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Autopsy Pathology of Diabetic Nephropathy with Hypertensive Cardiovascular Disease and Anemic Heart Failure

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ABSTRACT

An autopsy was performed at Abia State University Teaching Hospital, Aba, Nigeria on a 58 year old woman who was known to be diabetic and hypertensive. Results of autopsy findings showed that the external description was cachectic, severely pale, cyanosed and jaundiced. The thorax, abdominal and pelvic organs were in their normal anatomic position. The trachea mucosa was hyperemic. The bronchi contained frothy fluid. The parietal pleural was shiny and smooth. The heart was enlarged and flabby. The parietal pericardium was smooth and shiny and not attached to the visceral pericardium. The tongue was pale, furred and have prominent papillae. The esophageal mucosa was hyperaemic. The cut sections of the liver showed a nutmeg appearance. The thymus was vestigial. The cut surfaces were cream colored and solid. The thyroid was grossly normal in size. The cut surfaces were brownish and showed colloid in areas. Both kidneys were grossly normal. The cut sections showed loss of corticomedullary differentiation. There were bilateral multicystic swellings on both ovaries. The cranium was intact with no fracture in the skull. The cerebral vessels showed few fatty streaks but no aneurysmal dilatation. Histologic section of the kidney showed numerous glomeruli with varying degree of sclerosis, and capillary basement membrane thickening. The cause of death was anemic heart failure, metastatic serous cystadenocarcinoma, and diabetic nephropathy with hypertensive cardiovascular disease. Thorough autopsies on all deaths of heart failure to study other associated pathologies was recommended.

Keywords: Autopsy, Anemic Heart Failure, Hypertensive Cardiovascular Disease, Diabetic Nephropathy, Histology

INTRODUCTION

Diabetic nephropathy is a serious complication of type 1 diabetes and type 2 diabetes. It's also called diabetic kidney disease. [1] Diabetic nephropathy affects the kidneys' ability to do their usual work of removing waste products and extra fluid from your body. Over many years, the condition slowly damages the kidneys' delicate filtering system. Early treatment may prevent or slow the disease's progress and reduce the chance of complications. [2] Kidney disease may progress to kidney failure, also called end-stage kidney disease. Kidney failure is a life-threatening condition. At this stage, treatment options are dialysis or a kidney transplant. [3] In the early stages of diabetic nephropathy, patients may not notice any signs or symptoms. In later stages, signs and symptoms may include worsening blood pressure control, protein in the urine, swelling of feet, ankles, hands or eyes, increased need to urinate, reduced need for insulin or diabetes medicine, confusion or difficulty concentrating, shortness of breath,

loss of appetite, nausea and vomiting, persistent itching, and fatigue. [4]

Anemia has been found to be a common complication of chronic heart failure, reducing oxygen delivery to the periphery. [5] Erythropoietin to correct anemia has a long history in the management of renal failure with complicating anemia. Chronic heart failure (CHF) is frequently associated with poor exercise tolerance and debilitating symptoms despite optimal modern treatment. [6] In the past we expected that this was due to the direct consequences of poor cardiac output and congested lungs. Other important pathophysiological disturbances in CHF occur as both short and long term consequences of the initial cardiac dysfunction. These include neurohormonal activation, cytokine release and trophic changes in skeletal muscle and the peripheral vasculature, and disturbances in reflex control systems. [7] The severity of symptomatic exercise limitation varies between patients, and bears little relation to the extent of the left ventricular systolic dysfunction measured at rest, or to markers of central haemodynamic disturbance. [8] Although there are several reasons for this, a research [9] has suggested that changes which occur in the periphery as a consequence of the systemic effects of heart failure, may have become the principal factors limiting exercise. [10] Many changes have been described in skeletal muscle, peripheral arterial and endothelial function, and in reflex cardiopulmonary control systems. These have all been implicated in being limiting features to exercise tolerance in CHF patients. In its most severe form this can lead to cardiac cachexia. [11] Major abnormalities in anabolic and catabolic hormones and in immune activation have been proposed to explain these developments in CHF. [11] Therapeutic advances in managing heart failure have followed these pathophysiological studies, as neurohormonal blockade rather than positive inotropic treatment is now the mainstay of CHF therapy.

