Imaging of Merkel Cell Carcinoma

What imaging experts should know

TO BE FAMILIER WITH RATIONALE BEHIND THE ORDER AND CREATE ACTIONABLE AND CLINICALLY RELEVANT REPORTS

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The purpose of this exhibit is to understand/review...

1. Pathophysiology and clinical behavior of Merkel cell carcinoma (MCC).
2. MCC’s aggressive features and the important role of imaging in the management.
3. The Merkel cell polyomavirus (MCPyV) as a cause of MCC.
4. Utility of a blood test that detects antibodies to the MCPyV.
5. Different clinical approaches in antibody producers and non-antibody producers.
6. Neuroendocrine features of MCC with somatostatin receptor (SSTR) expression which can be imaged by SSTR seeking nuclear medicine studies.
7. Various imaging manifestations of MCC on different modalities.
Where does MCC come from?

Merkel cells are found in the base of epidermis to dermis of the skin. They function as “touch receptors”.

Normal Merkel cells (shown in red), are connected to nerves (shown in yellow), signaling touch sensation.

Merkel cells are not the origin of MCC. MCC is named after ultrastructural and immunophenotypic resemblance to sensory Merkel cells in the skin \(^1\).

MCCs are most frequently found in the dermis but can arise from any layer of the skin from intraepidermal to subcutaneous \(^2\). Fundamental evidence on the MCC cell of origin is yet to come into view.

MCC is unique and tricky cancer

**Tricky clinical features**

MCC typically develops **rapidly** and manifests as firm, nontender, dome-shaped red, purple or violet nodule.

It has a predilection for the sun-exposed area, such as head, neck and extremities, however, it can occur anywhere in the body.

"It can fool even the best clinicians"
A majority of MCC lesions (56%) were **presumed to be benign** at biopsy.

**MCC is more lethal than melanoma**

Mortality rate

- ~40% for MCC
- ~8% for melanoma

**5-year survival rate from the 8th edition AJCC staging system**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Survival Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Localized disease</td>
<td>51%</td>
</tr>
<tr>
<td>Nodal involvement</td>
<td>35%</td>
</tr>
<tr>
<td>Distant metastasis</td>
<td>14%</td>
</tr>
</tbody>
</table>

**Increasing incidence**

2500 cases/year in US in 2013. The incidence is expected to increase to 2835 in 2020 and to 3284 in 2025.

From 2000 to 2013, solid cancers increased by 15%, melanoma by 57%, and Merkel cell carcinoma by **95%**.

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Becker JC et al., Ann Oncol, 2010


Risk factors

Risk factors:
- Age (> 65 years)
- Fair skin
- Sun exposure
- Chronic immune suppression
  - **Merkel cell polyomavirus** in ~80% cases.

Risk of developing MCC increases in patients with immunosuppression.
- HIV patient: 8 times greater  
  *Engels et. al. Lancet 2002;359:497-498*
- Organ transplant patient: 25 times greater  
- Chronic Lymphocytic leukemia: 40 times greater  

*However, 90% of MCC patients are not immune suppressed.*
The Merkel cell polyomavirus (MCPyV) is causally linked to 80% of cases whereas 20% are caused by extensive UV mutations. MCPyV is the only known human oncovirus in the polyomavirus family. Antibodies against MCPyV oncoprotein antigens are associated with tumor burden and serve as a “tumor marker”. The test is much cheaper (~$300/test) than imaging studies (3,000-$100,000/scan).

Different approach based on MCPyV oncoprotein antibody status.

Antibody producers

Routine radiologic scans can be reduced as the antibody serves as “tumor marker”.

Non-Antibody producers

Must be followed by frequent imaging studies.
65-year-old woman with stage I MCC of the left cheek s/p wide local excision and negative SNLB on 02/2016.

(a) Post-operative oncoprotein titer was negative and contrast CT showed no evidence of disease. Oncoprotein antibody titer had been continuously increased which prompted imaging evaluation.

(b) Contrast Neck CT shows a small enhancing nodule in the left parotid gland.

