Case Report

Pancreatic metastasis of a primary osteosarcoma with genomic profiling analysis: case report and review of the literature

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Background: Osteosarcoma is a common primary bone malignancy in children but is relatively rare in adults. Metastasis at diagnosis is common with the lungs being the most commonly involved organ. Pancreatic metastasis is rare and often occurs later with disease progression. Risk factors for pancreatic spread remain unknown. Metastasis within the pancreas can be symptomatic causing pancreatitis, jaundice or gastric outlet obstruction. Lesions can be solid, cystic or mixed solid and cystic. Management options of pancreatic involvement include a combination of surgery and chemoradiotherapy.

Case Description: We hereby present a case of an 18-year-old patient with metastatic osteosarcoma with a mixed solid/cystic pancreatic mass causing gastric outlet obstruction who underwent a palliative surgical resection. Next generation sequencing was performed on the diagnostic sample of the tumor. In addition, we summarize the existing literature on pancreatic metastasis in osteosarcoma.

Conclusions: Pancreatic metastasis of osteosarcoma is an uncommon presentation and typically occurs in late-stage disease. Few published cases exist and to our knowledge no documented genetic analysis of pancreatic metastasis from osteosarcoma is present in the literature. Next generation sequencing done in this case may provide insight on genetic predisposition of pancreatic metastasis. Surveillance and management strategies for pancreatic involvement remains unclear and further study is warranted.

Keywords: Osteosarcoma; pancreatic metastasis; genomic profiling; case report

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Introduction

Osteosarcoma is the most common primary bone malignancy. It demonstrates bimodal age distribution with peak incidence in early adolescence and in adults over the age of 65 (1). Considered uncommon in adults, it represents the 8th most common cause of childhood and adolescent cancer, accounting for 2.4% of all malignancies in children (2). In younger ages, osteosarcoma coincides with pubertal growth spurts as evidenced by increased incidence in adolescents ages (2). The second peak over the age of 65 is typically secondary to other bony pathology such as Paget's or previous irradiation (3). Males are more commonly affected than females. Black and Hispanic children are more commonly afflicted than Caucasians in childhood whereas the condition is more common in non-Hispanic white patients in adult-onset disease. Primary tumors are most commonly seen near the metaphyseal growth plates of long bones in the extremities such as the femur, tibia and the humerus (2). Most osteosarcoma are

sporadic although genetic predisposition, particularly in children, is common with germ-line mutations commonly identified in *TP53*, *RB1* and *RECQL4* (4,5). Unfortunately, 25% of patients have metastatic disease at time of diagnosis (4). In fact, 80% of patients are considered to have pulmonary micrometastasis (6). The lungs represent the most common site of metastatic disease (98%) with other sites including the skeletal system (37%), pleura (33%), cardiac system (20%), renal and hepatic (17%), and mediastinum (11%) (7). Metastasis of osteosarcoma to the pancreas is very uncommon. Due to better treatment modalities with increased survival rates, metastasis to more unusual sites are being increasingly identified (8). In this case report, we will discuss a case of a pancreatic metastasis from primary osteosarcoma with molecular analysis using next generation sequencing and review the existing literature of this rare presentation. We present the following case in accordance with the CARE reporting checklist (available at https://apc.amegroups.com/article/view/10.21037/apc-22-5/rc).

Case presentation

An 18-year-old previously healthy male presented with complaints of progressively worsening left knee pain. Magnetic resonance imaging (MRI) and biopsy confirmed a high grade, poorly differentiated, osteosarcoma of the distal left femoral diaphysis, metaphysis, and epiphysis (lateral condyle) with extension into the adjacent posterior and lateral thigh soft tissues measuring 7.1 cm × 6.6 cm × 10.4 cm. Staging demonstrated multiple scattered nodules throughout the lungs bilaterally measuring up to 5 mm suggesting micrometastasis. The patient was started on chemotherapy per the AOST0331 protocol, which included cisplatin, doxorubicin, and methotrexate. After 4 rounds of chemotherapy, the patient underwent limb salvage surgery with total knee replacement. Pathology demonstrated high grade osteoblastic osteosarcoma with moderate tumor necrosis (60–70%). One year later, the patient developed pulmonary macrometastatic disease and underwent thoracotomy and metasectomy and was started on adjuvant Regorafenib. He subsequently underwent radiation therapy of 2 contiguous lesions of the left upper lobe.

