showed atheromatous plaque Grade IV.

**Gastrointestinal System:** The mouth, tongue, teeth, pharynx and esophagus were unremarkable. The stomach contained about 100mls of semisolid particles and had a healthy mucosa. The small and large intestine, rectum and anus were essentially normal. The liver weighed 1250g. It had a smooth reddish-brown surface and a firm consistency. Serial sections were unremarkable. The gall bladder emptied readily into the duodenum; it contained clear bile and no gall stones were present. The pancreas weighed 100mg with a yellowish lobulated surface and rubbery consistency. The cut surfaces were unremarkable.

**Lymphoreticular System:** The tonsil was grossly normal. The thymus was unidentifiable. The spleen weighed 250g; it has a grey smooth capsule and a rubbery consistency. Cut surface showed a dark red wet appearance. The lymph node groups were not enlarged.

**Genitourinary System:** The renal capsules stripped with ease to reveal a smooth brownish subcapsular surface. Both kidneys weighed 100g each. Cut surfaces showed mild corticomedullary differentiation. The renal papillae, pelvis and ureters were grossly normal. The urinary bladder had a wall thickness of 0.8cm and had a healthy mucosa.

**Endocrine System:** The adrenals were grossly normal. Cut section showed golden yellow appearance. The thyroid gland was bilobed and grossly normal and cut sections were unremarkable. The pituitary gland was unremarkable.

**Reproductive System:** The uterus measured 5 x 3 x 2.5cm and the cervix was unremarkable. Section showed normal appearing endometrium and myometrium. The left and right fallopian tubes measured 8cm each. The corresponding ovaries measured 2.5 x 2 x 1.5cm each.

**Musculoskeletal System:** The muscle bulks were largely preserved. No fractures of the long bones noted.

**Central Nervous System:** The scalp and cranium were intact and no fracture seen. The brain weighed 1100g and showed flattening of gyri and narrowing of sulci. The meningeal coverings were transparent. There was no significant tonsillar herniation. The cranial vessels were however congested/prominent without aneurysmal dilatation and there was narrowing of the cerebral vessels with about 45% occlusion by atheromatous plaques. There was no epidural and subdural haemorrhage. Serial coronal sections of the cerebral hemisphere revealed massive intracerebral and intraventricular haemorrhage. There was also haemorrhage into the pons. The cerebellum appeared normal.

**Histological Findings**

**Lungs:** Section of lung tissue showed alveoli filled with pale amorphous substances and congested vascular channels.

**Liver:** Histologic section of the liver showed benign plates of hepatocytes and congested vascular channels.

**Spleen:** Histologic section of the spleen showed normal parenchyma and sinuses.

**Kidney:** Section of kidney showed numerous normal appearing glomeruli, tubules and renal parenchyma. Some of the renal tubules were lined by pale amorphous ghost outline of cells, most of which were sloughing off and forming eosinophilic cast in the lumen.

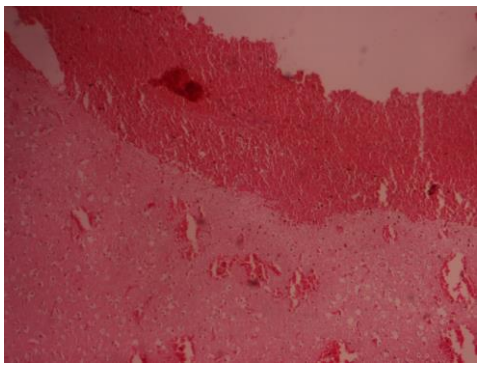
**Brain**

Histologic section of brain tissue showed extensive areas of haemorrhage within the brain parenchyma. There were clear halos around neuronal cell bodies with widening

of the Virchow's Robbins perivascular spaces and diffuse pale staining eosinophilic brain staining substances. There were no inflammatory cells.