(c) F$^{18}$ FDG PET/CT shows increased FDG uptake in the corresponding nodule. US guided biopsy was performed and recurrent MCC was pathologically confirmed.
# AJCC (American Joint Committee of Cancer) Staging 8th edition

<table>
<thead>
<tr>
<th>Stage</th>
<th>Primary Tumor</th>
<th>Lymph Node</th>
<th>Metastasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>In situ (within epidermis only)</td>
<td>No regional lymph node metastasis</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>I</td>
<td>Clinical* ≤ 2 cm maximum tumor dimension</td>
<td>Nodes negative by clinical exam (no pathological exam performed)</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>I</td>
<td>Pathological** ≤ 2 cm maximum tumor dimension</td>
<td>Nodes negative by pathologic exam</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>IIA</td>
<td>Clinical &gt; 2 cm tumor dimension</td>
<td>Nodes negative by clinical exam (no pathological exam performed)</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>IIA</td>
<td>Pathological &gt; 2 cm tumor dimension</td>
<td>Nodes negative by pathologic exam</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>IIB</td>
<td>Clinical Primary tumor invades bone, muscle, fascia, or cartilage</td>
<td>Nodes negative by clinical exam (no pathological exam performed)</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>IIB</td>
<td>Pathological Primary tumor invades bone, muscle, fascia, or cartilage</td>
<td>Nodes negative by pathologic exam</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>III</td>
<td>Clinical Any size / depth tumor</td>
<td>Nodes positive by clinical exam (no pathological exam performed)</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>IIIA</td>
<td>Pathological Any size / depth tumor Not detected (&quot;unknown primary&quot;)</td>
<td>Nodes positive by clinical exam, and confirmed via pathological exam</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>IIIB</td>
<td>Pathological Any size / depth tumor</td>
<td>Nodes positive by clinical exam, and confirmed via pathological exam OR in-transit metastasis***</td>
<td>No distant metastasis</td>
</tr>
<tr>
<td>IV</td>
<td>Clinical Any</td>
<td>+/- regional nodal involvement</td>
<td>Distant metastasis detected via clinical exam</td>
</tr>
<tr>
<td>IV</td>
<td>Pathological Any</td>
<td>+/- regional nodal involvement</td>
<td>Distant metastasis confirmed via pathological exam</td>
</tr>
</tbody>
</table>

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In transit metastasis
A tumor distinct from the primary lesion and located either
1) between the primary lesion and the draining regional lymph nodes
or
2) distal to the primary lesion

**NCCN guidelines**

1. **Introduction**
2. Origin of MCC
3. Clinical manifestation
4. Risk factors
5. McPyV
6. Staging
7. **NCCN guideline**
8. Immunotherapy
9. Role of imaging
10. SNLB
11. CT pattern
12. MRI pattern
13. FDG PET/CT
14. SSTR imaging
15. CNS metastasis
16. Bone metastasis
17. Metastasis to uncommon organs
18. PRRT
19. Take home messages

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**Clinical N0**
- Wide resection + Adjuvant radiation therapy to the primary tumor or observation
- **Sentinel lymph node biopsy (SNLB)**
  - SNLB (+) baseline imaging if not performed. Clinical trial preferred.
  - SNLB (-) Observation or radiation therapy to the nodal basin in high-risk patients

**Clinical N+**
- **Imaging studies**
  - FNA or core biopsy
- **M0** Multidisciplinary tumor board. Node dissection and/or radiation therapy
- **M1** Follow clinical M1 pathway
- **Consider open biopsy**
- Follow clinical N0 pathway

**Clinical M1**
- Multidisciplinary tumor board
- *Clinical trial if available
- *Consider
  - Systemic therapy
  - Radiation therapy
  - Surgery
  - Best supportive care

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*Bichakjian et.al. J Natl Compr Canc Netw 2018;16(6):742–774*
**Immunotherapy has changed NCCN guidelines**

<table>
<thead>
<tr>
<th>Year</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016</td>
<td>Chemotherapy only</td>
</tr>
<tr>
<td>2017</td>
<td>Immunotherapy (Pembrolizumab) was listed as one of the systemic therapy options</td>
</tr>
<tr>
<td>2018</td>
<td>Immunotherapies (avelumab, pembrolizumab, nivolumab) are <strong>preferred as 1st line therapy</strong></td>
</tr>
</tbody>
</table>

Checkpoint inhibitors disable the **PD-L1 (programmed cell death-ligand 1)** protein on cancer cells, activating the immune system to attack the tumor cells.

Cytotoxic chemotherapy is associated with a high initial objective response rate (ORR), however responses are **seldom durable** with the median progression-free survival (PFS) of about 94 days, and toxicity is considerable.  


