# Clinical Implication of Hypervascular Enhancing Foci on Arterial Phase Images of Multiphase Dynamic CT in the Cirrhotic Liver

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# Clinical Implication of Hypervascular Enhancing Foci on Arterial Phase Images of Multiphase Dynamic CT in the Cirrhotic Liver

Directed by Professor Choon Sik Yoon

The Master's Thesis
Submitted to the Department of Medicine
and the Graduate School of Yonsei University
in partial fulfillment of
the requirements for the degree of Master of Medicine

Sung Ho Hwang

June 2007

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Yonsei University

June 2007

## Acknowledgements

Innumerable individuals have helped to make this manuscript possible. I would like to thank my professors and colleague in Radiology, especially Doctors Choon Sik Yoon, Jeong-Sik Yu and Yong Han Paik for their continued encouragement and support. The support of my parents is also greatly appreciated.

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# Abstract

## Clinical Implication of Hypervascular Enhancing Foci on Arterial Phase Images of Multiphase Dynamic CT in the Cirrhotic Liver

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**Objective:** Our purpose was to determine the significance of small hypervascular enhancing lesions exclusively on arterial phase images of dynamic CT in the cirrhotic liver.

**Methods:** One-hundred-sixty-nine enhancing lesions (> 5 mm and < 30 mm) on the arterial phase images of dynamic CT in 67 patients with cirrhotic liver, not distinguished from background hepatic parenchyma on equilibrium phase images without hypoattenuation density on portal phase images, were subjected to a retrospective assessment in terms of the lesion-growth in addition to the location, size and contour of the lesions depending on the final diagnoses of the individual lesions.

**Results:** Twenty-eight (17%) of the 169 enhancing lesions were hepatocellular carcinomas (HCCs). All of the 43 wedge-shaped, subcapsular lesions were benign, and 126 nodular or irregular lesions were subcapsularly (benign, n = 59;

HCC, n = 11) or centrally (benign, n = 39; HCC, n = 17) located. Significant differences were found between HCCs and benign lesions in terms of their shape (p = 0.002) and location (p = 0.041), and the positive and negative predictive values (PPV, NPV) of centrally-located lesions for diagnosing HCC were 21% and 85%, respectively. The PPV and NPV for diagnosis of HCC based on the lesion-growth were 90% and 93%, respectively.

**Conclusions:** Because of the low PPV of non-wedge-shaped, centrally-located early enhancing lesions in the diagnosis of HCC, the serial follow-up for examining lesion growth is essential to the correct diagnosis of the small arterial hypervascular lesions in the cirrhotic liver.

Key words : multiphase dynamic CT, cirrhotic liver, hepatocellular carcinoma, arterioportal shunt

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## I. Introduction

Hepatocellular carcinoma (HCC) is the most common primary hepatic malignancy worldwide, and most of the lesions develop in the cirrhotic liver from hepatitis B or C, or chronic alcohol abuse.[1] Contrast-enhanced dynamic CT plays an important role in the periodic screening or follow-up imaging of the cirrhotic liver for early detection and characterization of HCCs, and the majority of HCCs are hypervascular compared to the background parenchyma during arterial phase imaging. On portal venous or equilibrium phase images, HCCs usually show decreased attenuation relative to the liver parenchyma (also referred to as "washout" of intralesional contrast).[2-5] For the small hypervascular HCCs, however, an iso-attenuating or iso-intense appearance relative to the liver parenchyma on portal venous and equilibrium phase images of CT or MR imaging is common and frequently presents a diagnostic dilemma. The differential diagnosis for small arterial hypervascular lesions in this setting

includes an area of perfusion abnormality such as non-tumorous arterioportal shunts (APSs), HCCs, localized fibroses and unusually hypervascular benign or borderline cirrhotic nodules.[6-11]

To establish a diagnostic and therapeutic strategy for the small hypervascular lesions in the cirrhotic liver, we assumed that the CT features of these early enhancing lesions would reflect the nature and clinical behavior of the lesion. In the present study, we tried to determine the significance of small hypervascular enhancing lesions in the cirrhotic liver exclusively defined on arterial phase images of multiphase dynamic CT depending on their contour, size and location of the lesions on the initial CT in addition to the interval growth on the follow-up imaging studies.

## II. Materials and Methods

### *1. Patients and CT*

This retrospective study was approved by our Committee for Clinical Investigations and conducted according to the institutional review board rules for departmental review of records for research, and informed consent from subjects was waived. Electronic medical records of the patients who were examined in at least one session of multiphase IV contrast-enhanced dynamic CT for the assessment of focal lesions in the cirrhotic liver over a 5-year period (from April 2000 to December 2004) were searched by personnel from the clinical information center.

Multiphase contrast-enhanced dynamic CT (unenhanced, arteral, portal, and delay phases) was performed with a helical CT scanner (HiSpeed Advantage; General Electric Medical Systems, Milwaukee, WI) or a multichannel unit (Somatom Sensation 16; Siemens, Erlangen, Germany). After the intravenous administration of 150 mL of iodinated contrast agent (Ultravist 300; Schering AG, Berlin, Germany) given by an automatic injector (EnVisionCT; Medrad, Pittsburgh, PA) at 3 mL/s, arterial, portal and delay phase imaging was started after a delay of 25-30 seconds, 65-70 seconds and 150 seconds, respectively, on the helical scanner. In the multichannel unit, a 15-second delay from the time of 100 Hounsfield units of aortic enhancement was set as the starting time of arterial phase imaging using the SmartPrep technique[12] followed by portal phase imaging conducted at 30 seconds from the start point of arterial phase imaging. Three-minute delayed equilibrium phase imaging was added for the triple-phase imaging. All scans were sent to the picture archiving and communication system (PACS) for interpretation on the PACS workstations.

