VPM 152 - Pigment and other tissue deposits

Shannon Martinson
http://people.upei.ca/smartinson/
Case 1:

Signalment: 2 month old heifer beef calf

Clinical History: Lateral recumbency for 4 days. Tachycardia, tachypnea and dyspnea noted on PE. The calf died following being tube fed by owner

1. Describe the changes in the heart.

2. What could responsible for the white colouration?

3. What is your morphologic diagnosis for these changes?

4. Can you think of a possible cause?

5. Do you think this represents metastatic or dystrophic change and why?

6. What are possible sequellae of the heart lesion?
Case 2:

Signalment: Adult FS dog

Clinical History: Found in the backyard of someone who did not own the dog. The dog would not eat or drink and was brought to the veterinarian. On PE the dog was dull, weak, dehydrated and had pale mm, a CRT=3sec, oral ulcers, and was retching. Blood work indicated increased urea, creatinine and hyperkalemia

1. Please look at all the specimens, but describe the changes only the changes in the heart. (There is also a large artery from a cow with a similar more severe lesions for your interest).

2. What could responsible for the white colouration of the lesion?

3. What is your morphologic diagnosis for these changes?

4. Can you relate this lesion back to the kidney (which is firm, pale, tan and shrunken with bosselation of the surface and a loss of corticomedullary distinction)?

5. Do you think this represents metastasic or dystrophic change and why? What are some other causes of this form of deposition?
Case 3:

Signalment: Adult ewe

Clinical History: None provided.

1. Please look at all the specimens, but describe the changes only the changes in the heart and brain.

2. What could be responsible for the discolouration of the organs?

3. What is the source of this pigment?

4. What is your morphologic diagnosis for these changes?

5. What do you think the significance of this change is?

6. Can you think of a significant disease that arises from the cells that produce this pigment (hint, look at the provided pluck from a cat)?
Case 4:

Signalment: 7 year old male Wheaton terrier

Clinical History: Vomiting and lethargy. An exploratory laparotomy was performed. An abnormal spleen was identified and removed.

1. Please describe the changes in the spleen.

2. What could account for the texture and the colour of the capsular lesion? Look at the histology image.

3. What causes these pigments to form/become deposited?

4. What do you think the significance of this change is?
Case 5:

Signalment: 9 year old. MC, Standard Poodle

Clinical History: Presented with vomiting and diarrhea. The dog had been diagnosed with glomerular disease 2 years previously. Additional clinical findings included suspected aspiration pneumonia and a heart murmur. The dog was euthanized.

1. Please describe the changes in the kidney.

2. Look at the kidney following the application of Lugol’s iodine (see image). What do you think this tells us (what is your suspected diagnosis)?

3. What further tests could be done to help confirm the diagnosis and what would you expect to see?

4. What are possible sources of this deposited substance?
Case 6:

Signalment: Adult male ball python

Clinical History: Recently purchased (3 weeks ago). The snake hadn’t eaten since acquired. He was found dead one morning.

1. Please describe the changes in the kidney and heart.

2. What types of deposits might have this appearance?

3. What further tests could be done to help confirm which is present and what would you expect to see?

4. Look at the provided image. What is your morphologic diagnosis?

5. What is the pathogenesis of this disease (ie what steps lead to the deposition of this substance)?
Case 7:

Signalment: Adult caribou

Clinical History: none provided

1. Please describe the changes in the liver.

2. What types of pigments can cause black colouration in an organ?

3. What type of pigment do you suspect in this case?

4. How is this pigment produced and why do you think it is present in this winding pattern?
Case 1

Signalment:
- 2 month old heifer beef calf

Clinical History:
- Lateral recumbency for 4 days
- Tachycardia, tachypnea and dyspnea
- Calf died following being tube fed by owner
Case 1

Scattered throughout the left and right ventricular myocardium and endocardium are multifocal, irregular, 1–3 cm streaky areas of white discolouration.
Scattered throughout the left and, less so, the right ventricular myocardium and endocardium are multifocal, irregular, 1 – 3 cm, streaky areas of white discolouration.
Case 1

• Myocardial degeneration, necrosis and mineralization, multifocal, acute, severe

