

## Multiple Faces of Focal Nodular Hyperplasia

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Focal nodular hyperplasia (FNH) is the 2<sup>nd</sup> most common benign liver tumor with the prevalence of about 0.9% in the general population<sup>(1)</sup>. FNH is a focal aggregate of normal hepatocytes that is abnormally arranged and held together by a fibrous meshwork<sup>(2)</sup>. It is considered to be a regenerative lesion, not a neoplasm and believed to be triggered by vascular malformation or vascular injury<sup>(3)</sup>. FNH is usually small ( $\leq 5$  cm), has no capsule, and contains central scar which is composed of blood vessels and bile ductules. Typical findings of FNH in various imaging findings are as following<sup>(4-6)</sup>:

**US:** iso-echoic, or slightly hypo-/hyper-echoic to the adjacent liver, make it difficult to visualize (Figure 1A).

**CT:** iso-/hypo-density mass at plain CT; homogeneous intense enhancement with reticular or lobulated contour at arterial phase (except for central scar), iso-density to normal liver at portal venous phase, and enhancement of central scar at delayed phase CT (Figure 1 B-E).

**CT Angiography (CTA):** a large feeding artery in central scar location, which gives out peripheral branch in a "spoke wheel pattern" and a large draining vein (Figure 2)

**MR:** iso-/low-signal intensity (SI) at T1W; iso-/high-SI at T2W with hyperintense central scar; enhancement pattern after IV gadolinium is similar to CT (Figure 3). MR with IV gadolinium is very sensitive and specific for diagnosis of FNH.

**MR with Kupffer cells specific contrast agent (Resovist):** signal drop at T2, except for the central scar, secondary to uptake of Kupffer cells within FNH (Figure 4).

**MR with hepatobiliary specific contrast agent (Primovist):** signal gain at T1 delayed 15-30 min

hepatobiliary phase, except for central scar, secondary to uptake of hepatocytes within FNH (Figure 5).

**Sulfur colloid radionuclide scan:** positive uptake about 50% of cases secondary to Kupffer cells within FNH.

**HIDA scan:** may show delayed excretion and washout because bile ducts within FNH do not connect to the main biliary tree.

However, atypical appearance of FNH is not uncommon and found in more than 50% of cases<sup>(7,8)</sup>. This article is a pictorial essay of multiple features of atypical FNH, emphasizing on appearance of visual images. Brief explanation for each atypical finding is provided, if possible.

**Atypical #1: no central scar** (Figure 6)

Central scar may not be visualized, particularly small FNH. About 1/3 of FNH may not show central scar, which makes it difficult to distinguish it from hepatic adenoma or hepatocellular carcinoma (HCC).

