Vertebral hemangioma: radiologist, do you really know what you are dealing with?

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Learning objectives

Our purposes are:

To illustrate the **spectrum of imaging features of vertebral hemangioma** (VH), a very common finding at CT and MRI imaging of the spine, usually discovered incidentally.

To describe the **most common findings** of VH but also the **rarest and most confounding ones**.

To underline the **CT and MRI key points** that allow the correct diagnosis.

We also propose a **simple diagnostic strategy** to guide the Radiologist in addressing **differential diagnosis** such to avoid potential diagnostic pitfalls.

Enjoy your lecture!
Background

EPIDEMIOLOGY

VH is the most common benign tumour of the spine, first described by Virchow in 1867[1, 2]. It has an estimated incidence of 10% to 12% in the population based on large autopsy series and a large review of plain spine films[3].

The prevalence of VHs seems to increase with age and the peak incidence is in the fourth to sixth decade. A slight female predominance (M:F=1:2) has been observed[1,4].

Multiple VHs can occur in about a third of cases with most lesions found in the lower thoracic and lumbar vertebrae[4,5].

HISTOPATHOLOGY

The etiology of VH remains still unknown. According to WHO classification of tumours of soft tissue and bone (see Fig. 2 on page 6), VH is considered to be a benign vasoformative neoplasm of bone or in alternative a developmental condition of endothelial origin[6].

Macroscopically, VH appears as a soft well demarcated dark red mass that can also have a honey-comb appearance with intralesional sclerotic bone trabeculae and scattered blood-filled cavities[6].

VHs are usually confined to the vertebral body but occasionally they can involve the entire body of the vertebra and extend into the pedicles, arches and spinous processes[1]. Rarely an aggressive type of VH may extend into the epidural space and cause spinal cord compression (see Fig. 3 on page 6)[1,4,5].

Histologically, VH is composed of multiple thin fully developed blood vessels, surrounded by fat infiltrating the medullary cavity between sclerotic bony trabeculae (see Fig. 4 on page 7)[1,5]. The trabeculae become thickened due to reinforcement of the osseous network adjacent to the vascular channels of the lesion that have caused bone rarefaction[7].
On the basis of their predominant vascular channels, VHs are classified in four main groups: capillary, cavernous, arteriovenous or venous. The most common histologic type is cavernous hemangioma[8].

The tissutal composition of VHs can vary from predominantly fatty lesions to hemangiomas composed largely of vascular stroma with little or no adipose tissue. It has been suggested that VHs with a high fat content may represent an inactive form of this lesion, whereas lesions with a low fat content are associated with moderate to intense hypervascularization and are more likely to cause pain or neural compression[1,8,9].

**CLINICAL FEATURES**

The vast majority of VHs are asymptomatic and do not usually display malignant behaviour. Asymptomatic VHs are generally lesions that do not expand or extend beyond the vertebral body[3,10].

Nevertheless, a rare locally aggressive subtype of VH, representing only 1% to 2% of lesions, may extend epidurally and cause spinal canal stenosis, spinal cord and nerve roots compression, epidural hemorrhage and vertebral compression fracture or a combination of these effects. Patients with aggressive VHs complain of pain and/or symptoms of neurologic compression[4].

Aggressive VHs can occur in patients of any age, with a peak prevalence in young adults, and preferentially in the thoracic spine [8,9,10]. Pregnancy may contribute to the development of aggressive and symptomatic VHs, which is hypothesized to be due to an increase in blood volume and cardiac output[2,4].

Aggressive VHs are difficult to differentiate from other spinal lesions because they can mimic a primary bony malignancy or a metastatic disease, both clinically and radiologically[11].

**TREATMENT OPTIONS**

Asymptomatic VHs have an excellent prognosis and regular monitoring of these lesions is not necessary unless back pain or neurologic symptoms occur[12].