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. 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. [12] Chronic high blood pressure puts a strain on the heart and makes it harder for it to pump blood. However, people who manage their high blood pressure can greatly reduce their risk of heart failure. 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. [13] An Autopsy is performed on a dead body primarily to determine the cause of death. This study reviews a case report on the autopsy reports of death from diabetic nephropathy with hypertensive cardiovascular disease and anemic heart failure at Abia State University Teaching Hospital, Nigeria.

MATERIALS AND METHODS

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

RESULTS

Clinical Notes:

A 58 year old known diabetic and hypertensive woman of African descent presented to the medical outpatient Department of Ohafia General Hospital, Abia State, Nigeria with anorexia of 3 months duration, associated with constipation and progressive abdominal

distension. There was associated progressive weight loss, and occasional low grade fever. She was a known diabetic and hypertensive for about 6 years, diagnosed in a peripheral clinic. She came with computer tomography scan report that revealed intra-abdominal tumour in addition to the presenting complaints. On examination, she was moderately pale, anicteric with gross ascites and pitting pedal edema up to the knee. There was no lymph node group enlargement. Chest examination showed respiratory rate of 28 cycles per minute, bronchovesicular breath sound, low pulse volume and pulse rate of 96 beats per minute. The blood pressure of 150/100mmHg was recorded. The heart sounds recorded were S1 and S2. Abdominal examination showed gross ascites with nonpalpable abdominal viscera. Laboratory investigations done showed random blood sugar on admission of 512mg/dl; haemoglobin estimation of 7mg/dl, packed cell volume of 20%, white cell count of 5.2 x 10^9, neutrophil count of 46%, and lymphocyte count of 53%. She was placed on antihypertensive drugs with diuretics and subcutaneous insulin. However, on the 5th day of admission, her condition continued to deteriorate and on the 7th day she was confirmed dead when all efforts to resuscitate her failed.

Autopsy Findings

External Description: The body of a middle aged woman of African descent. She was cachectic, severely pale, cyanosed and jaundiced. There was gross ascites with pitting pedal edema up to the knee. No lymphadenopathy in any of the major lymph node groups.

Thorax, Abdominal and Pelvic Organs In Situ: There was no demonstrable pneumothorax. The thorax, abdominal and pelvic organs were in their normal anatomic position. There was excess serous fluid in both pleural cavity measuring 350mls and ascites of about 5 liters of gelatinous tan colored fluid. The parietal peritoneum and the inferior surface of the diaphragm had numerous and varying sized pale white to yellow nodular deposits. The cut surfaces of these masses showed cream to grayish white surfaces.

Respiratory System: The tracheobronchial tree was patent down to the bronchioles. The trachea mucosa was hyperemic. The bronchi contained frothy fluid. The parietal pleural was shiny and smooth. The left and right lungs were shiny and heavy and weighed 250g and 260g respectively.

Cardiovascular System: The heart was enlarged and flabby. It weighed 450g. The parietal pericardium was smooth and shiny and not attached to the visceral pericardium. The tricuspid, pulmonary, mitral and aortic valves measured 13.5cm, 8cm, 9.5cm and 7cm respectively. The right ventricular free wall measured 0.4cm, while the left ventricular free wall measured 1.7cm, and one of the papillary muscles measured 1.6cm in external diameter. There were few fatty streaks in the aortic intima. The coronary arteries had normal distribution. The coronary ostia were patent. The atrial walls were free of patches or plaques. Serial sections of the myocardium showed no evidence of old or recent ischaemic injuries. The thoracic and the abdominal aorta showed severe atherosclerosis Grade 5.

