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Cardiac metastasis in 42-years-old man with osteosarcoma right proximal tibia presenting with dyspnea and hypotension: A rare case report

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Abstract

Cardiac metastasis is rare. A 42-years-old nursing officer was a known case of osteosarcoma proximal tibia diagnosed one year ago; treated with curettage and chemotherapy. He had bilateral pedal oedema with exertional dyspnea for one month due to Doxorubicin induced cardio-depressant effect causing heart failure. For painful swelling at previous surgical site, above knee amputation was done. One week after surgery, he had increasing dyspnea for 3 days with drop in SaO_2% and high D dimer. Therefore, he was treated as a case of pulmonary embolism with low molecular weight heparin. And, he developed hematemesis and melaena. HRCT chest revealed pulmonary metastasis; lower limb doppler was normal. Echocardiogram showed echogenic mass in right ventricle (3.5 cm x 5.5 cm) suggestive of metastatic tumor in right ventricle. Later, he developed hypotension and succumbed. Autopsy findings revealed tumor mass (12.5 x 17.5 cm) in right ventricle wall extending into right ventricular cavity; multiple metastasis in both lungs, inner thoracic wall and liver. Histology findings were discussed.

Keywords: Osteogenic sarcoma; Dyspnea; Hypotension; Pulmonary embolism; Metastatic tumor in right ventricle.

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Introduction

Cardiac metastases are more common than primary cardiac tumors [1]. Most malignant tumors can metastasize to the heart. With the advances in cancer treatments, the incidence of cardiac metastases is increasing [2]. Detection of cardiac metastases is crucial for management and prognosis. Therefore, a transthoracic echocardiogram should be performed in cancer patients particularly if they have any cardiac warning signs; dyspnea, chest pain, or a new heart murmur. Clinical manifestations in patients with cardiac metastases depend on their size, location, and extent of infiltration. The most common site was pericardium [1,3]. They are usually silent. They are identified only in autopsy or they are incidentally discovered during tumor staging or follow-up evaluations [4]. Cardiac metastases from osteosarcoma are exceptional. Osteosarcoma is the most common primary bone malignancy characterized by a high degree of malignancy [5] having strong invasiveness and rapid disease progression. Therefore, mortality rate is extremely high. The common site of osteosarcoma is the metaphysis of long tubular bones. The main mode of spread is hematogenous spread; the imaging findings of cardiac involvement by osteosarcoma are seldom reported.

Case presentation

A 42-years-old nursing officer had swelling over right proximal tibia one year ago; it was found to be due to osteosarcoma. Extended curettage to tumor area, plate and screw fixation was done one year ago; six cycles of adjuvant chemotherapy (3-weekly AP regimen) were given with Doxorubicin and Cisplatin. Then, total 5 cycles of 1st line palliative chemotherapy were given for recurrent metastatic setting; initial 2 cycles included Doxorubicin and Isocyanide. And further 3 cycles were withheld as cumulated dose of Doxorubicin exceeded 400 mg/m^2. He suffered bilateral pedal oedema with exertional dyspnea for one month; it was probably due to cardio-depressant effect of Doxorubicin therapy which he received 3 months ago. Above knee amputation was done as he had massive swelling and pain around proximal tibia on right side, one year after initial diagnosis. Following surgery, he had increasing shortness of breath for 3 days with decreased SaO_2% and high D dimer (5.97 ug/ml). Therefore, he was treated as pulmonary embolism with low molecular weight heparin. And, he developed hematemesis and melaena. On examination, he had hypotension (99/72 mmHg) and tachycardia (111/min). SaO_2 was 99% with oxygen 5L/min. He was fully conscious; reduced air entry in right lower zone was noted. ECG revealed sinus tachycardia; heart rate was 100/minutes (Figure 1). Chest radiograph showed mild cardiomegaly, bilateral pleural effusion and multiple alveolar shadows in both lungs. High resolution CT (HRCT) chest revealed pulmonary metastasis; lower limb doppler was normal (Figures 3 & 4). Echocardiogram showed echogenic mass in right ventricle measuring 3.5 cm x 5.5 cm; no visible mass in pulmonary tree; mildly dilated right atrium and right ventricle; normal LV function (LVEF-60 %); bilateral moderate pleural effusion. It was suggestive of metastatic tumor in right ventricle (Figures 5,6,7). Full blood counts revealed hemoglobin was 10.7 gm%; total WBC count was 22.8 x 10^9/L (neutrophil leukocytosis); platelet count was 149 x 10^9/L. Blood urea was 18.8 mg/dL; serum creatinine was 0.63 mg%; serum sodium was 133 mmol/L; serum potassium was 3.12 mmol/L and serum chloride was 98 mmol/L. He was treated with antibiotics, proton pump inhibiter, tramadol, antacids, oxygen therapy, fluid and electrolyte correction and nutrition. However, his blood pressure was still low; tachycardiac and features of bilateral pleural effusion pronounced. Blood parameters were poor; hemoglobin was 11.8 gm%; Total WBC count was 36.8 x 10^9/L; platelets count was 166 x 10^9/L; Troponin T level was 19.8 ng/L (normal range 0-14); serum sodium was 135 mmol/L; serum potassium was 3.69 mmol/L; serum chloride was 94 mmol/L; blood urea was 21.28 mg/dL; serum creatinine was 0.49 mg%; blood uric acid was 3.21 mg/dL. The condition became deteriorated; blood pressure dropped further even with two inotropes; heart rate was increasing; dyspnea was more severe; and the patient expired. Autopsy showed a large fleshy mass measuring 12.5 x 17.5 cm over heart; it extended into right ventricle (Figures 8,9). Histology was compatible metastatic tumor (Figures 25,26,27). There was no pericardial effusion. There were yellowish blood stain pleural fluids in both lung; 1,000 cc in right pleural cavity and 800 cc in left thoracic cavity. Multiple metastatic nodules were found in the inner surface of thoracic cavity (Figure 10). There were multiple metastatic nodules in both lungs. Gross view and histology are demonstrated in Figures 13,14,22,23,24. There were multiple metastatic nodules in liver; they were seen in both capsular surface and cut section. Gross view and histology are demonstrated in Figures 11,12,17,18,19. Histology from metastatic lymph node is illustrated in Figures 20,21.