Surveillance imaging on December 2020 demonstrated no signs of pancreatic involvement (*Figure 1A*), however, subsequent surveillance imaging done on May 2021 (two years after diagnosis) demonstrated an increase in size of multiple pulmonary metastases in addition to a new finding of a partially calcified mixed solid/cystic pancreatic lesion within the pancreatic tail and adjacent regional stranding, concerning for metastatic disease or focal pancreatitis (*Figure 1B*). Patient denied abdominal pain. Lipase was elevated at 782 U/L [>3 upper limit of normal (ULN)]. Liver enzymes were normal. Due to concern for possible drug induced pancreatitis, Regorafenib was held. Follow-up magnetic resonance cholangiopancreatography (MRCP) in June 2021 demonstrated an enlarging mixed cystic and solid mass lesion, appearing to originate from the superior aspect of the pancreatic body/tail measuring up to 6.8 cm. The patient underwent endoscopic ultrasound (EUS) with fine needle aspiration (FNA). EUS confirmed a large (6.4 cm × 6.3 cm) mixed solid cystic exophytic mass arising from the pancreatic tail. The mass itself was seen to have thin septations and was multicystic with no clear communication with the main pancreatic duct. The pancreas was otherwise normal. FNA was completed with 8 cc of blood-tinged amber fluid. Fluid was sent for cytology, chemistry and next generation sequencing (PancreaSeq®, University of Pittsburgh Molecular Lab). Carcinoembryonic antigen (CEA) and amylase were within normal limits. Cytology demonstrated cystic contents consisting of a few strips and clusters of bland ductal type epithelial cells and scattered degenerating cells (likely histiocytes and epithelial cells). No malignancy, including osteosarcoma was noted. PancreaSeq identified multiple genomic alterations

Highlight box

Key findings

- Our case report highlights a rare presentation of a patient with pancreatic metastasis of osteosarcoma.

What is known and what is new?

- Osteosarcoma metastasis to the pancreas is unusual. We summarize the existing literature of this uncommon presentation.
- We performed next generation sequencing on the pancreatic metastasis which identified multiple copy number alterations in TP53, PTEN, RNF43, and SMAD4.

What is the implication, and what should change now?

- Improvement in therapies for osteosarcoma is leading to more unusual sites of metastasis which should affect surveillance strategies and intervals.
- Further study is needed on optimal therapies for management of pancreatic metastasis of osteosarcoma.
- Next generation sequencing could provide insight into genetic predisposition for pancreatic metastasis of osteosarcoma. This could eventually impact surveillance and management strategies.

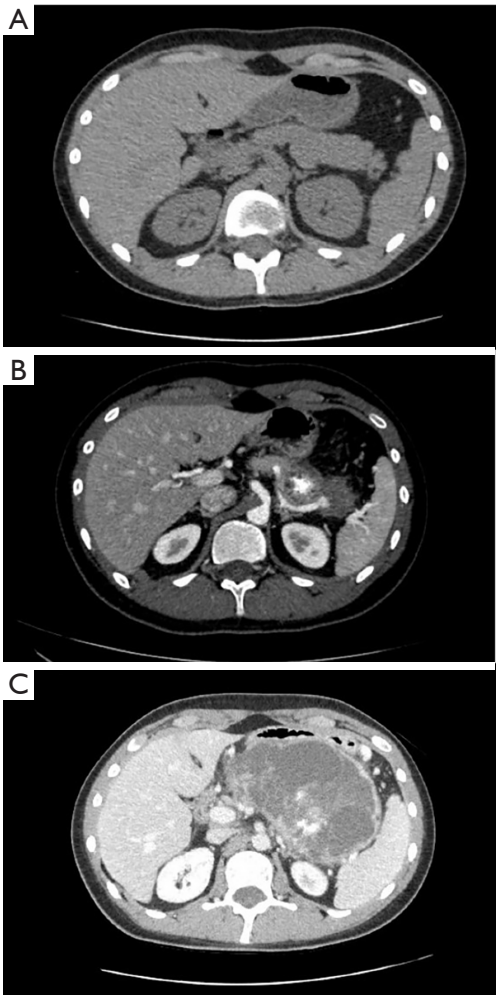

Figure 1 Serial surveillance CT showing interval development of pancreatic metastasis (A) Surveillance CT done in December 2020. At this time, the pancreas was seen without any signs of metastasis. (B) Surveillance CT done in May 2021. First signs of metastasis to the pancreas were seen at this time. (C) CT abdomen and pelvis done in July 2021, which showed interval enlargement of pancreatic metastasis. CT, computed tomography.