Food and drug administration (FDA)-recently approved immunotherapy with PD-1/PD-L1 antibody have demonstrated promising long-term benefit.  

Role of Imaging

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Local staging, surgical planning and radiation planning

Sentinel lymph node biopsy

Systemic staging

Evaluation of treatment response and follow-up
**Sentinel Lymph node biopsy (SNLB)**

Procedure:
Tc99m Sulfur Colloid
1 mCi (0.2 micron Filtered) was injected intra-dermally at four locations around the primary tumor/tumor biopsy site.
Multiple static images of the expected lymph nodal basin to localize SNL.
Hand probe is used to confirm the node.

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**Occult nodal metastasis is common** in MCC and SNLB should be recommended for all patients with primary MCC.

Patients with a positive SLN have a higher risk of in-transit recurrence and may benefit from adjuvant radiation with inclusion of the in-transit field in amenable cases.

*J.R. Sims et al. Sur Oncol 2018 27; 11-17*

In patients with stage I and II MCC, SNLB is more sensitive than FDG PET/CT


SLNB should be performed before wide local excision or Mohs micrographic surgery, because surgical excision before SLNB may alter the lymphatic drainage patterns.
For anatomically complex areas, or when planar image are difficult to interpret, SPECT/CT can be performed to localize SNL. In addition, higher contrast resolution of SPECT allows visualization of foci undetected on planar images.

In our institution, SPECT/CT is routinely performed for MCC of the head and neck due to its anatomical complexity.

Planer images obtained after radiotracer injection around the primary lesion (red arrow) in the right cheek. There is a faint uptake below the injection site (yellow arrow). However, it is difficult to localize the focus on planar images only.

SPECT/CT demonstrates the focus in the right parotid gland. There is additional node in the right submental region which was not visualized on planar images.
Primary lesion can manifest as cutaneous or subcutaneous mass/nodule or skin thickening.

Many patients are referred for imaging after resection of the primary lesion.

In-transit and satellite cutaneous metastases can occur. MCC have tendency to “skip”

Patients with nodal or presumed metastatic MCC with no identifiable primary skin lesions can have **better prognosis**.

→ An antitumor immune response has been proposed to underlie both the primary tumor regression and improved patient outcomes in such patients.

*Vandeven et.al*  *Clin Cancer Res* 2018 15;24(4):963-971
Evaluation of local invasion

In locally advanced disease or advanced metastatic disease, imaging plays an important role determining the localization of the lesion and identifying loco-regional invasion to surrounding organs.

This is especially important in head and neck disease, due to its anatomical complexity.

Accurate evaluation is necessary for surgical and/or radiation therapy planning.
MCC has a **high propensity for nodal metastasis** with 27-31% presenting with clinical nodal disease. In addition, another 16-38% have occult nodal metastasis determined by SNLB.

*J.R. Sims et al. Surgical Oncology 2018 27; 11-17*

**Liver Metastasis**
- Non-specific hypoattenuating lesion.
- Typically hypoenhancing compared with surrounding liver parenchyma.
- Hyperechoic on Ultrasound

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MRI patterns of primary/regional MCC lesions

MRI characteristic of MCC
- Skin thickening
- Subcutaneous reticular stranding
- Subcutaneous soft tissue mass
- Perifascial muscular and intramuscular metastases.
- Adjacent large lymph node masses with retained, compressed internodal fat

MR signal
- Isointense on T1WI
- High intensity on T2WI, Fat saturated T2WI.
- Diffuse enhancement on Gadolinium administration
- Tumor necrosis.
- Large lesions can demonstrate inhomogeneous signal intensity on both T1WI and T2WI.
- Focal central increased signal on T2WI within large lesions has been described as being associated with histologically proven central necrosis and hemorrhage (Skeletal Radiol 1998; 27:396–399)

The skin, subcutaneous masses, and reticular stranding histologically were found to be caused by lymphangitis carcinomatosa and soft-tissue lymphatic metastases.

Anderson et. al. AJR 2005; 185:1441–1448
F<sup>18</sup>-FDG PET/CT is a good modality for staging and increasingly used.

PET-CT is useful for detection of nodal involvement and distant metastasis.