Discussion

Most malignant tumors can metastasize to the heart. Cardiac metastases are rare [1]. It is vital to detect cardiac metastases because it is important for management and prognosis. In cancer patients, a transthoracic echocardiogram should be performed in the presence of any cardiac warning sign, including dyspnea, chest pain, or a new heart murmur. It is important to diagnose any cardiac metastasis because it may worsen the patient's prognosis. They are usually silent and found in either routine checkup or postmortem examination [4]. Autopsy reports on incidence of cardiac metastases varied and their detection rate increased with advancement in imaging [2,3]. Cardiac manifestations in patients with cardiac metastases depend on their size, location, and extent of infiltration [1]. Most common site was pericardium [2,3]; it caused pericardial effusion and cardiac tamponade. Metastasis to myocardium was less common; it produced arrhythmia and heart failure [2]. Intracardiac tumor rarely produced symptom unless large [1]. Endocardial involvement resulted in valvular dysfunction and embolism. In this patient, cardiac metastasis was detected with transthoracic echocardiogram. In autopsy, the size of cardiac metastasis was nearly 4 times that of the size measured in echocardiography done one week earlier. Reported symptoms in patients with cardiac metastasis were chest pain, palpitation, arrhythmia and fever. Asymptomatic cases were detected in either routine examination or follow up examination [2,3]. In this patient, he had exertional dyspnea and pedal oedema one month. It was due to doxorubicin induced myocardial injury causing heart failure though initial echocardiogram was normal. Then, he developed severe dyspnea and hypoxia; it was probably due to pulmonary embolism as D dimer was high. Later, the blood pressure dropped. Presenting symptoms as hypotension/shock was

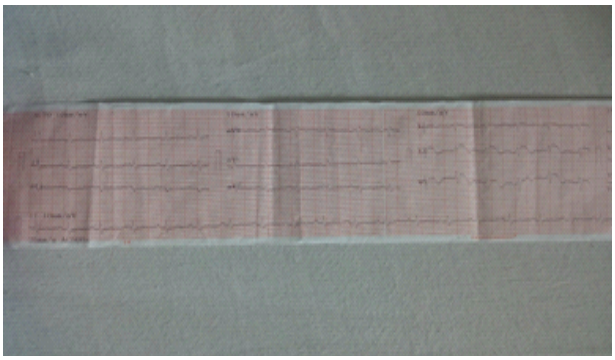

Figure 1: ECG showing sinus tachycardia, ST elevation in V2 & V3.

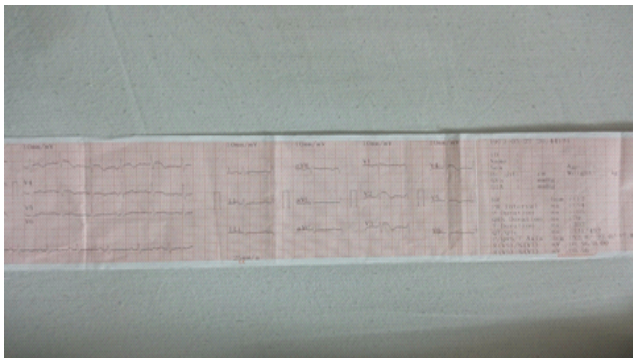

Figure 1.b: ECG showing sinus tachycardia, ST elevation in V2 & V3.

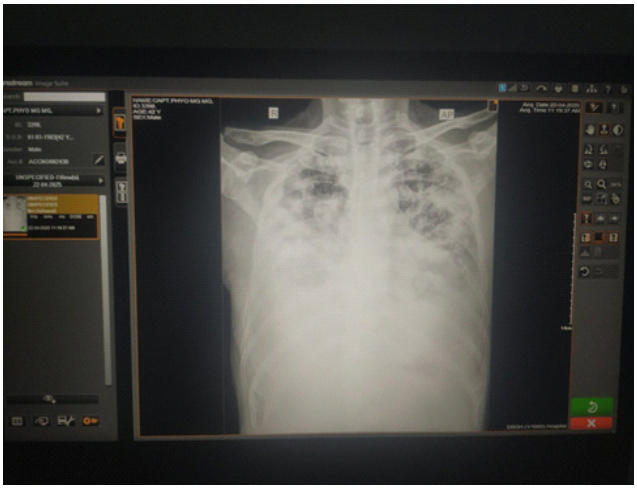

Figure 2: Chest radiograph showing cardiomegaly, bilateral pleural effusion, multiple alveolar shadows over both lungs.

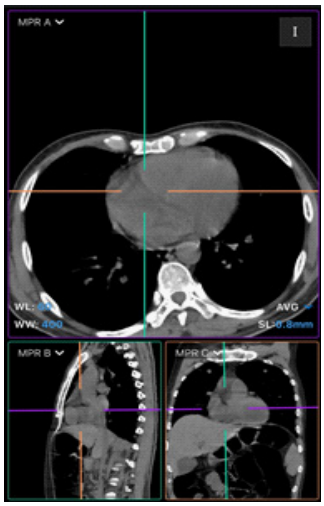

Figure 3a: HRCT chest and abdomen showing hypodense area on right side of right ventricle and normal liver.