**Atypical #2: pseudocapsule** (Figure 7)

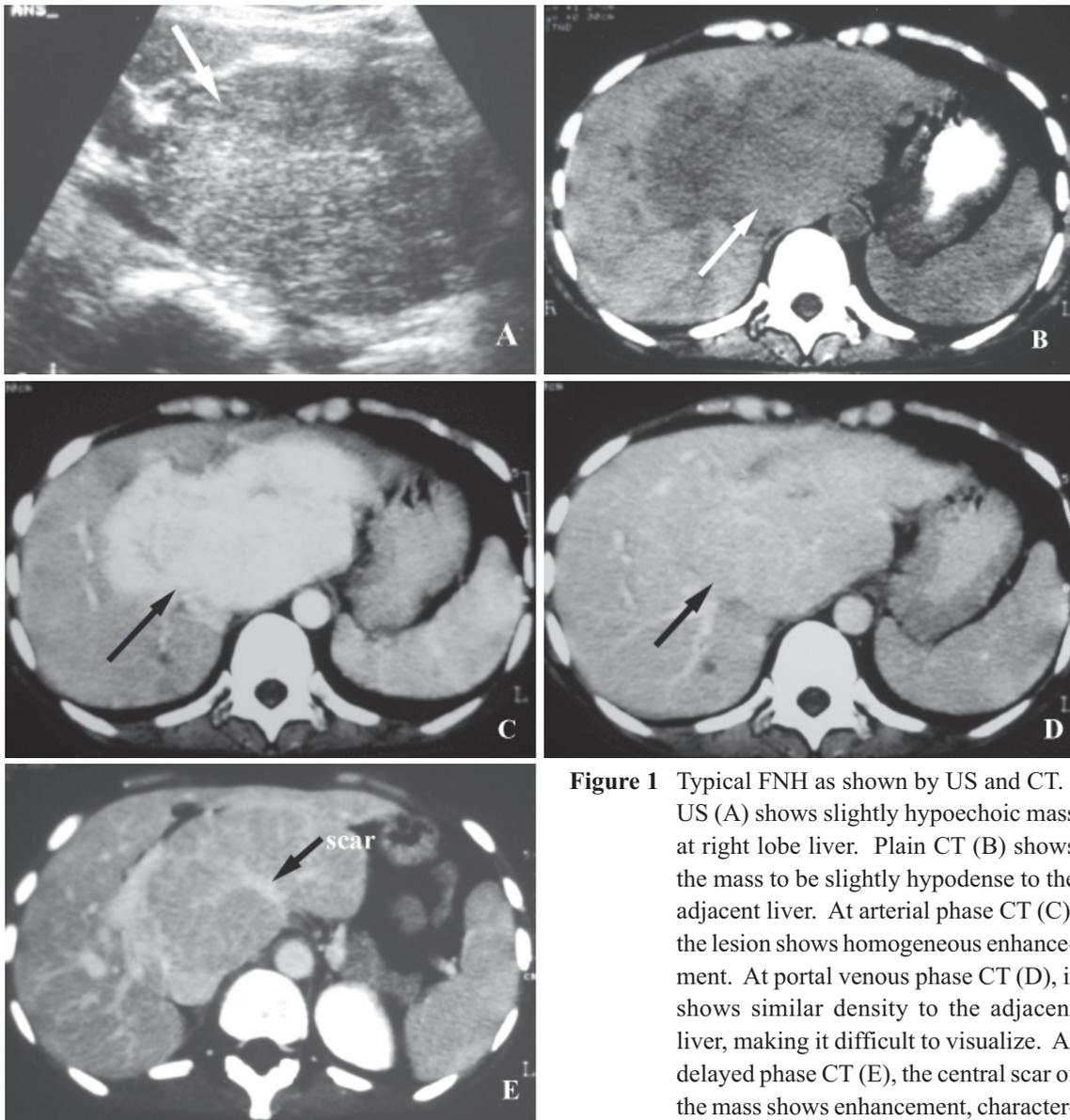
FNH is a lesion with no capsule. Pseudocapsule is usually secondary to compressed adjacent liver parenchyma or compressed adjacent vessels. Sometimes, inflammatory reaction around the lesion may cause the finding of pseudocapsule. FNH with pseudocapsule may mimic capsulated HCC, particularly if FNH has no typical central scar.

**Atypical #3: multiple FNHs** (Figure 8)

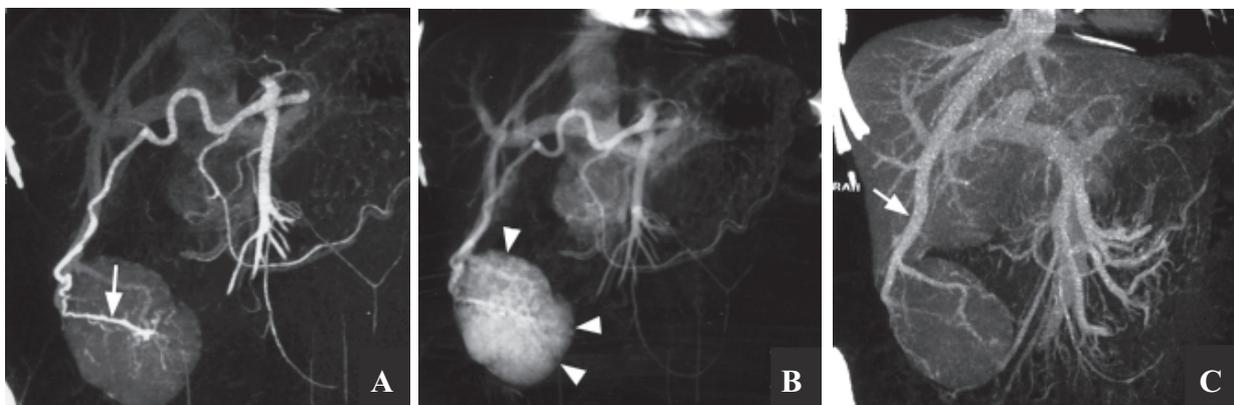
Multiple FNHs occur in about 20-25% of cases, making it difficult to distinguish from hypervascular metastasis.