Staging F<sup>18</sup>-FDG-PET significantly influenced treatment decisions in approximately one-third of cases of MCC and should be considered in the routine pre-treatment work-up. Post-treatment PET was not found to be prognostic.


FDG-PET/CT performed as part of the initial management strategy tended to upstage MCC patients with more advanced disease.


66-year-old female with MCC of the right cheek. Staging F<sup>18</sup>-FDG PET/CT demonstrates hypermetabolic primary mass in the right cheek and hypermetabolic right submandibular lymph node (arrows), suggesting metastatic nodal disease.
MCC is a unique cutaneous neuroendocrine tumor (NET) and exhibits somatostatin receptor (SSTR) on the tumor cell surface.

MCC has higher affinity to SSTR type 2A and 5, like other NETs.

If tumor expresses SSTR, Somatostatin Analogue can be used for treatment in selective patients.

In\textsuperscript{111}-Pantetretotide scintigraphy (OctreoScan\textsuperscript{TM})

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Radiolabeled Indium\textsuperscript{111}-Pantetretotide

Commercially available Somatostatin receptor binding radiotracer that can be used for scintigraphic imaging.

It has high affinity to SSTR type 2 and type 5 (remember MCC has high expression of SSTR type 2 and 5), to a lesser extent with subtype 3, and not at all with subtype 1 and 4.

(i)Whole body planar image demonstrates several foci of increased radiotracer uptake, some greater than liver. Fused SPECT/CT localized these foci in the Sternum (ii), right external iliac node (iii), soft tissue mass around the right proximal femur (iv), as well as in left subpectoral soft tissue, right calvarium, right scapula, left proximal humerus or cardiophrenic node (Images not shown)
Ga$^{68}$ Somatostatin analogue PET/CT

70-year-old male with MCC of the left posterior knee s/p wide resection. Ga$^{68}$ Dotatate PET/CT demonstrates intense radiotracer uptake in the left supraclavicular, mediastinal, retroperitoneal and pelvic regions (arrows), suggesting metastatic disease with somatostatin receptor expression.

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**SSTR analogue PET** has higher sensitivity for **bone, soft tissue and brain** disease but lower sensitivity for liver and lung disease compared to CT. Combined PET/CT has a significant impact on patient management. 

*Buder et al. BMC Cancer 2014, 14:268*
<table>
<thead>
<tr>
<th>Biomechanism</th>
<th>In(^{111}) Pentetreotide (OctreoScan(^{TM}))</th>
<th>Ga(^{68}) Dotatate (NETSPOT(^{TM}))</th>
<th>F(^{18})-FDG</th>
</tr>
</thead>
<tbody>
<tr>
<td>SSTR binding (mainly SSTR type 2 and 5)</td>
<td>SSTR binding (mainly SSTR type 2)</td>
<td>Glucose metabolism</td>
<td></td>
</tr>
<tr>
<td>Physical half life</td>
<td>2.8 days</td>
<td>68 minutes</td>
<td>110 minutes</td>
</tr>
<tr>
<td>Camera</td>
<td>Gamma camera/SPECT</td>
<td>PET</td>
<td>PET</td>
</tr>
<tr>
<td>Principle mode of decay</td>
<td>Electron Capture</td>
<td>Positron decay</td>
<td>Positron decay</td>
</tr>
<tr>
<td>Production</td>
<td>Cyclotron</td>
<td>Generator</td>
<td>Cyclotron</td>
</tr>
<tr>
<td>Timing of scan after tracer injection</td>
<td>24 hours (4 hour, 48 hour, 72 hour scan can be considered)</td>
<td>45-60 minutes</td>
<td>60 minutes</td>
</tr>
<tr>
<td>Spatial resolution</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Quantification of lesion activity</td>
<td>No</td>
<td>Yes (SUV)</td>
<td>Yes (SUV)</td>
</tr>
<tr>
<td>Patient preparation (may vary depending on institution)</td>
<td>No dedicated fasting is needed</td>
<td>No dedicated fasting is needed</td>
<td>At least 6 hours of fasting is need. Strict glucose control is needed for diabetic patients</td>
</tr>
</tbody>
</table>
Which PET scan to be used for MCC? F18-FDG vs Ga\textsuperscript{68} - Somatostatin analogue

In Gastrointestinal NET, there is an inverse relationship between World Health Organization (WHO) or The European Neuroendocrine Tumor Society (ENETS) tumor grade based on Ki-67 and SSTR expression rate.