Morph Diag
• Myocardial degeneration, necrosis and mineralization, multifocal, acute, severe
Myocardial degeneration, necrosis and mineralization, multifocal, acute, severe

Does this represent dystrophic or metastatic mineralization?
Case 1

Dystrophic mineralization

- This is white muscle disease (WMD) – it represents mineralization secondary to myocardial necrosis
- Necrosis in the heart and skeletal muscle occurs as a result of selenium / vitamin E deficiency

What are possible sequellae to this?
Does this represent dystrophic or metastatic mineralization?
Sequelae: Affected animals often develop heart failure leading to pulmonary edema and congestion – edema can predispose to pneumonia which was also seen in this case.
Signalment:
• Adult FS dog

Clinical History:
• Found in the backyard
• Would not eat or drink
• PE: dull, weak, pale mm, dehydrated, CRT=3sec, oral ulcers, retching
• Blood work: Hyperkalemia
Case 2
Case 2

Description

Multifocal irregular streaky, white (chalky) plaque-like deposits are present in the endocardium of the atria and the intima of the pulmonic trunk.
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Case 2

Endocardial mineralization, multifocal, acute, moderate

Morph Diag
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- Endocardial mineralization, multifocal, acute, moderate

Can you relate this lesion back to the changes in the kidneys?
Case 2

Does this represent dystrophic or metastatic mineralization?

Metastatic calcification (there is some argument that uremia may actually damage the tissues....)

End-stage kidney (kidney failure) → Causes abnormal Ca and P metabolism

FYI:
• P not excreted from kidneys
• Vit D not activated in the kidney
  • Ca not absorbed from GIT
  • Parathyroid stimulation → ↑PTH → release of Ca from bone and increased Ca absorption in GIT → ↑Ca:P product → Calcification of soft tissues

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  - Parathyroid stimulation $\rightarrow$ ↑PTH
    $\rightarrow$ release of Ca from bone and increased Ca absorption in GIT
    $\rightarrow$ ↑Ca:P product $\rightarrow$ Calcification of soft tissues

What are other causes of metastatic mineralization?
- Primary hyperparathyroidism
- Vitamin D toxicosis
- Paraneoplastic syndrome (lymphoma for example)

Can you relate this lesion back to the changes in the kidneys?
Case 3

Signalment:
• Adult ewe

Clinical History:
• None provided
• Incidental findings at necropsy
Case 3

Description

Scattered multifocally within the leptomeninges overlying the cerebrum of the brain and in the intima of the aorta, there are stippled deposits of back pigment.
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What is responsible for the discolouration of these organs?
Melanin

What is the source of this pigment?
Melanocytes
Case 3

Morph Diagnosis

What is responsible for the discolouration of these organs?
Melanin

What is the source of this pigment?
Melanocytes
Morph Diagnosis

- Melanosis, multifocal, meninges and aortic intima

What is the significance of this change?

Incidental – not of any significance!

Melanocytes occur normally at many sites in the body (varies by species/breed)

When increased term melanosis is used
Can you think of any significant diseases which may arise from melanocytes and cause pigmented lesions?
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Melanoma
Signalment:
- 7 year old male Wheaton terrier

Clinical History
- Vomiting, lethargy
- Exploratory laparotomy performed
- Abnormal spleen identified and removed.
Case 4

Multifocal small (1–4 mm) white to yellow-brown gritty plaques are scattered over the capsular surface.

The splenic margins are rounded.
Case 4

**Description**

- Multifocal small (1 – 4 mm) white to yellow-brown gritty plaques are scattered over the capsular surface
- The splenic margins are rounded

**What deposits could account for the gritty texture? The yellow-brown colour?**
Histology:
- Mineral deposits – Impart gritty texture and white colour
- Hematoidin – bright yellow colour
- Hemosiderin – brown-gold colour
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What causes hematoidin and hemosiderin to form?
Both are breakdown products of erythrocytes and are deposited at sites of congestion / hemorrhage:
- Hemosiderin – represents stored iron that is recovered from hemoglobin
- Hematoidin – locally precipitated bilirubin

Siderofibrosis = Gamna-Gandy bodies (FYI only)
- Incidental finding/senile change
- Possible sequela of prior hemorrhage
- Not a good reason to remove a spleen
Signalment:
- 9 year old. MC, Standard Poodle