Aggressive and symptomatic VHs may have a variable prognosis, depending on size of lesion, degree of epidural extension and presence/absence of cord compression[12].
Given to the highly vascular nature of these lesions, an accurate preoperative radiological diagnosis of aggressive VH is important to prevent unexpected hemorrhagic complications. Many recent reviews recommend the routine use of angiography, both as a diagnostic and therapeutic tool since it allows embolization of the lesion, as a preoperative adjunct or sole therapy[11].

After diagnosed correctly, several treatment modalities are available in the management of aggressive VHs. These modalities include a combination of observation, embolization, sclerotherapy, surgery with decompression and/or resection and stabilization, vertebroplasty or radiotherapy[4,11].

Despite the number of treatment strategies, there is still no consensus on the best therapeutic options for aggressive VHs, thus a multidisciplinary approach is preferable, including interventional radiology.
Fig. 2: WHO Classification of Bone Tumours

© Fletcher CDM, Krishnan Unni K, Mertens F. World Health Organization Classification of Tumours: Pathology and Genetics of tumours of soft tissue and bone. 2002.
Fig. 3: Multiple benign-appearing small blood vessels (green arrows) with interspersed fibroadipose tissue adjacent to a fragment of bone (blue arrow).

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Anatomical Considerations

- The vast majority of VHs are confined to the vertebral body; they can be small or occupy the entire vertebral body
- Uncommonly they can involve the posterior elements (10-15%) and extend into:
  - the pedicles
  - the arches
  - the spinous processes.

Rarely an aggressive type of VH may extend into epidural space and cause spinal cord and nerve roots compression.

Fig. 4

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Imaging findings OR Procedure details

As mentioned above, VH includes a spectrum of clinical entities from the incidental asymptomatic lesion to the rare aggressive tumour that may compress the spinal cord. This range of clinical expressions corresponds to a wide range of imaging features, making the diagnosis difficult.

Most of VHs have a typical appearance with routine radiography, CT and MRI. Nevertheless, in a review by Cross et al, it was demonstrated that characteristic findings associated with VHs were absent in 35% of plain films, 20% of CT scan and 48% of MRI scans of aggressive lesions, resulting in an inability to make a correct diagnosis[10].

The relative amount of adipose tissue and vascularity within a VH may vary and this issue affects not only the clinical features but also the imaging appearance: VHs with a high fat content may represent an inactive form of this lesion, whereas lesions with a low fat content are associated with moderate to intense hypervascularization and are more likely to display an aggressive behaviour[1,8,9,11].

Hereunder, we describe the most common findings of VH but also the rarest and most confounding ones, usually associated with clinically aggressive VHs.

GENERAL IMAGING FEATURES

- **Location**: usually confined to the vertebral body, that can be partially or completely involved; occasionally VHs can expand the cortical margins. In 10-15% of cases they can extend into the posterior elements (pedicles, arches, spinous processes). The aggressive type of VH may have an extra-osseous soft tissue extension into the paraverteral and/or epidural space.
- **Size**: variable. They can be small or occupy the entire vertebral body.
- **Morphology**: most cases are well-circumscribed. Aggressive lesions may have poorly defined margins.
- **Number**: solitary or multiple (30%).

RADIOGRAPHY

Typical radiographic aspect of VH:
• Prominent, thickened trabeculae vertically coursing through the vertebral body, giving the lesion a striated aspect, described as the "corduroy cloth" or "honeycomb" appearance (see Fig. 5 on page 14 and Fig. 6 on page 14).
• Normal cortex with distinct cortical margins.
• Maintained vertebral body height.
• Partial involvement of vertebral body.
• Normal neural arch and pedicles.

Warning! You can also find these features, more often associated with an aggressive behavior…

• Irregular vertical trabeculation.
• Involvement of entire vertebral body and even of neural arches and pedicles.
• Abnormal vertebral body texture.
• Poorly defined and expanded cortex.
• Non visualization of the pedicles, simulating a metastasis.
• Loss of vertebral height.
• Vertebral collapse.
• No abnormality (normal study).