Gastrointestinal System: The tongue was pale, furred and had prominent papillae. The esophageal mucosa was hyperaemic. The stomach contained about 250mls of semisolid food particles and the mucosa showed focal areas of hyperaemia. The duodenal, jejunal, ileal and colonic mucosa were grossly normal. The liver was enlarged and weighed 1700g. The Gleason capsule was smooth. The liver took on a variegated mottled appearance, reflecting hemorrhage and necrosis in the centrilobular regions. The cut sections of the liver showed a nutmeg appearance. The gall bladder was grossly normal, contained about 10mls of bile draining freely at the ampular of Vater. No stones were seen.

Lymphoreticular System: The thymus was vestigial. The tonsils and neck glands were grossly normal. There were 4 enlarged mesenteric lymph nodes with the largest one measuring 3x3x2.5cm and the smallest one measure 2x2x1cm. The cut surfaces were cream colored and solid. The spleen was enlarged and weighed 250g. The capsule was brownish and smooth. Cut surfaces were dark brown in appearance and cut edges were rolled out.

Endocrine System: The thyroid was grossly normal in size. The cut surfaces were brownish and showed colloid in areas. The pancreas was unremarkable and weighed 80g. The cut section was uniformly tan colored. The adrenals were unremarkable. The cut surface was golden yellow in appearance.

Genitourinary System: Both kidneys were grossly normal. The left kidney weighed 160g and the right kidney weighed 150g. The capsules stripped with ease revealing fine granular subcapsular surfaces. The cut sections showed loss of corticomedullary differentiation. The pelvicalyceal systems were widened and the ureters were patent. The urinary bladder mucosa was unremarkable.

Reproductive System: There were bilateral multicystic swellings on both ovaries. The left ovarian mass measured 10x7x6cm and weighed 25g while the right ovarian mass measured 14x10x9cm and weighed 30g. The cut surfaces of these masses showed multicystic spaces containing serous fluid. The cyst wall measured 0.6cm uniformly. The fallopian tubes were grossly normal and measure 12cm each. The uterus was grossly normal and measured 10x7x5cm, and harbored two intramural fibroid nodules measuring 4x3x3cm each. The cervix was unremarkable. The vagina wall and mucosa 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, no fracture in the skull. The meninges were smooth and shiny. The brain was bilaterally symmetrical and showed narrow sulci and flat gyri. The cerebral vessels showed few fatty streaks but no aneurysmal dilatation. There was no uncal or tonsillar herniation. Serial sections of the cerebrum showed well differentiated white and gray matter. The cerebellum and midbrain were unremarkable.

Histological Findings

Ovarian Tumour: Histologic section of the ovary showed varying sized cystic cavities lined by atypical serous epithelial cells, with pleomorphic and hyperchromatic nuclei forming micropapillae in some areas. Also in the fibrous stroma were smaller malignant glands. The invasive tumor formed epithelioid cell clusters with a generous amount of eosinophilic cytoplasm. There was minimal inflammatory reaction.

Nodular Masses: Section of the tissue showed well circumscribed tumor nodule containing sheets, nest and cords of malignant epithelial cells forming tubules in places as seen in the ovary. There was prominent tissue desmoplasia.

Lymph Nodes: Histologic sections of lymph nodes showed distorted architecture by sheets and nests of pleomorphic and hyperchromatic epithelial cells similar to the ovarian tumor cells.

Lungs: Histologic section showed alveoli lined by pneumocytes containing amorphous eosinophilic material. Some of the alveoli contained inflammatory cells predominantly polymorphonuclear inflammatory cells. Also seen were congested blood vessels. No tumor cells were seen.

Liver: Histologic section of the liver showed foci of necrosis surrounded by edematous stroma containing benign plates of hepatocytes. The vascular channels were congested.

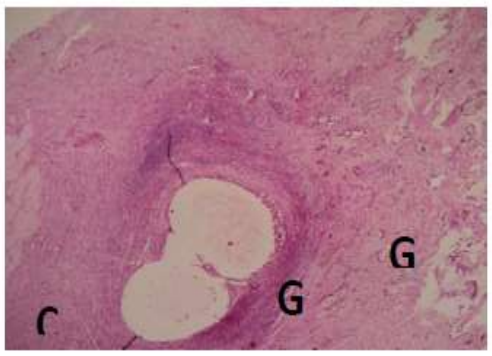
Spleen: Sections showed fibrocollagenous capsule with trabecular ramifying into the substance of the spleen. The splenic matrixes were populated by mixture of lymphocytes, macrophages, fibroblast and red cells.