**Final Diagnosis:** Hypertensive cardiovascular disease with massive intracranial haemorrhage.

**Cause of Death:** (a) Massive Intracranial Haemorrhage (b) Hypertensive Cardiovascular Disease



Micrograph 2 (x100): Section of brain showed extensive intracerebral haemorrhage

## DISCUSSION

In the autopsy performed on a 51 year old man who died of congestive cardiac failure, he was pale with mild peripheral and central cyanosis, and pitting pedal edema up to the knee. The coronary arteries and vessels were patent and had normal anatomical distribution. Before his death, this patient was diagnosed 12 years ago with hypertension. He had difficulty with breathing, moderate exercise intolerance, and mild paroxysmal nocturnal dyspnoea. Long term high blood pressure aggravated his condition which led to congestive heart failure. Chien-Chieng et al.[5] reported in their study that there was an increased risk of congestive heart failure which was associated with carbon monoxide poisoning. They suggested close follow-up of cardiac function for patients exposed to high levels of carbon monoxide. According to an autopsy report by Akang et al.[8], intake of alcohol and recreational drugs were contributing factors to death from cardiovascular heart disease. In our study, both lungs were subcrepitant and the cut surfaces of both lungs showed exudation of free frothy fluid and bits from all the lobes floats in water. The pericardium of the cardiovascular system was smooth and shiny and was covered by moderate pericardial fat. The heart was grossly enlarged and weighed 450g. This case of a 51 year old man of African origin with 12 years history of poorly controlled high blood pressure presented a hypertensive heart disease, a common complication of chronicity. The progressive leg swelling, exercise intolerance, dyspnoea, paroxysmal nocturnal dyspnoea and body weakness resulted from heart failure. Several autopsy reports[9-11] have revealed similar findings in people with cardiovascular heart disease. This is also evident in tachypnea, tachycardia, raised JVP, S3 heart sound and hepatomegaly in the patient. The progressive ascites and pitting pedal edema resulted from failed renin-angiotensin-aldosterone effect in control of body fluid due to the failing kidney. The autopsy revealed flabby and grossly enlarged heart, pleural effusion, pulmonary edema, chronic passive congestion in the liver congestion in the spleen and ascites. These are commonly associated with congestive heart failure. The shocked kidneys are caused by the effect of the long-standing hypertension on the kidney, in the form of benign nephrosclerosis, acute tubular necrosis. Aligbe et al. [12] have indicated an association of renal complications in autopsies of death from hypertensive cardiovascular diseases. This is confirmed on the microscopic hyperplastic arteriolosclerosis and acute tubular necrosis progressing to renal failure. The progressive damage to the kidney and the decompensated heart (congestive heart failure) lead to severe fluid accumulation as seen in extension to the lungs and brain in this patient leading to the death of the patient. Kade et al.[13] published a case report of a 33 year-old man who was diagnosed with mild rhabdomyolysis with acute kidney failure following exposure to carbon monoxide. The kidney failure

resolved in 2 weeks after exposure to hyperbaric oxygen. This supports the theory that exposure to carbon monoxide could have contributed to the aggravation of the condition leading to the death of this subject.

In the case of a 60 year old woman who died of hypertensive cardiovascular disease with massive intracranial haemorrhage, the brain weighed 1100g and showed flattening of gyri and narrowing of sulci. The meningeal coverings were transparent and there was no significant uncal or tonsillar herniation. The cranial vessels were however congested without aneurysmal dilatation and there was narrowing of the cerebral vessels with about 45% occlusion by atheromatous plaques. Long-term exposure to air pollutants could be a risk factor that worsened the condition of this patient. Dorairaj et al.[14] reported a strong association between high levels of ambient air pollution, higher systolic blood pressure and incident hypertension. There was no epidural and subdural haemorrhage in this woman and the serial coronal sections of the cerebral hemisphere revealed massive intracerebral and intraventricular haemorrhage. Dan et al.[15] reported intracranial haemorrhages in the autopsy of a 74 year old patient diagnosed with hypertensive cardiovascular disease. Histologic section of her brain tissue showed extensive areas of haemorrhage within the brain parenchyma. Several studies[16,17] have maintained that chronic high blood pressure has been the major predisposing factor of death from hypertensive cardiovascular disease. The American heart association has long considered blood pressure less than 140 over 90mmHg as normal for adults. Spontaneous (non-traumatic) intraparenchymal hemorrhages occur most commonly in middle to late adult life, with peak incidence at about 60 years. [7] Hypertension is the most common underlying cause of primary brain parenchymal hemorrhage, accounting for more than 50% of clinically significant hemorrhages and for roughly 15% of deaths among individuals with chronic hypertension. [2] Hypertension causes a number of abnormalities in vessel walls, including accelerated atherosclerosis in larger arteries as seen in this case; hyaline arteriolosclerosis in smaller vessels; and, in severe cases, proliferative changes and frank necrosis of arterioles. This has been revealed in several autopsy reports[15,18,19] of hypertensive cardiovascular disease.

## CONCLUSION

The 51 year old man who died of hypertensive cardiovascular disease was also found to have moderate cerebral edema, pulmonary edema and chronic pyelonephritis. The 60 year old woman in addition to hypertensive cardiovascular disease was found to have a massive intracerebral haemorrhage and pulmonary edema. A thorough autopsy of deaths from hypertensive cardiovascular disease was recommended as this will reveal other associated pathologies from other organs of the body.

**Conflict of Interest:** None

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