COMPUTED TOMOGRAPHY

Because of the complex anatomy of the vertebrae, CT scanning is more sensitive than plain radiography for evaluating the extent of osseous involvement and the degree of cortical bone loss.

Typical CT aspect:

• Well-circumscribed hypodense lesion centered in the vertebral body with striated appearance, best appreciated on coronal and sagittal reformatted images, due to the thickened trabeculae longitudinally oriented (Fig. 7 on page 15).
• On axial images small dots of high attenuation, representing thickened vertical trabeculae, seen in cross-section (the polka-dot sign), interspersed with areas of fat attenuation within the lesion (Fig. 7 on page 15).
• Maintained vertebral body height.

Warning! You can also find these features, more often associated with an aggressive behaviour…

• Irregular vertical trabeculae.
• Poorly defined and expanded cortex.
• Lytic zones, even in the pedicles, simulating a metastasis.
• Extra-osseous soft tissue extension (into the paravertebral and/or epidural space).
• Aggressive lesions show avid contrast-enhancement.

MAGNETIC RESONANCE

MRI features largely depend on the proportion on fat and vascularity of the lesions and sagittal and axial T1 w.i. sequences are the most useful to characterize composition[8,9,10,13,14].

Areas of high fat content within VH appear as areas of high signal intensity. Increased signal intensity on T2-weighted images is related to the water content of the lesion, due to both the amount of vessels and interstitial edema of VH, and also to the adipose tissue of the lesion.

Moreover, MRI is the best imaging modality for the evaluation of the aggressive characteristics and axial T2 w.i. and enhanced T1 w.i. well depict epidural extension and neural compromise[12].

Typical MRI aspect of VH with high fat content:

• Hyperintense on T1 w.i. and T2 w.i. with a mottled appearance (Fig. 8 on page 16).
• On sagittal images, low-signal-intensity vertical struts within the lesion, corresponding to the thickened trabeculae (Fig. 8 on page 16).
• On axial images, intraosseous signal voids, corresponding to the vertical trabeculae seen in cross sections, that appear as punctate areas of iso- to hypointensity within the intra-osseous portion of the tumor. This MRI feature resemble the "polka-dot" sign seen on axial CT images (Fig. 8 on page 16).
• Well defined lesion with sharp margination in relation to fat marrow (Fig. 9 on page 17).
• Variable degree of contrast enhancement, more often avid (Fig. 9 on page 17).
• On fat saturation sequences (T2 w.i. and enhanced T1 w.i.), VHs typically retain some high signal due to vascular components. These feature permit to distinguish VH from focal fatty marrow (Fig. 9 on page 17).

This typical MRI aspect enter in differential diagnosis whit these pathologies (Fig. 10 on page 18).

• Focal fatty marrow (Fig. 11 on page 19)
• Degenerative end-plates, type II (Fig. 12 on page 20)
• Spinal radiation treatment (Fig. 13 on page 21).
• Paget's disease (**Fig. 14 on page 22**)

**Warning!** Occasionally VHs with a minor fatty content are isointense to hypointense on T1 w.i, and can simulate **bone metastasis**! Signal voids are the most useful additional MR imaging since their detection within the hypointense lesion on T1 w.i addresses to a VH.

In these cases CT appearance can be still classic for VH and permit to distinguish these two clinical entities. Furthermore remember that metastasis characteristically extends into pedicles (**Fig. 17 on page 25**).

**Aggressive VH's aspect on MRI imaging.**

- Isointense to hypointense on T1 w.i.
- Hyperintense on T2 w.i.
- Avid contrast enhancement.
- Extra-osseous soft tissue extension (into the paraverteral and/or epidural space).
- Extraosseous components tend not to show high signal intensity on T1-weighted images owing to the paucity or absence of adipose tissue.
- Pathologic fracture, vertebral collapse.