In non-MCC NETs, Ga\textsuperscript{68}-somatostatin analogue PET/CT is recommended for lower grade tumor with low Ki 67 expression (<20%). FDG PET is recommended for higher grade, more aggressive tumor.

Little is known regarding association between SSTR expression, tumor grade and Ki 67 in MCC.

Preliminary study showed that Ga\textsuperscript{68}-somatostatin analog PET/CT provides good and equally diagnostic performance as F18-FDG PET. These results do not suggest that 18F-FDG PET/CT should be replaced by 68Ga-somatostatin receptor imaging. It could, however, be considered in selected cases of SSR positive MCC, i.e., “personalized medicine.”

Taralli et al. EJNMMI Research 2018 8:64
### Imaging patterns of MCC Central nervous system metastasis

<table>
<thead>
<tr>
<th>1. Introduction</th>
<th>Solid enhancing nodule/mass with vasogenic edema</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Origin of MCC</td>
<td></td>
</tr>
<tr>
<td>3. Clinical manifestation</td>
<td></td>
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<tr>
<td>4. Risk factors</td>
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<td>5. McPyV</td>
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<td>11. CT pattern</td>
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<td>12. MRI pattern</td>
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<tr>
<td>13. FDG PET/CT</td>
<td></td>
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<tr>
<td>14. SSTR imaging</td>
<td></td>
</tr>
<tr>
<td>15. CNS metastasis</td>
<td></td>
</tr>
<tr>
<td>16. Bone metastasis</td>
<td></td>
</tr>
<tr>
<td>17. Metastasis to uncommon organs</td>
<td></td>
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<tr>
<td>18. PRRT</td>
<td></td>
</tr>
<tr>
<td>19. Take home messages</td>
<td></td>
</tr>
</tbody>
</table>

#### MRI patterns

- **Solid enhancing nodule/mass** with vasogenic edema
- **Cystic mass with enhancing solid component**
- **Enhancing mass with hemorrhage**
  - *Note linear hyperintensity along the right cerebellar mass on T1WI and with blooming on susceptibility weighted image (SWI)*
- **Leptomeningeal metastasis**
MCC metastasis to the brain

Rare. Previously reported Brain metastasis site include

<table>
<thead>
<tr>
<th>Lead author</th>
<th>Site of brain metastasis</th>
<th>Primary site</th>
<th>Modality</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jacob AT</td>
<td>R thalamus</td>
<td>R parotid gland.</td>
<td>MRI</td>
<td>Cystic mass with enhancing nodule.</td>
</tr>
<tr>
<td>Honeybul</td>
<td>Left temporal lobe</td>
<td>No primary lesion found. Dx’d by R axillary nodal metastasis.</td>
<td>MRI</td>
<td>Enhancing parenchymal nodule.</td>
</tr>
<tr>
<td>Feletti</td>
<td>Pituitary</td>
<td>R groin</td>
<td>MRI</td>
<td>Heterogeneous enhancing mass.</td>
</tr>
<tr>
<td>Abul-Kasim</td>
<td>Leptomeninges</td>
<td>Unknown</td>
<td>MRI</td>
<td>Enhancing meningeal nodule with surrounding vasogenic edema. Leptomeningeal thickening.</td>
</tr>
<tr>
<td>Seaman</td>
<td>L cerebellopontine angle</td>
<td>Right groin</td>
<td>CT, MRI</td>
<td>Heterogeneously enhancing in the intracranial extraaxial mass with vasogenic edema.</td>
</tr>
<tr>
<td>Barkdull</td>
<td>R cerebrum</td>
<td>scalp</td>
<td>CT, MRI</td>
<td>Direct intracranial invasion from bone metastasis to the calvarium.</td>
</tr>
</tbody>
</table>

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Bone metastasis

FDG PET or Bone scintigraphy has higher sensitivity for osseous metastasis than CT


SSTR analogue PET has higher sensitivity for bone metastasis than CT

Buder et al. BMC Cancer 2014, 14:268

Osseous involvement of MCC, although rare, has been described in facial bones, cranium, tibia and spine.

Case 1: CT demonstrates sclerotic lesion in the left ischium (yellow arrow). Tc99m-MDP bone scintigraphy demonstrates focal radiotracer activity (black arrow).