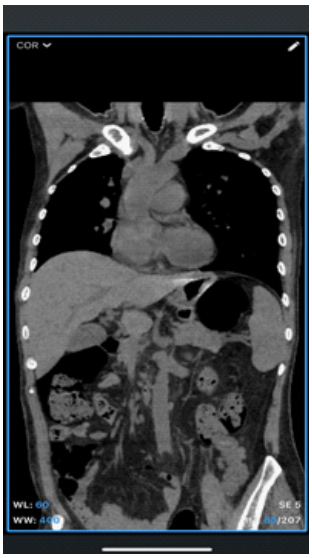

Figure 3b: Coronal view of HRCT chest and abdomen showing hypodense area on right side of right ventricle and normal liver.

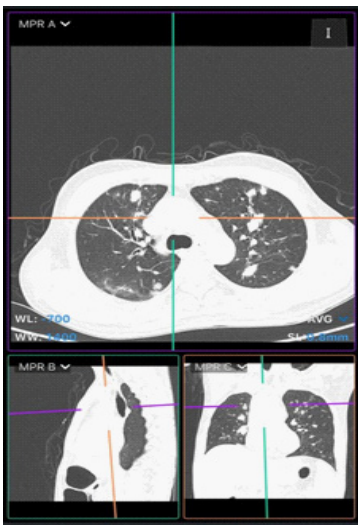

Figure 4: Cross sectional view of HRCT chest (Aortic window) showing rounded nodules more on right side suggestive of pulmonary metastasis.

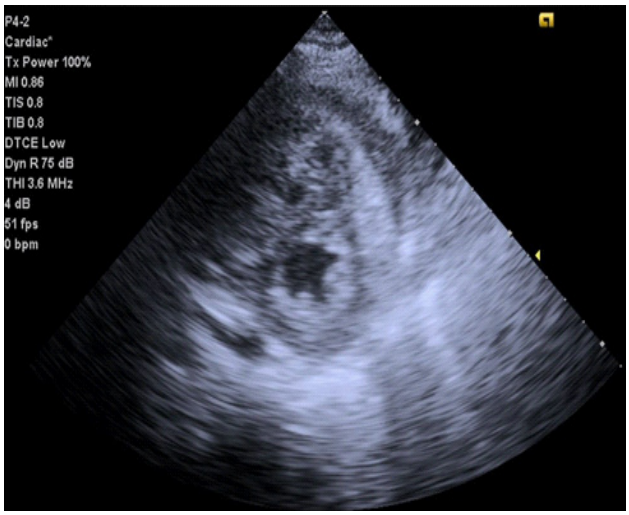

Figure 5: Echocardiogram demonstrating metastatic mass in the Right Ventricular cavity in parasternal short axis view.

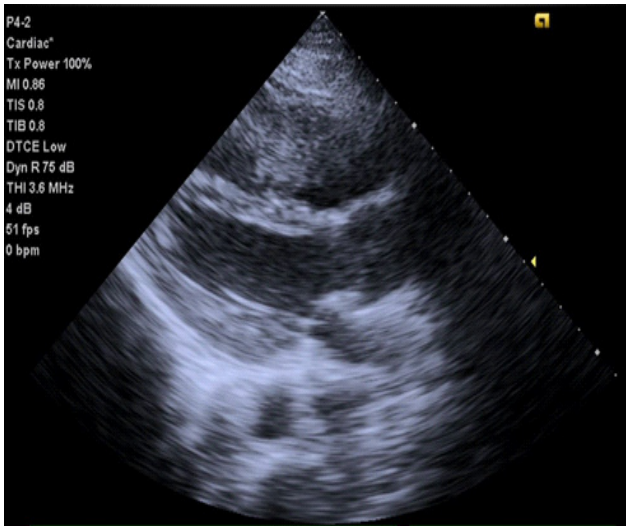

Figure 6: Echocardiogram revealing metastatic mass in Right Ventricular Cavity in parasternal long axis view.

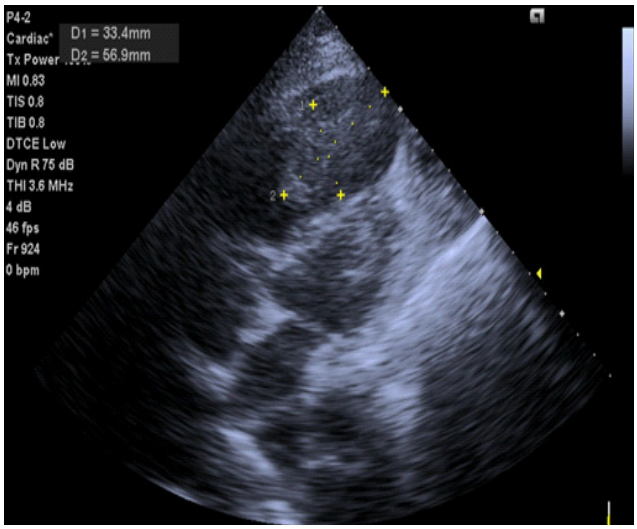

Figure 7: Echocardiogram showing 33.4 mm x 56.9 mm dimension of mass in right ventricular cavity.

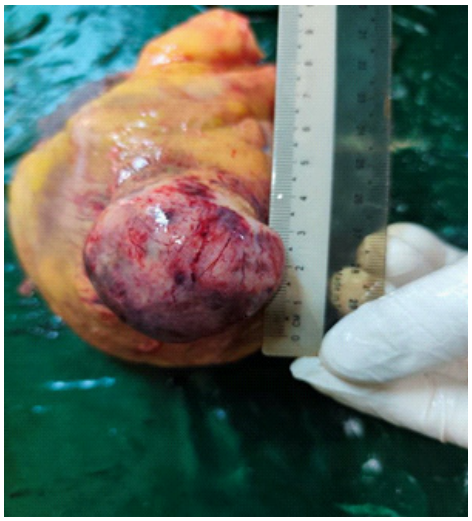

Figure 8a: Post-mortem examination of heart; a large fleshy mass measuring 12.5 x 17.5 cm over heart and congested vessels over the mass.