**Atypical #4: telangiectatic FNH**

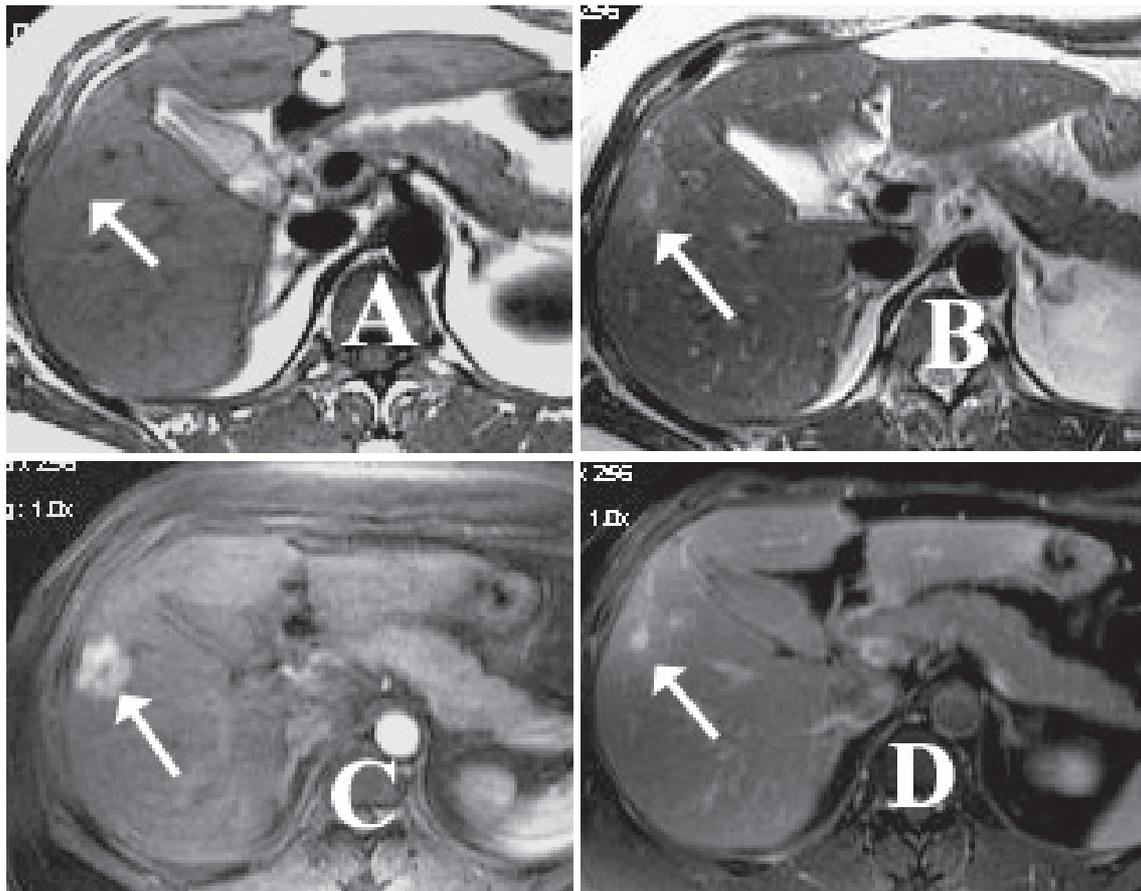
This type of FNH is uncommon, found in about 10% of cases<sup>(1)</sup>. Histopathology shows atrophic hepatic plates, dilated sinusoids, and few and short fibrous septa<sup>(9)</sup>. Imaging findings vary and include