including *PTEN*, *TP53*, *RNF43*, and *SMAD4* copy number alterations, however no gene mutations (such as point mutations, insertions, or deletions) were noted. Patient was discharged with no complications. A subsequent computed tomography (CT) abdomen with contrast was completed in July 2021 to re-assess the pancreatic lesion demonstrated interval enlargement of both pancreatic mixed cystic/solid mass (*Figure 1C*) and pulmonary nodules, as well as the development of a new hypoenhancing lesion in the left inferior renal pole, concerning for metastasis. Given interval

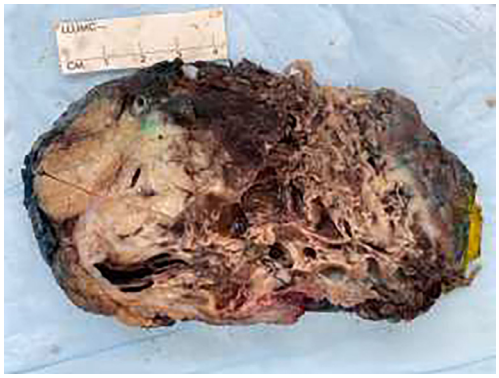

Figure 2 The 15 cm resected exophytic pancreatic mass representing osteosarcoma metastasis.

enlargement of cystic/solid mass concerning for metastasis, leading to an almost complete inability of the patient to tolerate oral intake due to mass effect on the stomach, the patient was referred to surgical oncology for palliative measures and underwent *en-bloc* resection of the mass, distal subtotal gastrectomy, *en-bloc* spleen-preserving distal pancreatectomy, *en-bloc* resection of transverse mesocolon and Roux-en-Y gastrojejunostomy reconstruction. The 15 cm mass (*Figure 2*) was found to be occupying the entire lesser sac and fixed to the mid and distal pancreas, the entire posterior wall of the stomach, the transverse mesocolon, and the splenic vasculature distally. Pathology from mass resection demonstrated metastatic high-grade osteosarcoma with positive perineural and perivascular invasion (*Figure 3*). Due to progressive disease, the patient later elected to proceed with palliative care.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

Pancreatic metastasis of osteosarcoma remains uncommon however with improving treatment modalities for osteosarcoma metastasis to uncommon sites are being increasingly recognized. Based on our literature review, there have been only 20 documented cases of pancreatic metastasis from osteosarcoma (*Table 1*).

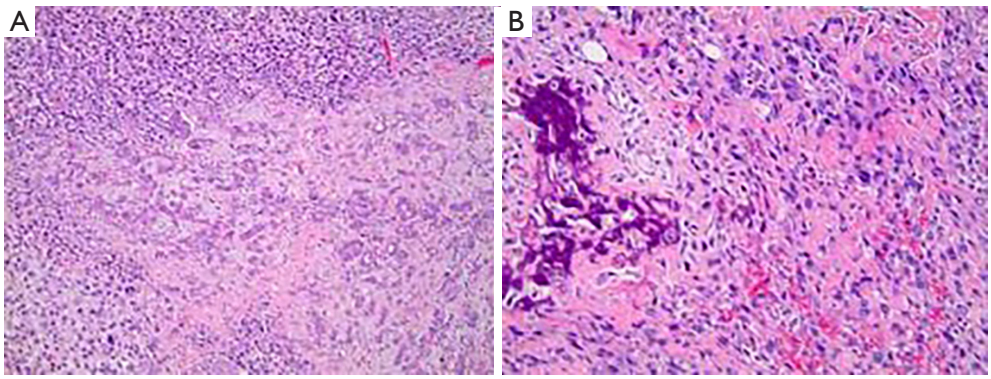

Figure 3 Pathology of pancreatic mass resection. (A) Pancreas with metastatic osteosarcoma. Image obtained at ×10 magnification. (B) Osteosarcoma with malignant osteoid. Image obtained at ×20 magnification. Both (A) and (B) are stained with H&E staining. H&E, hematoxylin and eosin.