Clinical History:
- Presented with vomiting and diarrhea
- Glomerular disease for at least 2 years duration.
- Aspiration pneumonia
- Heart murmur III / IV
- Dog was euthanized.
Case 5

The kidney is diffusely pale yellow to gold and has multifocal small depressions in the capsular surface and a finely pitted appearance on section.
Case 5

Description

- The kidney is diffusely pale yellow to gold and has multifocal small depressions in the capsular surface and a finely pitted appearance to the cortex on section.
Case 5

Description

- Multifocal random small punctate dark foci were seen scattered over the entire cortical surface of both kidneys after Lugol’s iodine was applied.
Suspected diagnosis
- Renal amyloidosis

Further tests?
- Histopathology with Congo red staining

Possible sources of amyloid?
- Misfolded protein:
  - Protein AA from serum amyloid A in chronic inflammation*
  - Protein AL from Ig light chains (plasma cell neoplasia)
  - Family amyloid (Shar pei)
  - Endocrine amyloid (via neoplasia or degeneration)

*Chronic inflammation
Case 5

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  - Endocrine amyloid (via neoplasia or degeneration)
Case 6

Signalment:
• Adult male ball python

Clinical History:
• Recently purchased (3 weeks)
• Wasn’t eating
• Found dead
Case 6

Normal heart

Normal kidney
Case 6

• Chalky - soft, granular white - yellow, plaque-like deposits are present diffusely in the pericardial sac and on the epicardial surface.

• Similar deposits are present multifocally within the renal parenchyma and the kidneys are markedly swollen (enlarged).
Case 6

Description

- Chalky-soft, granular white-yellow, plaque-like deposits are present diffusely in the pericardial sac and on the epicardial surface.
- Similar deposits are present multifocally within the renal parenchyma and the kidneys are markedly swollen (enlarged)
What types of deposits could account for this appearance?

- Urates / uric acid*
- Mineral? Possibly

How could you tell the difference?

- Histology
- Cytology (when fresh)
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- Urates / uric acid*
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How could you tell the difference

- Histology
- Cytology (when fresh)

What would your morph diagnosis be if this is urate:

- Pericardial and renal (visceral) gout, diffuse and multifocal to colaescing (respectively), severe

What is the pathogenesis of urate deposition in tissues?

Gout tophus
Case 6

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How could you tell the difference

- Histology
- Cytology (when fresh)

What would your morph diagnosis be if this is urate:

- Pericardial and renal (visceral) gout, diffuse and multifocal to coloescing (respectively), severe

What is the pathogenesis of urate deposition in tissues?

Renal disease or
Dehydration or
Excess dietary protein

Increased uric acid production or decreased excretion (=end product of nitrogen metabolism in reptiles)

Increased uric acid in the blood (hyperuricemia)

Uric acid deposition in tissues

Gout tophus
Case 7

Signalment:
• Adult Caribou

Clinical History:
• None provided
Several round cyst-like cavities, each surrounded by a thick fibrous capsule, are present multifocally within the liver. Within these structures, there are coiled trematodes. The flukes are leaf shaped, dorsoventrally flattened, measuring ~5 – 8 cm x 3 – 4 cm with an oral and ventral sucker. Fine black tortuous linear tracts are present multifocally in the parenchyma.
Several round cyst-like cavities, each surrounded by a thick fibrous capsule, are present multifocally within the liver. Within these structures, there are coiled trematodes.

The flukes are leaf shaped, dorsoventrally flattened, measuring ~5 – 8 cm x 3 – 4 cm with an oral and ventral sucker.

Fine black tortuous tracts are present multifocally in the parenchyma.
Case 7

What types of pigments can cause black colouration in an organ?

- Melanin
- Anthracosis
- Acid hematin
- Parasite derived pigment

Which type of pigment do you think is present in this case?

- Parasite derived pigment

How is the pigment produced and why do you think the pigment is present in this pattern?

- The flukes ingest blood and digest it producing pigment → iron porphyrin or hematin
- Pigment exits via the oral opening (no other exit) = fluke puke
- The pattern reflects migration of the flukes thorough the liver
Questions?