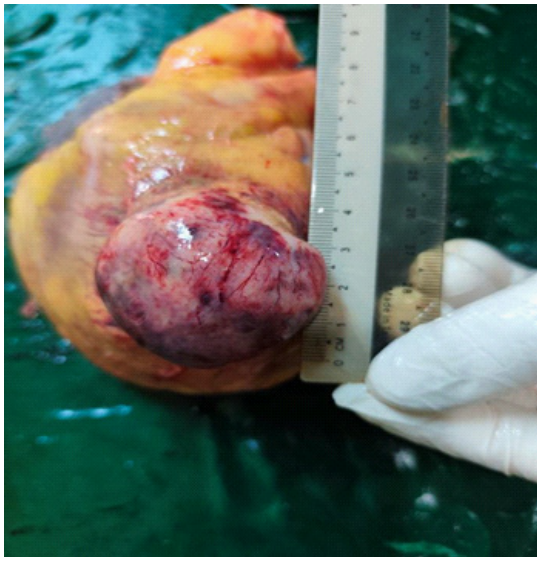

Figure 8b: Post-mortem examination of heart; a large fleshy mass measuring 6.5 x 17.5 cm over heart and congested vessels over the mass.

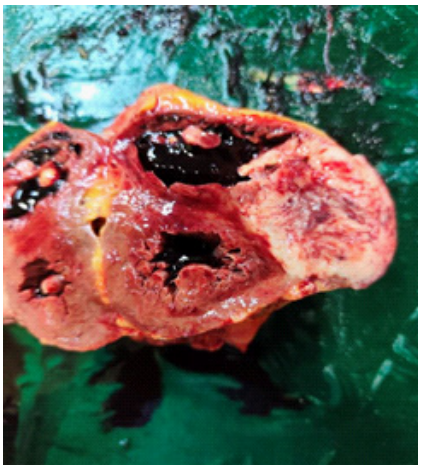

Figure 9: Post-mortem examination of cut section of heart; a large fleshy mass arising from myocardium of both right ventricle and left ventricle; invasion into endocardium of right ventricle.

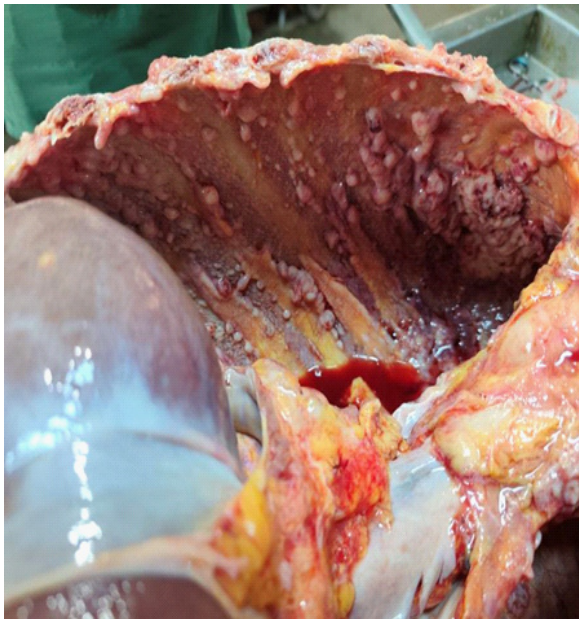

Figure 10a: Autopsy of inner surface of thoracic cavity showing multiple metastatic nodules over parietal pleura.

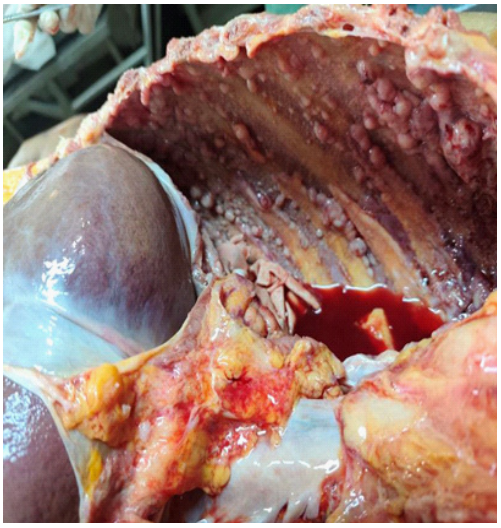

Figure 10b: Autopsy of inner surface of thoracic cavity showing multiple metastatic nodules over parietal pleura.

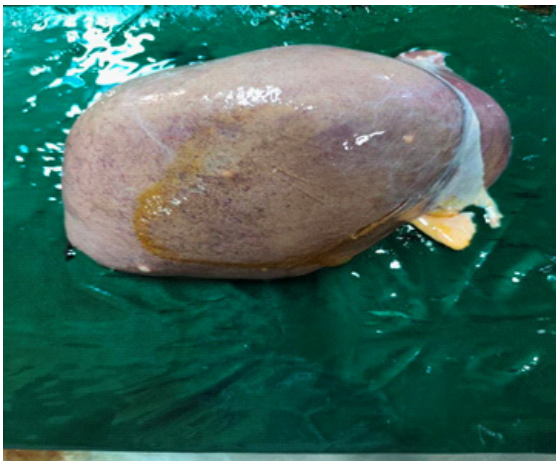

Figure 11: Autopsy of liver showing whitish multiple metastatic nodules over external surface of liver.