Case 2:
No suspicious bone lesion is identified on CT.
Both Tc99m-MDP bone scintigraphy and F18-FDG PET/CT demonstrate increased radiotracer activity in the right iliac crest (arrow). The lesion was biopsied and confirmed as metastatic MCC.

Tc99m-MDP Bone Scintigraphy
Tc99m-MDP Scintigraphy
MCC can metastasize to weird places...

In advanced malignancy with widespread disease, metastasis can occur in uncommon organs. In our experience at one of the largest MCC centers in the world, it is felt that **MCC metastases to these organs might occur earlier than previously anticipated.**

### Pancreas (case 1, 2)
Metastasis to the pancreas is rare in general. However, our preliminary data (unpublished) shows MCC has higher rate of pancreas metastasis than melanoma (5% vs < 1%)

### Muscle (case 3)
Posterior planar image and SPECT/CT of In-111 Pentetreotide scintigraphy show faint radiotracer uptake in the left psoas muscle (arrows). Post-Gadolinium fat suppressed MRI shows irregular enhancing mass in the left psoas muscle (arrow head)

### Colon (case 4)
Coronal contrast CT shows wall thickening in the distal ilium with aneurysmal dilatation and partial small bowel obstruction.
MCC can metastasize to weird places...

Heart
Cardiac metastasis is rare. Due to its invasiveness, it is difficult to obtain pathologic confirmation. Thus, imaging is important to establish diagnosis of this rare manifestation.

Case 1
Metastasis to the right atrium was irradiated and diminished. However, several months later the patient had recurrence at the base of the left ventricle.

Case 2

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MCC can metastasize to weird places...

73-year-old man with recurrent MCC. In$^{111}$-Pentetreotide scintigraphy (A1,2) show increased radiotracer uptake within the bilateral maxillary sinuses, left supraclavicular lymph node, right adrenal gland (not shown) and right atrium (arrows), indicating somatostatin receptor expression within these known sites of MCC recurrence.

F$^{18}$-FDG PET/CT (B1,2) shows increased FDG uptake in the same areas (arrows).
Peptide Receptor Radionuclide therapy (PRRT)

- Delivers radionuclides directly to tumor cells via SSTR.
- Used for SSTR-positive metastatic well-differentiated GI NETs in Europe since 1990s.
- Retrospective analysis showed promising results for GI NETs.

The SSTR binding peptide is paired with a **beta particle emitting radioisotope** using a chelator (bonding agent). The beta particle irradiate tumor cells.

- Delivers radionuclides directly to tumor cells via SSTR.
- Used for SSTR-positive metastatic well-differentiated GI NETs in Europe since 1990s.
- Retrospective analysis showed promising results for GI NETs.

**[90Y-DOTA^0,Tyr^3] Octreotide**

**[177Lu-DOTA^0,Tyr^3] Octreotide**

**[177Lu-DOTA0,Tyr^3] Octreotate**

**Somatostatin analogue (peptide)**

**SSTR**

FDA recently approved Lutetium **177**-Dotatate for GI NETs in Jan 2018.

Currently, there are a few case reports that demonstrated favorable result on MCC.


However, MCC is very radiosensitive tumor and further investigation is warranted to evaluate efficacy of PRRT on MCC as it might have potential benefit.

**Kunz PL. J Clin Oncol 2015;33:1855.**
Take home messages

1. Introduction
2. Origin of MCC
3. Clinical manifestation
4. Risk factors
5. McPyV
6. Staging
7. NCCN guideline
8. Immunotherapy
9. Role of imaging
10. SNLB
11. CT pattern
12. MRI pattern
13. FDG PET/CT
14. SSTR imaging
15. CNS metastasis
16. Bone metastasis
17. Metastasis to uncommon organs
18. PRRT
19. Take home messages

- MCC is an aggressive cutaneous cancer with tricky clinical manifestation
- Merkel Cell Polyoma Virus (MCPyV) is causally linked to its development
- Antibody to the MCPyV oncoprotein can be used as a “tumor marker” in antibody producers
- Immunotherapy is now a first line systemic therapy
- MCC has unique neuroendocrine features with somatostatin receptor expression which can be used for molecular imaging such as In\(^{111}\) based scintigraphy (SPECT/CT), Ga\(^{68}\) based PET/CT, or potentially Peptide Receptor Radionuclide Therapy (PRRT)
References