**Figure 1** Typical FNH as shown by US and CT. US (A) shows slightly hypoechoic mass at right lobe liver. Plain CT (B) shows the mass to be slightly hypodense to the adjacent liver. At arterial phase CT (C), the lesion shows homogeneous enhancement. At portal venous phase CT (D), it shows similar density to the adjacent liver, making it difficult to visualize. At delayed phase CT (E), the central scar of the mass shows enhancement, characteristic of FNH.

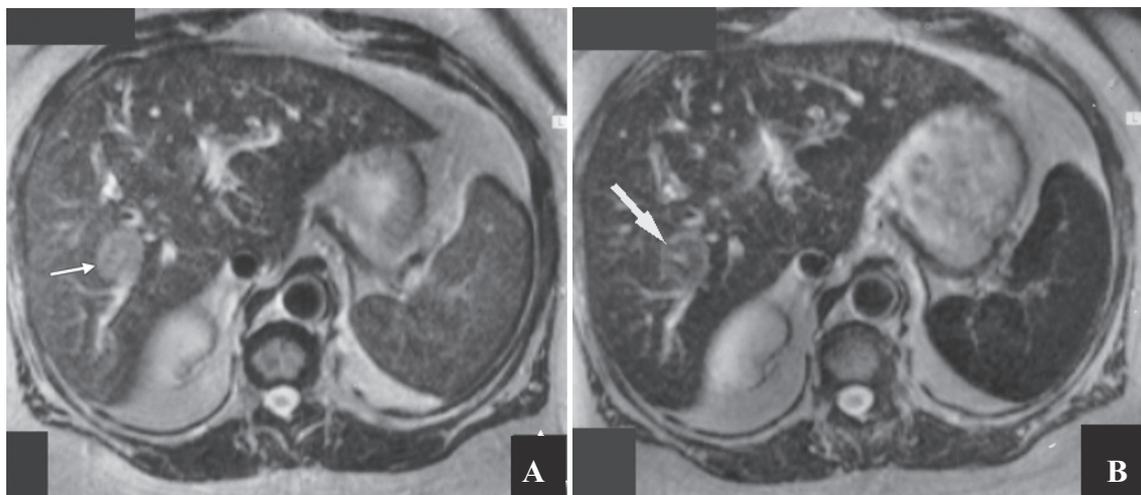


**Figure 2** Typical FNH as shown by CT angiography (CTA). At arterial phase, a large feeding artery is noted in the central scar location (A) which gives out peripheral branches showing a "spoke wheel pattern" (B). A large draining vein is noted at the venous phase (C).



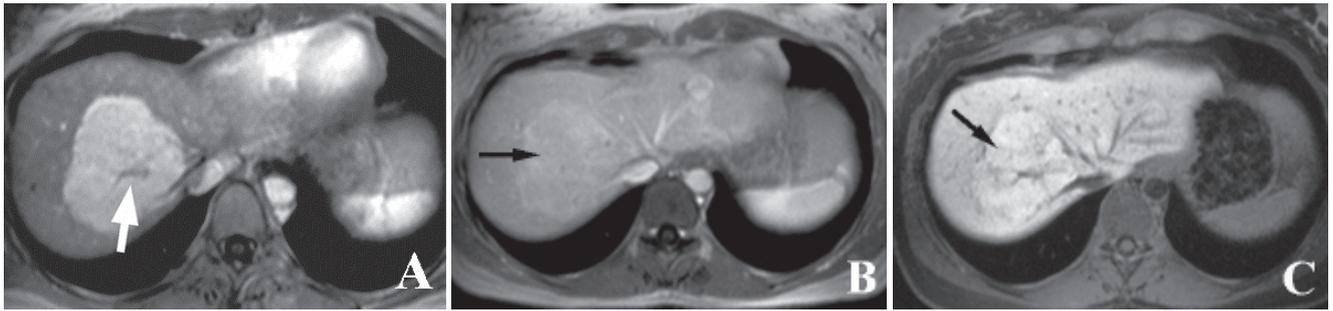
**Figure 3** Typical FNH as shown by MRI.