**Keep in mind these tumors that may enter in the differential diagnosis with an aggressive VH (**Fig. 15 on page 23**)!**

- Bone metastasis (**Fig. 16 on page 24** and **Fig. 17 on page 25**)
- Plasmocytoma (**Fig. 18 on page 26**)
- Lymphoma (**Fig. 19 on page 27**)
- Chordoma (**Fig. 20 on page 28**)
- Aneurismal bone cyst (**Fig. 21 on page 29**).

**ANGIOGRAPHY**

- Angiography confirm the vascular nature of VH (**Fig. 22 on page 30**).
- The arteriographic appearance is usually characteristic: dilatation of arterioles of the vertebral body, multiple blood pools in the capillary phase, and, finally, intense opacification extending beyond the normal hemivertebral territory throughout the entire vertebral body.
- Appearance ranging from normal vascularity to marked hypervascularity.
- Aggressive lesions stain vividly.
- Opacification beyond normal cortical limits corresponds to extension of the VH into the paravertebral soft tissues and the spinal canal.

**NUCLEAR MEDICINE FINDINGS**
• On fluorodeoxiglucose (FDG) positron emission tomography (PET) VHs usually appears as hypometabolic regions.
• On bone scintigraphy, the appearance of VHs ranges from photopenia ("cold lesions") to a moderate increase in radiotracer uptake.
• On Tc-99m red blood cell SPECT, VHs are areas of increased uptake.
Images for this section:

Fig. 5

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Plain Film: VH vs Paget

VH can be differentiated from Paget’s disease (PD), in which characteristic sclerotic lines, due to hypertrophy and thickening of the trabecular bone, are parallel to the end plates.

In PD, the combination of trabecular bone hypertrophy and thickening at the end-plates with apposition/absorption on the periosteal/endosteal surfaces at the anterior and posterior vertebral borders leads to the “picture frame” sign.

Look again at the lateral view (A) depicting a VH affecting the body of L4 with typical vertical striations and then compare it with the lateral radiograph (B) of a patient affected by PD (courtesy of Dell’Atti et al, Skeletal Radiol 2007) that demonstrates expansion of the vertebra with characteristic sclerotic lines parallel to the end plates due to trabecular hypertrophy, an “early” sign of PD.

The trabecular thickening in PD is coarser than the more delicate pattern seen in VH. In addition, the “picture frame” appearance is not seen in VH.

Artistic images are taken from “Ross et al, Diagnostic Imaging – Spine. 2004. Amyris”
CT: Typical Aspect of VH

Sagittal (A) and coronal (B) reformatted CT images (bone algorithm, 1.25 and 2.5 mm thickness) of an asymptomatic and incidental VH of L4 (A) show sparse, vertically oriented, thickened trabeculae surrounded by hypodense fat and interspersed blood vessels. The cortex appears normal and there is no extra-osseous extension.

This axial CT image of the same Patient (C) demonstrates typical polka-dot appearance of the VH. This sign is seen on transverse CT images of vertebral bodies. It's produced by thickened trabeculae seen in cross section as small punctate areas of high attenuation, simulating the polka-dot pattern on clothing. Remember that the trabecular thickening is due to reinforcement of the osseous network adjacent to the vascular channels of the lesions that have caused bone resorption.

Fig. 7

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Fig. 8

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RM: Typical Aspect of VH # 2

Here it is an essay of the typical RM appearance of VH in different sequences:
- Hyperintense on sagittal T1 WI (A)
- Hyperintense on sagittal T2 WI (B)
- Hyperintense on coronal T2 WI with fat-suppression (C) due to vascular components within the lesion. The signal from the fatty amount of the lesion is instead suppressed.
- Hyperintense with contrast enhancement on sagittal (D) and coronal (E) T1 WI post gadolinium with fat suppression.

Fig. 9

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Differential Diagnosis # 1

The DDX for a “typical” VH includes….