Kidney: Histologic section of the kidney showed numerous glomeruli with varying degree of sclerosis, and capillary basement membrane thickening. There was proliferation of mesangial cells and accumulation of pale eosinophilic material in the Bowman's space. The renal vascular channels showed hyaline and hyperplastic arteriolosclerosis. Most of the renal tubules were lined by pale amorphous ghost outline of cells, most of which were sloughing off. Also there was thickening of the tubular basement membrane. Within the interstitium were pale amorphous material with few infiltrates of chronic inflammatory cells.

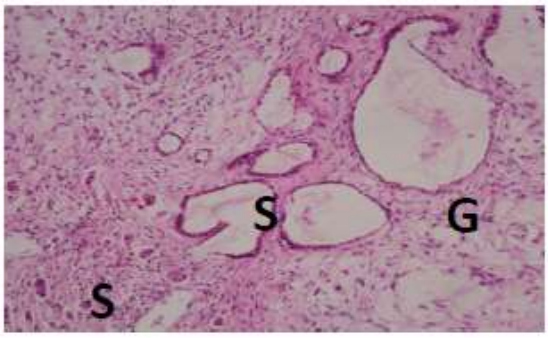
Brain: Histologic section of the brain showed moderate cerebral edema. No metastatic deposits were seen.

Final Diagnosis: Metastatic serous cystadenocarcinoma with diabetic nephropathy and severe anemia.

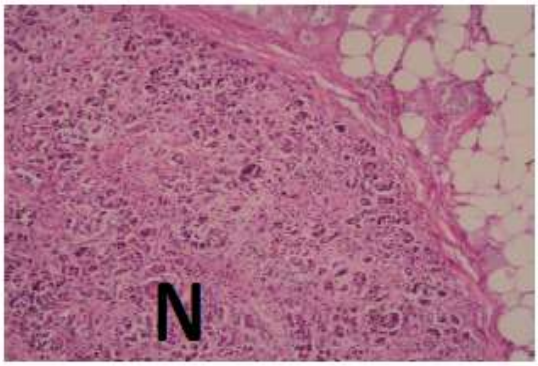
Cause of Death: (a) Anemic heart failure
(b) Metastatic serous cystadenocarcinoma
(c) Diabetic nephropathy with hypertensive cardiovascular disease



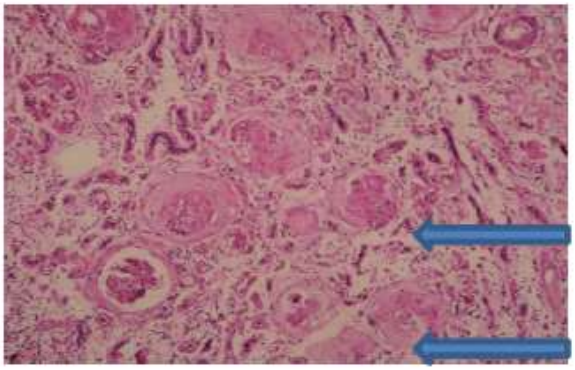
Micrograph 1 (x100): Section ovary showing corpora albecantes (C) and malignant serous glands in fibrous ovarian stroma (G).