T1W shows the mass to be similar signal intensity (SI) to the adjacent liver (A), making it difficult to visualize. T2W shows the mass to be slightly high SI. However, the dot of central scar shows hyperintense SI, characteristic of FNH (B). At the arterial phase gadolinium, the mass shows reticular contour with homogeneous enhancement, except for central scar (C). At the delayed phase, the central scar enhances, characteristic of FNH (D).



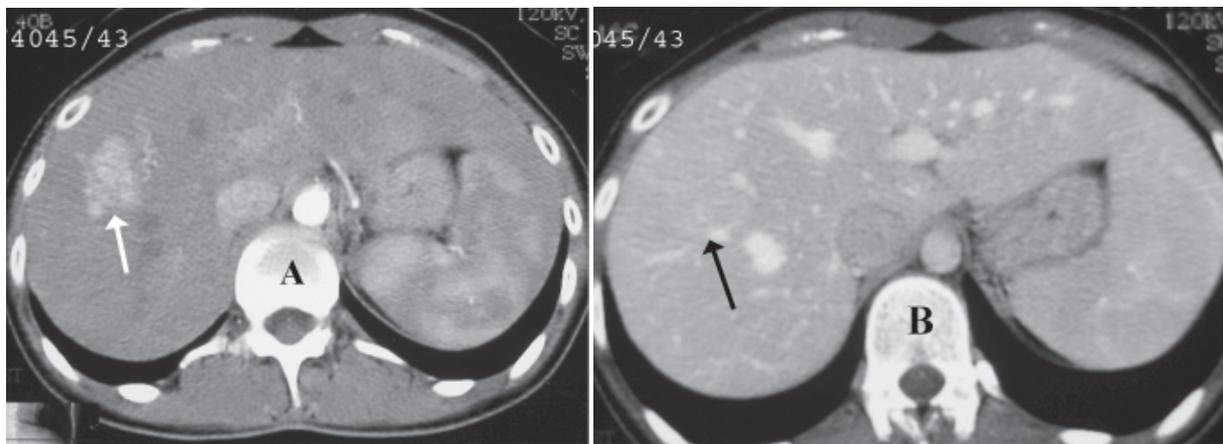
**Figure 4** Typical FNH as shown by MRI with Kupffer cells specific contrast agent.

T2W MRI, prior to contrast injection (A), shows the lesion to be of high SI. After injection of Resovist (B), there is significant signal drop within the lesion. The central scar is now easily recognized as a bright dot because of the lower background signal of the mass.



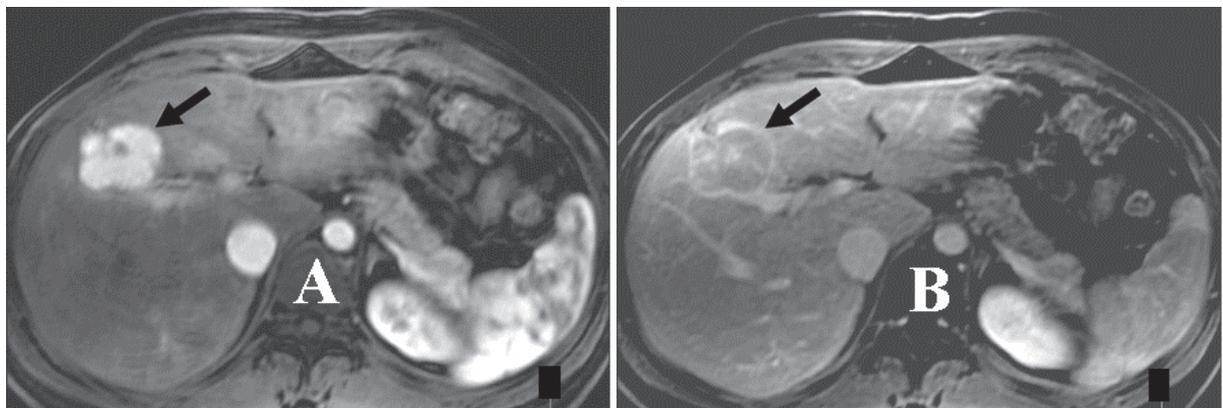
**Figure 5** Typical FNH as shown by MRI with hepatobiliary specific contrast agent.