- Focal fatty marrow
- Degenerative end-plates, type II
- Paget’s disease
- Spinal radiation treatment

….let’s think to all these clinical entities when you “deal” with a suspected typical VH!

Fig. 10

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Focal fatty marrow vs VH

Here on the left an incidental focal area of hyperintensity on T1 WI (A) and T2 WI (B) in the vertebral body of L4.

What is it? It’s a VH?

Focal fatty marrow appears as incidental rounded focus of hyperintensity on T1 WI (A) and T2 WI (B), as VH like. Furthermore, when fat signals are removed, T1 WI post-gadolinium with fat suppression (C) demonstrates hypointensity and absence of contrast enhancement. VH instead typically retains some high signal due to vascular components and could have a variable degree of contrast enhancement.

Fig. 11

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Modic type II end-plates degeneration vs VH

Modic type 2 changes are hyperintense on T1 WI and isointense or slightly hyperintense on T2 WI.

To easily identify Modic type II changes you must look for:
• parallel involvement of two adjacent end-plates about an intervertebral disc level.
• severe disc degeneration with loss of disc space height.

The CT study (C) confirms the diagnosis of Modic type II changes demonstrating severe sclerosis of two adjacent end-plates associated with loss of disc space height.

Do you know that...
Modic type 2 changes are associated with conversion of normal red hemopoietic bone marrow into yellow fatty marrow as a result of marrow ischemia?

Fig. 12

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Fig. 13

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Paget’s disease vs VH

Sagittal T1 WI (A) and T2 WI (B) show diffuse heterogeneous bone marrow signal containing fat. It seems to appreciate multiple focal areas of hyperintensity in C2 to D1 vertebral bodies.

What are them, multiple hemangiomas?

Don’t hurry up and note also the thickened low signal cortex.

Don’t you think it could be... Paget’s disease (PD)?

The same Patient performed a CT study that depicts a striated appearance that can resemble “the polka-dot sign” on axial sections through vertebral body (D). However, in sections adjacent to the vertebral plate (C), horizontal Pagetic thickened trabeculae are seen, interspersed among adipose tissue. This aspect permits differentiation from VH.

Furthermore, in PD the vertically oriented trabeculae haven’t a regular pattern like instead in VH have.

Fig. 14

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Differential Diagnosis # 2

The DDX for an “atypical” VH includes:

- Bone metastases
- Lymphoma
- Plasmocytoma
- Chordoma
- Aneurismal bone cyst

...let’s think to all these clinical entities when you “deal” with a suspected atypical VH!

Fig. 15

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Metastasis vs VH #1

The MRI study demonstrates a discrete lesion with low signal on sagittal T1 WI (A) and high signal on sagittal T2 WI (B) in L3 vertebral body of a patient affected by prostate cancer.

Furthermore, on axial T1 WI with fat suppression post-gadolinium administration (C) the lesion enhances vividly.

Is it a metastasis or a VH with a minor fatty content?

The patient undergoes another MRI lumbar study one month after the end of hormonotherapy.

On sagittal T1 WI (D) and T2 WI (E) with fat suppression the lesion now appears smaller thus supporting the hypothesis of a vertebral metastasis from hormonoresponsive prostate carcinoma.

Fig. 16

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MRI study of a Patient with low back pain demonstrates a lesion involving the body and the left transverse process of L3 which appears hypointense on T1 WI (A and C), hyperintense on T2 WI (B) and slightly hyperintense on enhanced T1 WI with fat suppression (D). Note also the extension into epidural space (red arrow). Even if the Patient have no history of cancer, these MRI features are highly suspicious for bone metastasis.

The same Patient performed a CT study that depicted a striated appearance on coronal (E) reformatted images and the polka-dot sign on axial scan (F), findings that oriented for the hypothesis of an aggressive VH. Patient underwent surgical decompression for the epidural extension causing cord compression. The lesion was histologically diagnosed as VH.