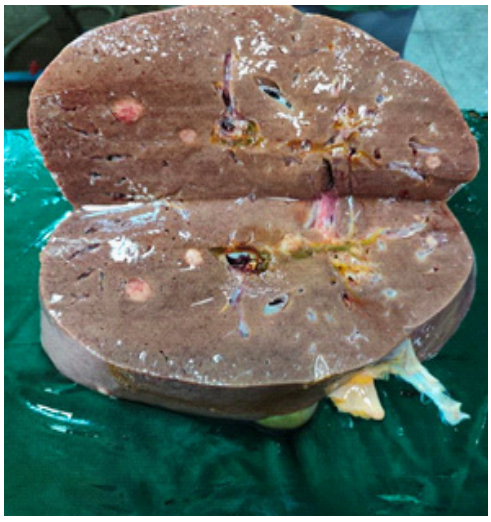

Figure 12: Autopsy of cut section of liver showing multiple metastatic nodules with intervening normal liver parenchyma.

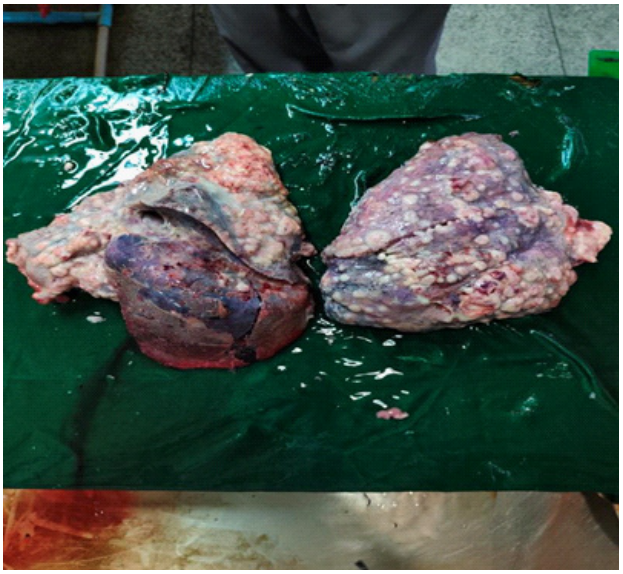

Figure 13: Autopsy of right lung showing multiple metastatic nodules over external surface.

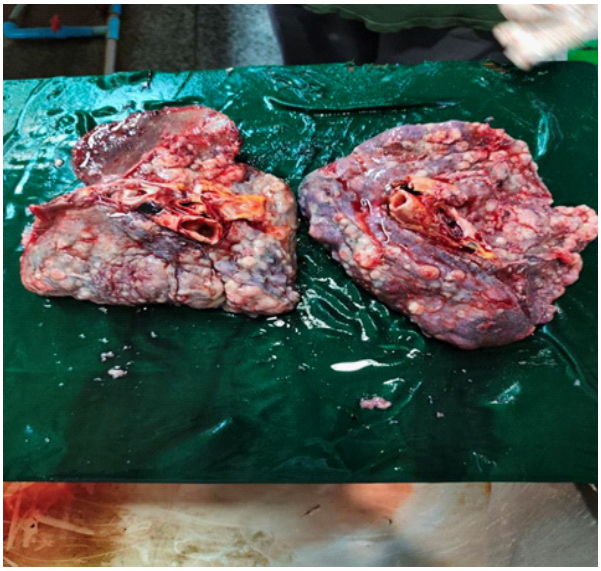

Figure 14: Autopsy of left lung showing multiple metastatic nodules over external surface.

Figure 15: Nodules from metastatic areas were taken at post-mortem examination to proceed histology.

Figure 16: Nodules from metastatic areas of lung and liver were taken at post-mortem examination to proceed histology.

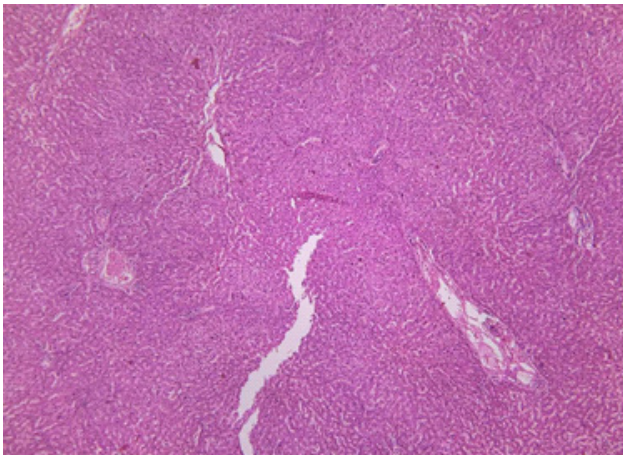

Figure 17: Liver histology taken from normal area showing liver lobule with central vein and surrounding portal tracts; and brown pigment is lipofuscin, a normal finding.

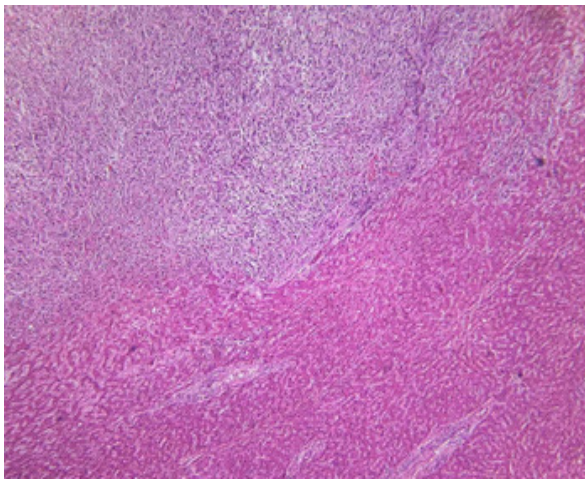

Figure 18: Liver histology showing metastatic infiltration area (upper part) and normal area (lower part); infiltration of diffuse sheets of neoplastic cells.