At the arterial phase (A), there is homogeneous enhancement of the mass, except for the central scar, secondary to hypervascularity property of the mass. At the portal venous phase (B), the mass and the adjacent liver become isodense and difficult to visualize. At the delayed hepatobiliary phase, there is signal gain within the mass secondary to hepatic cells uptake of the contrast.



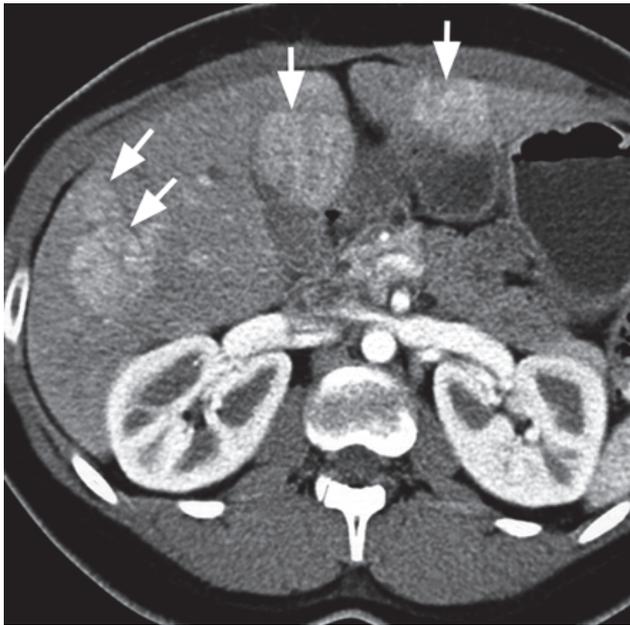
**Figure 6** Atypical #1, no central scar

Arterial phase CT (A) shows hypervascular mass with reticular border. At the portal venous phase CT (B), the mass seems to be isodense with the adjacent liver. The central scar is not visualized.



**Figure 7** Atypical #2, pseudocapsule

T1W MRI, arterial phase post gadolinium, shows lobulated contour, and homogeneous enhancement mass, except for a dot of central scar (A). At the delayed phase there is a thin rim of pseudocapsule and a central scar now enhances.



**Figure 8** Atypical #3, multiple FNHs  
Arterial phase CT shows multiple hypervascular masses, which proved to be multiple FNHs.

multiplicity, lack of central scar, heterogeneity secondary to hemorrhage/necrosis, hyperintense at T1 and T2 with persistent enhancement secondary to sinusoid dilatation<sup>(9)</sup>. In fact, both imaging findings and histopathology are difficult to distinguish telangiectatic FNHs from hepatic adenomas. Some prefer calling these lesions telangiectatic adenomas. The author never sees a real case of telangiectatic FNH, but there is a good reference article for those who are interested in this subject<sup>(9)</sup>.

### CONCLUSIONS

1. Typical FNH is easy to diagnose, particularly with MRI.

2. If typical, FNH can be left alone because it is a regenerative lesion, not a neoplastic lesion.

3. Unfortunately, typical FNH is found in less than 50% of cases.

4. Atypical FNH may mimic hepatic adenoma, HCC, or hypervascular metastasis.

5. If uncertain, biopsy, surgery or close follow-up is warranted.

### REFERENCES

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