Fig. 17

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Man, 64 y.o with lumbar pain and neurologic symptoms. Sagittal reformatted CT image (A) of L2 shows a predominantly lytic lesion centered in the vertebral body and involving the neural arch. It seems to appreciate a certain striate aspect due to vertical thickened trabeculae but axial scan (B) depicts the unique “mini brain” sign (blue arrow) created by a lytic vertebral lesion with cortical preservation. This is a characteristic sign of plasmocytoma.

Generally plasmocytoma has low signal on T1 WI and high signal on T2 WI. In this patient the lesion was dishomogeneously hypointense both on T1 and T2 WI. Axial contrast-enhanced T1 WI with fat suppression showed homogeneous enhancement of the lesion with epidural extension and cord compression (red arrow).

Fig. 18

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Man, 82 y.o. with symptoms suggestive of acute thoracic myelopathy. The CT study performed in emergency setting didn’t detect any abnormalities appreciable with the method (A and B).

The MRI investigation instead showed a posterior epidural mass, hypointense on T1 WI (B) and slightly hyperintense on T2 WI with fat suppression (C), centered at T4-T6 level and compressing the spinal cord. The lesion manifested also moderate enhancement (D) on T1 WI post gadolinium with fat saturation.

Analogous signal intensity changes with a mottled diffuse pattern were observed also in the vertebral bodies at D4-D6 level, suggestive of permeative bone marrow infiltration. These MRI features were suspected for non Hodgkin lymphoma. The biopsy confirmed the MRI suspect. Generally aggressive VHs are extremely hyperintense on T2 WI and this aspect provides a useful distinction from lymphoma.

Fig. 19

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Coronal reformatted CT image (A) of this 74 y.o. man complaining of low back pain showed a destructive lytic lesion centered in the body of L4. Note polycyclic margins with scalloped appearance (red arrows) on axial CT scan (B).

The MRI study confirmed the presence of this well-circumscribed lesion, hypointense on T1 WI (C, see blue arrow) and hyperintense on T2 WI (D) (even with fat suppression, E) with the evidence of multiple hypointense fibrous septa within the lesion (green arrow).

These features oriented for a chordoma that was afterwards histologically proven. The high signal intensity on T2 WI reflects the high water content of chordoma.

VHs too are extremely hyperintense on T2 WI but you can easily address DDX by noting multiple septa and hemosiderin foci, both appearing hypointense on T2 WI, which are typical findings of chordoma.

Fig. 20

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Some Authors include aneurysmal bone cyst (ABC) in the differential diagnosis of aggressive VHs

- Expansile benign neoplasm containing thin-walled, blood-filled cavities with fluid-fluid levels.
- ABC predominantly afflict children.
- Imaging findings:
  - More often arises in neural arch and usually extends unilaterally to produce an eccentric paravertebral lesion.
  - Commonly extends also into vertebral body (70-90%) and epidural space.
  - Balloon-like expansile and osteolytic lesion.
  - Cortical thinning with focal destruction common, well depicted on CT scanning.
  - Tumor matrix absent.
  - On MRI, cystic spaces with fluid-fluid levels of mixed signal intensity due to hemorrhage and blood product sedimentation, best appreciable on T1 and T2 WI.
  - Enhancement at periphery, septae between cysts.
  - A distinct solid variant of ABC exists that shows usually diffuse enhancement.


Solid variant of aneurysmal bone cyst of the thoracic spine: a case report


Fig. 21

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Angiography: Typical Aspect of VH

Angiography can be an useful confirmatory test since the arteriographic appearance is usually characteristic, ranging from normal vascularity to marked hypervascularity (A).

Aggressive lesions usually stain vividly and intense opacification extends beyond the normal hemivertebral territory throughout the entire vertebral body and also the neural arch and in the epidural space (B).

In this VH, the digital subtraction angiographic (DSA) study (C) demonstrates abnormal pooling of contrast in the entire vertebral body (red arrow) and also in the posterior arch (green arrow).