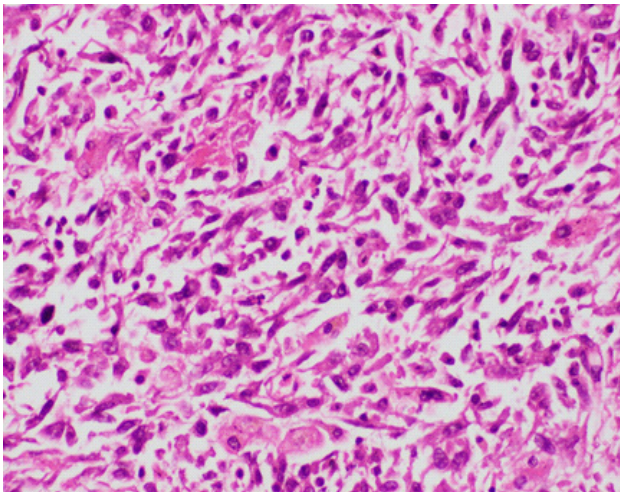

Figure 19: Histology from metastatic area liver (40x magnification) revealing infiltration of diffuse sheets of pleomorphic cells with vesicular to hyperchromatic nuclei and eosinophilic cytoplasm; and mitoses are also noted.

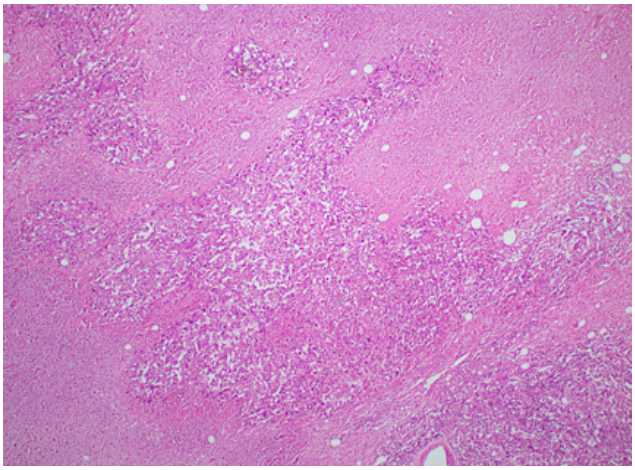

Figure 20: Histology from metastatic Lymph node (x4) demonstrating infiltration of diffuse sheets of neoplastic cells arranged in nests and clusters.

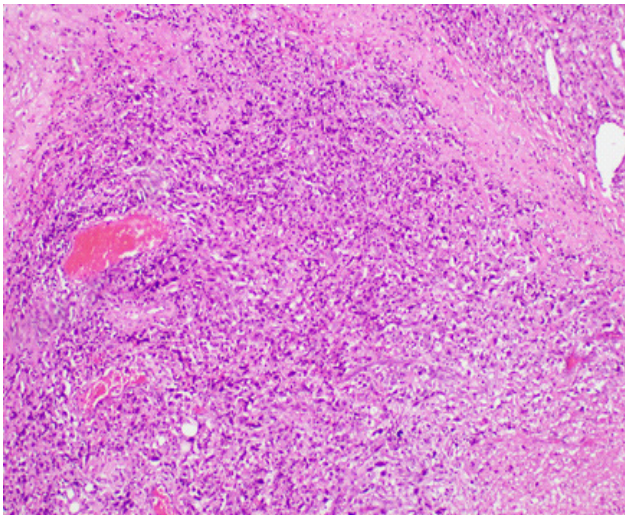

Figure 21: Histology of metastatic Lymph node (x10) showing metastatic area; infiltration of neoplastic cells with pleomorphic vesicular to hyperchromatic nuclei and eosinophilic cytoplasm.

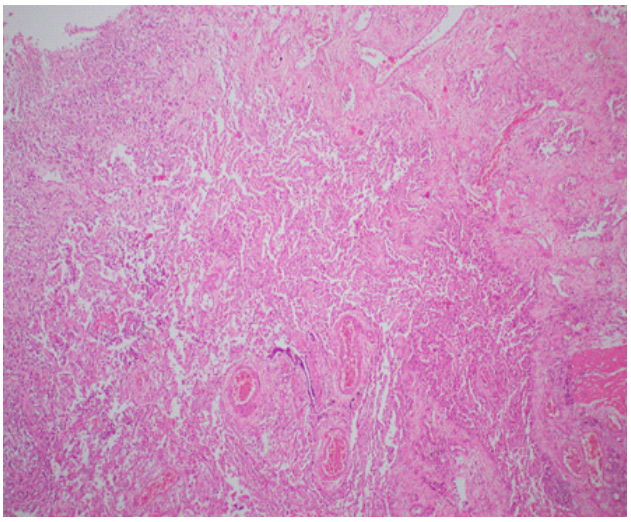

Figure 22: Histology from Normal Lung Tissue (x10) revealing broncho-vascular bundle which includes pulmonary arteries and bronchus; and bronchial glands.