Aside from our case, there has been no documented genetic analysis of pancreatic metastasis from osteosarcoma. It has been shown that osteosarcoma with positive *PTEN* expression has been associated with high differentiation on histology, less chance of metastasis, and higher overall 5-year survival rate (24). It is thought that *PTEN* expression can inhibit osteosarcoma cell proliferation and migration (25). USP17 upregulation, through stabilization of *SMAD4*, has been thought to promote cell proliferation, epithelial-mesenchymal transition, and metastasis in osteosarcoma (26). Activation of HIF-1α and AP-1 genetic pathways have revealed a possible increase in metastatic potential (27). In addition, APEX1 expression in osteosarcoma has also been found to be an independent predictor of local recurrence and metastasis, owing to chemoresistance and radioresistance (28). Genetic markers that increase the risk of osteosarcoma development include germline variants in *TP53*, *RB1*, and *RECQL4* genes (5). *ATRX* germline variant has also been thought to be associated with an increase in the risk of osteosarcoma development (5). In our patient, PancreaSeq identified multiple copy number alterations in *TP53*, *PTEN*, *RNF43*, and *SMAD4*. As noted above, alterations in *PTEN* and *SMAD4* have been associated with increased risk of metastasis.

In our case, pancreatic metastasis was identified 2 years after diagnosis which is consistent with the existing case reports. In addition, despite an elevated lipase, our patient was asymptomatic which is consistent with the published cases (7). It is worthy to note that most documented pancreatic involvement in osteosarcoma represents solitary solid lesions (9). In our particular case, the patient's pancreatic metastasis was found to be a single metastatic cystic/solid lesion. There are very few cases in the literature of pancreatic cystic or cystic/solid metastasis from osteosarcoma. The pathophysiology of cystic metastasis is unknown. Current theories include necrotic degeneration of a solid mass or pancreatitis from the metastasis leading to fluid collections (9). Necrotic degeneration of solid masses is seen more commonly in neuroendocrine tumors, ductal adenocarcinomas, and acinar cell carcinoma (9). Pseudocyst formation secondary to metastasis-induced acute pancreatitis could be possible in the case of our patient given his elevation in lipase. Though it is important to note that regorafenib can increase serum lipase and even cause pancreatitis (29). Diagnosis of pancreatic metastasis can be confirmed with EUS-FNA although if inconclusive and the index of suspicion remains high, surgery may be warranted. Management of pancreatic metastasis of osteosarcoma involves a combination of surgery, radiation and chemotherapy. Surgical resection is considered an important modality of local control of metastatic sites although the benefit in osteosarcoma metastasis to the pancreas appears unproven (4). Ideal candidates for surgical resection should have isolated resectable pancreatic metastasis after complete staging or limited other sites also amenable to resection. Typical pancreatic resection approaches such as pancreaticoduodenectomy or distal pancreatectomy are favored over enucleation or central pancreatectomies owing to higher local recurrence rates despite less morbidity (12). Osteosarcoma is not a radiosensitive malignancy and thus radiation is typically offered as adjuvant therapy for consolidation treatment or as definitive management in unresectable disease.