Fig. 22

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Conclusion

VH, a very common finding at CT and MRI imaging of the spine, includes a spectrum of clinical entities from the incidental asymptomatic lesion to the rare aggressive tumour that may compress the spinal cord.

This range of clinical expressions corresponds to a wide range of imaging features, making the differential diagnosis difficult.

Radiologists should be aware of the various imaging features of the aggressive type of VHs because they can simulate more sinister pathologies like a primary bony malignancy or metastasis.

The relative amount of adipocytes, vessels and interstitial edema of a VH may vary and this issue dictates the MRI imaging appearance.

Awareness of the range of MRI features is important since this is frequently the initial investigation in patients presenting with symptoms of neural compression.

In case of atypical MRI aspect, in order to provide a complete differential diagnosis, we propose a simple diagnostic strategy (Fig. 23 on page 33) to guide the Radiologist to reach the correct diagnosis such to avoid potential diagnostic pitfalls.

Since CT is typical in the vast majority of cases (80% estimated), this is a useful confirmatory test if MRI features are suspicious but non diagnostic of aggressive VH.

Also angiography can be an useful adjunct in the evaluation of aggressive VHs, both as a diagnostic and therapeutic tool since it allows embolization of the lesion, as a preoperative strategy or sole therapy.

See Case n°1 (Fig. 24 on page 33)

See Case n°2 (Fig. 25 on page 34 and Fig. 26 on page 35)
Fig. 23: Our diagnostic strategy - MRI first performed.

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Case n°1

Male, 52 y.o. Story of colorectal cancer with disease free FUP. MRI performed for low back pain.

Fig. 24

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The initial MRI examination of the lumbar spine demonstrates mild and inhomogeneous hyperintensity of L4 vertebra on both sagittal T1 WI (A) and T2 WI (B), sclerotic and thickened end-plates and a slight loss of vertebral body height. The CT examination (C, D), however, shows typical coarsely trabeculated lesion involving the body and the posterior elements of L4, highly suggestive of VH. The posterior elements are also expanded. Notice the “polka-dot” sign on axial scan (D). In consideration of the Patient’s story of colorectal cancer, a PET-CT examination (E) was also performed and it was negative for metabolically active disease.
MRI investigation of the lumbar spine showed mottled hyperintensity both on sagittal T1 WI (A) and T2 WI (B) involving the entire vertebral body of T8 and intrasosseous signal voids (green arrows) within the lesion on axial images (D, E). These MRI findings were highly suggestive for VH even if the lesion didn’t enhance after gadolinium administration (C and E). This behaviour reflected the prevalent fatty stroma of this VH.

Moreover, in the epidural posterior space between T8 and T10 vertebrae, MRI study also depicted an expansile lesion causing cord compression, isointense on T1 WI (A) and hyperintense on T2 WI (B) with intense enhancement (C and E) on T1 WI post gadolinium with fat suppression (see pink arrows). The MRI features of this extradural lesion supported the hypothesis of an aggressive extra-osseous portion of VH which involved the entire body of D8 and extended into the neural arch. The hypointensity on T1 WI reflected the major vascular content within this portion of the lesion. To better assess this hypothesis a CT study was performed...

Fig. 25

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The CT study confirmed the hypothesis of VH of T8 vertebral body depicting the typical striated appearance on sagittal reformatted image (A) and the polka dot sign on axial scan (B and D), well appreciable also in the volume rendering (C).

The CT examination depicted also erosion and enlargement of the neural arch (D, blue arrows), due to the epidural lesion which demonstrates a soft tissue attenuation (C, red arrow).

Digital subtraction angiography (DSA) at T8 vertebral level confirmed the vascular nature of the lesion and demonstrated abnormal pooling of contrast in the vertebral body that extend beyond the hemivertebral territory throughout the entire vertebral body and also into the posterior arch (E, F).

Embolization was not performed and surgical option was preferred. Diagnosis of VH was confirmed by histological analysis.

Fig. 26

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References