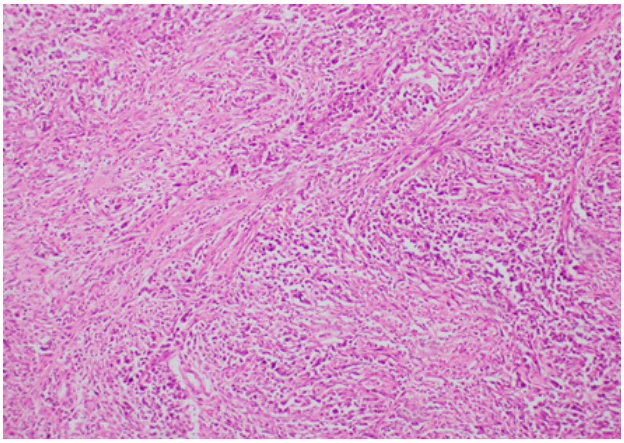

Figure 23: Histology of Lung Tissue (x10) magnification demonstrating infiltration of diffuse sheets and nests of pleomorphic neoplastic cells with vesicular to hyperchromatic nuclei and eosinophilic cytoplasm which invades the subepithelial stroma.

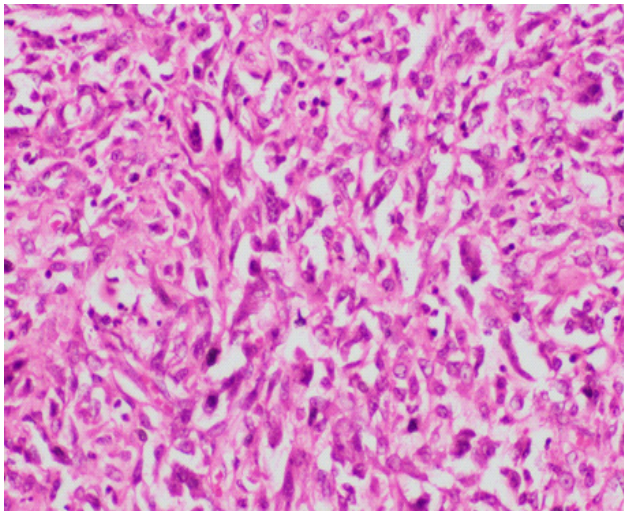

Figure 24: Histology of Lung tissue (x40) illustrating pleomorphic neoplastic cells with vesicular to hyperchromatic nuclei and eosinophilic cytoplasm with infrequent mitoses.

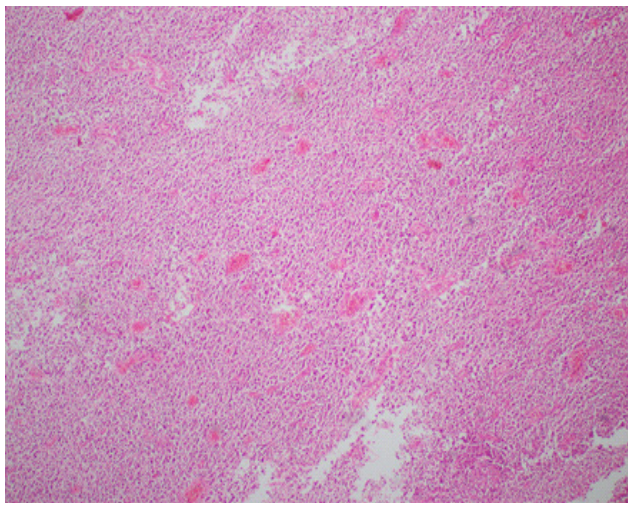

Figure 25: Histology of Cardiac Tissue (x10) from normal area where cell layer is continuous with endothelial lining of the systemic vasculature.

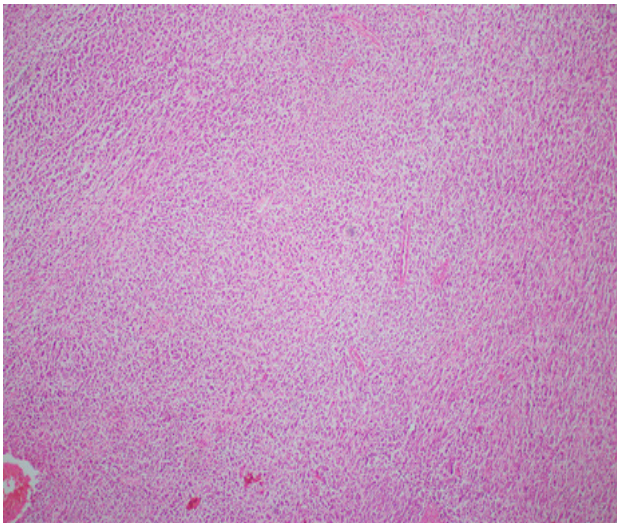

Figure 26: Histology of cardiac tissue (x10) showing infiltration diffuse sheets of pleomorphic neoplastic cells with vesicular to hyperchromatic nuclei and eosinophilic cytoplasm.

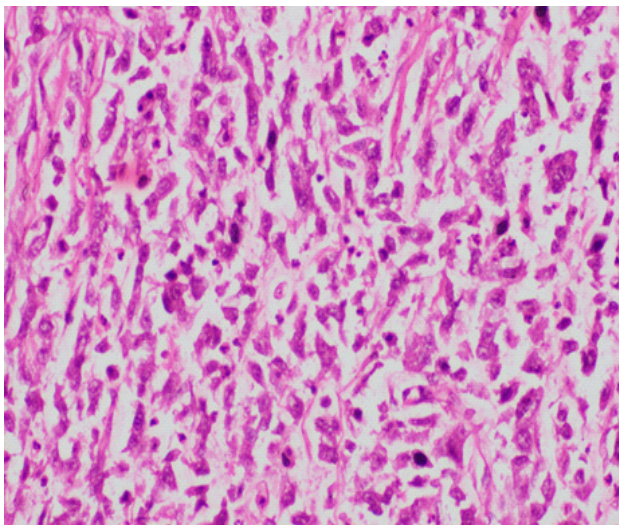

Figure 27: Histology of cardiac tissue (x40) magnification showing pleomorphic neoplastic cells with vesicular to hyperchromatic nuclei and eosinophilic cytoplasm with infrequent mitoses.