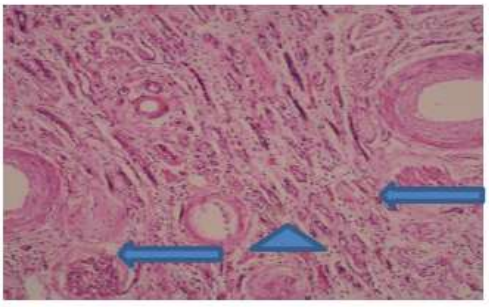
Micrograph 2 (x400): Section of ovary showing malignant serous glands(G) and stromal invasion(S)



Micrograph 3 (x100): Well circumscribed tumour nodule (N) within omental tissue



Micrograph 4 (x100): section of kidney show glomerular sclerosis (arrows), edematous interstitium with mixed inflammatory cells infiltrate



Micrograph 5 (x100): Section of kidney, showing hyperplastic arteriolitis (arrows) and thickened glomerular basement membrane (arrow head)

DISCUSSION

In the case of a 58 year old woman who died of diabetic nephropathy with hypertensive cardiovascular disease, histologic section of the lungs showed alveoli lined by pneumocytes containing amorphous eosinophilic material. Some of the alveoli contained inflammatory cells predominantly polymorphonuclear inflammatory cells. Also seen were congested blood vessels. Obiajulu et al.[14] reported similar findings in an autopsy report carried out at in Lagos, Nigeria. In Nigeria, environmental pollution is a major factor that contributes to lung and renal diseases. People live their whole lives inhaling environmental pollutants such as carbon monoxide, nitrogen dioxide and particulate matter. Other pollutants that people are exposed to include lead, arsenic, particulate matter and sulfur dioxide. Nwachokor et al. [15] reported that exposure to environmental pollutants were secondary contributing factors that led to the death of many residents. These residents recorded similar clinical findings as seen in this study. Autopsy of this study revealed that the trachea mucosa was hyperaemic, the bronchi contained frothy fluid, and the parietal pleural was shiny and smooth. The left and right lungs were shiny and heavy and weighed 250g and 260g respectively. The heart was enlarged and flabby, and weighed 450g. These were also reported in similar studies carried out in the autopsy pathology of dead bodies in which diabetes and kidney damage were diagnosed. [16,17] The parietal pericardium in this study was smooth and shiny and not attached to the visceral pericardium. There were few fatty streaks in the aortic intima and the coronary arteries had normal distribution. The atrial walls were free of patches or plaques. Dorairaj et al. [18] have reported an association of elevated blood pressure and diabetes mellitus in an autopsy performed in India.

The patient in our study was a 58 year old known diabetic and hypertensive female of African descent who presented with intra-abdominal mass, anorexia, constipation and progressive abdominal distension. Autopsy findings of hypertensive heart disease reported by Kade et al. [19] showed the chronicity of the hypertension and diabetes mellitus in their patient, resulting in decompensatory changes of flabby heart and hypertrophy. The failed heart in our autopsy resulted in accumulation of fluid in tissue interstitium and potential spaces. This can be worsened by ascites which is also commonly associated with ovarian tumors. Serous adenocarcinoma present in this patient added significantly to the disease burden in the patient. Serous adenocarcinoma was commonly associated with peritoneal carcinomatosis as evident by tumor spread to the peritoneum on autopsy. This was collaborated by Aligbe et al. [20] in their study in which there was an elaboration of cytokines and inflammatory mediators leading to tumor cachexia, anorexia, and fever as seen in their patient. The poorly controlled diabetes and hypertension in our patient affected the kidneys in the form of diabetic nephropathy and nephrosclerosis. The histologic section of the kidney showed numerous glomeruli with varying degrees of sclerosis, thickened capillary basement membrane and hyperplastic and hyaline arteriolosclerotic changes. This change resulted in the shocked kidney and probably anemia due to significant failure of erythropoietin production. Several autopsy reports [21,22] have reported an association of anemia with kidney failure and in all cases, exposure to air pollutants was reported as secondary contributory factors to the cause of death.

CONCLUSION

The autopsy report of this 58 year old woman revealed that she died of anaemic heart failure on a background of metastatic serous cystadenocarcinoma, diabetic nephropathy and hypertensive cardiovascular disease. It is strongly recommended that autopsies be performed in all deaths of heart failure to study other associated pathologies.

Declaration by Authors
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Conflict of Interest: The authors declare no conflict of interest.

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