Table 1 Summary of case reports of osteosarcoma with pancreatic metastasis

Age/ gender	Location of original OS	Time between diagnosis of OS and pancreatic metastasis	Location of pancreatic Mets	Size of pancreatic Mets	Description of pancreatic Mets	EUS-FNA	Tx of metastasis
35 F (7)	Distal right femur	5 years	Body	0.3×0.3×0.3 cm	Solid tan/white calcified	No	Resection with distal pancreatectomy with splenectomy
25 F (8)	Right femur	5 years	Head	10×4 cm and 5×3× 3 cm (two masses in continuity—dumbbell shape)	–	No; resection showed malignant osteoid tissue	Pancreato-duodenectomy
53 F (9)	Left distal femur	4 years	Genu, body, and tail	11×10 cm (after drainage several days prior)	Cystic/solid	Yes; FNA resulted in malignant cells with numerous multi-nucleated giant cells. Tumor was highly cellular with 17 mitoses per 10 HPF	Needle aspiration of cystic portion for symptom control
14 M (10)	Vertebral: T7-S1	6 years	Head	8×7.5 cm	Solid with calcifications and necrotic areas	No; Tru-cut biopsy	Deceased prior to treatment
15 F (11)	Distal left femur	4 years	Tail	15×15×12 cm, however only small solid portion invaded the pancreas	Cystic lesion with solid component invading the pancreas	Yes; showed pancreatic cells and plemorphic tumoral cells surrounded by osteoid matrix	Partial resection of pancreatic tail. Family refused adjuvant CTx
13 F (12)	Left fibula	14 months	Head	6×4×3 cm	Solid	No; resection showed rapidly proliferating osteoblasts with giant and atypical nuclei	Pancreato-duodenectomy
44 F (13)	Right fibular head	3 years	Tail	13 mm	Heterogenous solid	Yes; pancreatic islet tissue was also detected in tumor suggesting invasion of tumor into pancreatic body	Laparoscopic splenic-preserving pancreatic tail resection with post-operative CTx
33 M (14)	Right maxillary sinus	3 years	Body-tail junction	25 mm	Solid with thin central calcifications	Yes; showed poorly differentiated area consisting of small sized round fusiform cells and focal area of osteoid deposited in a fine lace-like pattern, as well as relatively well differentiated area with cartilage	Patient was undergoing CTx at the time the case report was written
18 M (15)	Proximal left tibia	4 years	Head	4.3×4.5 cm	Cystic-solid mass	Yes; amorphous pink-colored osteoid structures with pleomorphic multinucleated sarcomatoid cells	Pancreato-duodenectomy followed by CTx

Table 1 (*continued*)

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Age/ gender	Location of original OS	Time between diagnosis of OS and pancreatic metastasis	Location of pancreatic Mets	Size of pancreatic Mets	Description of pancreatic Mets	EUS-FNA	Tx of metastasis
66 F (16)	Femur	3 years	–	–	–	No	–
9 F (16)	Femur	1.5 years	–	–	–	No	CTx
14 F (16)	Femur	11 years	–	–	–	No	Pancreato-duodenectomy
57 M (17)	Femur	3 years	–	–	–	–	–
52 M (18)	Maxillary sinus	2 years	–	–	–	Yes	–
58 F (19)	Tibia	7.3 years	Head	3.5 cm	–	–	Radiation and CTx
57 F (19)	Femur	1.25 years prior to diagnosis of osteosarcoma	Tail	2.7 cm	Solid	No	Distal pancreatectomy followed by CTx
32 F (19)	Skull	5.4 years	Tail	19 cm	–	No	Distal pancreatectomy
28 F (19)	Femur	1.7 years	Body	4 cm	–	No	Distal pancreatectomy
63 F (20)	Left distal femur	–	Head	3.6×2.6 cm	Cystic	Yes; FNA showed metastatic malignant tumor with highly pleomorphic atypical tumor cells	None
38 F (21)	–	3.6 years	Head	5.7 cm	Cystic mass with calcifications	–	–
19 F (22)	Distal femur	5.5 years	–	5 cm	–	No	Pancreato-duodenectomy followed by adjuvant CTx
47 F (23)	Distal femur	3.3 years	Head	7 cm	Solid	–	Radiation therapy
42 F (23)	Left distal femur	–	Tail	–	Solid	–	Radiation therapy

OS, osteosarcoma; Mets, metastasis; EUS, endoscopic ultrasound; FNA, fine needle aspiration; Tx, treatment; F, female; M, male; HPF, high-power field; CTx, chemotherapy.

In summary, as treatment advances for osteosarcoma, more unusual sites of metastasis will occur. Genetic profiling of osteosarcomas may help identify if there are any particular genetic alterations that would give a predilection to non-pulmonary metastasis, as in the case of our patient. This could allow for patient centered surveillance strategies for a select group of individuals and allow for timely diagnosis and treatment.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://apc.amegroups.com/article/view/10.21037/apc-22-5/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://apc.amegroups.com/article/view/10.21037/apc-22-5/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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