rarely reported in patient with cardiac metastasis. This patient had hypotension, hypoxia, tachycardia; they were refractory to inotropes. Likely causes of cardiogenic shock in this patient were massive pulmonary embolism, acute myocardial infarction and cardiac tamponade. Massive pulmonary embolism was unlikely as he was already on heparin therapy though D-dimer was raised. And, autopsy did reveal patent pulmonary arteries. Acute myocardial infarction was not possible because ECG did not reveal ST changes; the coronary arteries were patent in autopsy; cardiac enzymes were marginally raised. And cardiac tamponade causing shock was excluded as echocardiogram did not show pericardial effusion; autopsy finding was compatible with normal pericardial cavity with thin layer of serous fluid. Therefore, the mechanism for causing hypotension by right ventricular metastatic mass in this patient was interesting. Metastatic mass in right ventricular caused right ventricular outflow obstruction during ventricular systole in this patient. And it led to reduced pulmonary artery blood flow. It again caused hypoxia and low volume in pulmonary veins; therefore, reduction in both left atrium filling and left ventricular volume. Finally, the end result was reduction in stroke volume and blood pressure. In this patient, hypotension was refractory to combined inotropes. Moreover, hypoxia due to reduction in pulmonary artery blood flow was aggravated by bilateral pleural effusion. Hypotension or cardiogenic shock again enhanced tissue hypoxia. And, vicious cycle resulted in torrential fall in blood pressure and hypoxia in this patient. Metastatic osteosarcoma with direct cardiac involvement was an exceptionally rare finding [6]. Osteosarcoma or osteogenic sarcoma is a primary malignant tumour of the skeleton characterised by the direct formation of immature bone or osteoid tissue by the tumour cells. It is highly malignant tumour. In this patient, the size of cardiac metastasis in autopsy was quadruple the size in echocardiogram; it showed rapid tumor growth over one week. And having multiple metastasis at various organs (both lungs, thoracic cavity, liver) over one year period even with chemotherapy pointed out the high invasiveness of osteosarcoma. Osteosarcoma had high propensity to metastasize; and it was the leading cause for treatment failure and poor prognosis [5]. This patient had local extensive curettage and chemotherapy. However, he had both local recurrence and distant multiple metastasis to several sites: lung, parietal pleura, myocardium and liver. It proved the high malignant nature of osteosarcoma. Osteosarcoma was the most common primary bone malignancy in adolescents [7]. The majority of cases (75%) occured before the age of 25 and a smaller group emerged in older adults [8,9]. The patient was 41 years at the time of diagnosis; therefore, it was included in smaller group. It is one reason for case reporting. Osteosarcoma primarily affects the extremities, particularly at the growth plates of long bones. The site of lesion in this patient was proximal tibia; therefore, it followed the general rule. It was reported that the majority of distant metastases was in the lung [10]; and, this patient had metastasis in both lungs and pleura. This patient had multiple metastasis to lung, parietal pleura (inner thoracic cavity), myocardium and liver. As shown in autopsy's photo, the mass over myocardium of right ventricle was large, measuring 12.5 x 17.5 cm. It was 4 times the size mentioned in echocardiogram done 7 days prior to death. Cardiac metastasis in patients with osteosarcoma was rare; the maximum size mentioned in previous report was less than 5 cm. Therefore, this patient had the largest size of cardiac metastasis among reported size. This is anther reason for sharing case report. The most common location was found to be right ventricle [11]; and this patient had metastasis the right ventricle too. Regarding the location of cardiac metastasis, one report mentioned left atrial mass initially thought as left atrial myxoma was found to be osteogenic sarcoma (Cardiac Osteosarcoma: A Case Report and Literature Review, n.d.) [12]. Another report in 2024 described cardiac metastasis in left atrium which float into left ventricle [13]. Right ventricular mass encroaching into interventricular septum was mentioned by Lee et al and Osati et al [14,15]. Because of aggressive nature of osteosarcoma, the survival was 1-5 years. The duration of survival in this patient was only one year. Iwata reported that distant metastasis was the leading cause of disease-related death [16]. The prognosis of patients with multi-systemic metastases from osteosarcoma was very poor; and standard chemotherapy was reported as less effective [17-19]. Moreover, Zhang et al suggested that newer treatment (Ezrin) was necessary for osteosarcoma metastasis [20]. This patient would have survived with Ezrin therapy. The value of non-enhanced thoracic computed tomography (NECT) in oncology was to detect cardiovascular involvement and lung metastases early; it could detect calcific cardiac metastases; and it could differentiate tumor thrombus from bland thrombus [21,22]. In this patient, the irregular opacity over lateral border of right ventricle and right lung margin in HRCT chest was suggestive cardiac metastasis; it was analyzed after seeing mass in right ventricle. Daneman et pointed out that calcification within the mass was suggestive of cardiac metastases in HRCT chest was recorded in two children with cardiac metastases from osteogenic sarcoma [23]. HRCT chest of this patient did not reveal calcification. Cardiac MRI was good in detecting very small, clinically asymptomatic cardiac metastases by osteosarcoma [24]. We could not do cardiac MRI in this case. Early diagnosis of cardiac metastasis was found to be associated with a better outcome [25], awareness of cardiac metastasis was crucial. All patients with malignancy should be screened for cardiac metastasis with thoracic imaging and echocardiogram.

Conclusion

Cardiac metastases are very rare yet pose significant medical concerns. It is usually silent and rarely attracts clinical attention. Cardiac metastases from osteosarcoma are exceptional. Osteogenic sarcoma is an uncommon tumor. Metastasis to myocardium is very rare. Detection of cardiac metastasis is crucial for management and prognosis.

Declarations

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