For patient selection, two radiologists including one attending radiologist with 12 years of experience in liver imaging and a junior resident retrieved the imaging data and conducted the PACS monitor preliminary review together. To be enrolled in the present study, a patient was required to have at least one small hypervascular enhancing lesion (larger than 5 mm and smaller than 30 mm) on arterial phase of dynamic CT examination but showed iso-attenuation that could

not be distinguished from the background cirrhotic liver on the equilibrium phase images. There should be no lesional hypoattenuation suggesting "washout" of contrast material on the portal venous phase images. For the diagnosis of HCC, the lesion was verified histologically or by a combination of clinical and radiological criteria.[13,14] These included increased tumor marker levels and interval tumor growth (≥ 2 mm in the longest dimension) during the follow-up examinations within one year. These criteria were used in addition to the sustaining nodular accumulation of iodized-oil (Lipiodol; Guerbet, Aulnay-sous-Bois, France) after transcatheter arterial chemoembolization (TACE) for the hypervascular lesions defined on the digital subtraction hepatic arteriography. For lesions larger than 1 cm, arterial hypervascularity with a subsequent washout appearance on follow-up CT or MR imaging combined with hypervascular tumor staining on hepatic arteriography was also defined as HCC according to the recent criteria stated by European Association for Study of the Liver (EASL), which were modified by a recent review.[15,16] The diagnosis of hypervascular benign lesions or pseudolesions was determined when the lesion disappeared or decreased in size based on results of serial imaging studies within one year. Lesions stable in size on follow-up dynamic CT or MR imaging study at least one year afterward were considered a pseudo- or benign lesion.[17] To rule out the possibility of a slowly growing tumor, patients with enhancing lesions that were stable or decreasing in size on serial CT scans and had less than 12 months of follow-up with dynamic CT scans were excluded. In

addition, hypervascular lesions corresponding to the typical small non-tumorous APS defined by hepatic arteriography were also regarded as a benign.[6] Thirty-four patients having 77 lesions on initial CT studies underwent hepatic arteriography during the follow-up period. Of these lesions, 28 HCCs with hypervascular tumor stainings were revealed in 20 patients and 34 typical non-tumorous APSs were detected in the tumor-free area during follow-up imaging studies in 17 patients. The remaining 15 hypervascular lesions were not defined on the hepatic arteriography. Consequently, the study population included 67 patients with 169 lesions (HCC, n = 28 in 20 patients; benign, n = 141 in 60 patients including 13 patients having HCCs coincidentally), which were marked by electronic arrows on the arterial phase images of the initial CT, and the images were saved for subsequent data analysis. The patient group included 43 men and 24 women aged from 37 to 77 years (mean age, 58.8 years).

***2. Image Analysis***

In the analysis of the imaging features of the early enhancing lesions, digitally stored image data were reviewed in conference by two different reviewers (with 5 and 14 years of experience in liver imaging, respectively) who had not participated in the initial patient selection and were blinded to the final diagnosis of the lesions. The contour (nodular, wedge-shaped, or irregular-amorphous) and the location (subcapsular or central) of the indicated

lesions were determined by a consensus of all the reviewers. The wedge-shaped lesion was defined as a subcapsular triangular lesion based on the liver capsule and centrally directed apex in the three-dimensional considerations. To be distinguished from the subcapsular lesions, the lesions completely embedded in the hepatic parenchyma were defined as central lesions regardless of the distance between the lesion and the liver surface. Two radiologists participated in the initial patient selection measured the longest dimension on a magnified view of the axial images with electronic calipers on the PACS monitor, and the size of the same lesion on follow-up imaging was also measured along the same direction for direct size comparisons of each lesion. The measurement process was repeated 14 days after the completion of the first measurement session by the same radiologists, and the mean value of two separate measurements was used for data description and subsequent analyses. The volume doubling time of the lesion was calculated as t divided by 10 (log d – log $d_0$), where the lesion diameter increased from $d_0$ to d in t days.[18] Chi-square and logistic regression tests were used to determine the differential values of the various imaging features between HCCs and pseudo- or benign lesions. A *P* value of less than 0.05 was considered statistically significant.

**III. Results**

The initial CT findings of the 169 small hypervascular enhancing lesions are summarized in Table 1. One-hundred-and-eleven (66%) of the total 169

lesions were smaller than 10 mm, and 58 (34%) were 10 mm or larger. Eighteen (64%) of 28 HCCs and 93 (66%) of 141 benign lesions were smaller than 10mm in the longest dimension on the initial CT examination. The mean size of all lesions was 9.9 mm in the longest dimension, and no significant size difference ($p = 0.44$) was detected between the pseudo- or benign lesions (9.9 mm) and HCCs (9.7 mm) on the initial CT.

One-hundred-and-eleven (66%) of all 169 lesions were subcapsularly and 58 (34%) lesions were centrally located. Eleven (10%) of the 111 subcapsular lesions and 17 (29%) of 58 intrahepatic lesions were HCCs. Considering the contour of the lesions, 83 (49%) were nodular, 43 (25%) showed irregular or amorphous appearance while the other 43 (25%) showed wedge-shape based on the liver surface. Seven (16%) of 43 lesions showing irregular-amorphous appearances and 21 (25%) of 83 nodular lesions and were HCCs (Fig. 1 and 2). All 43 wedge-shaped subcapsular lesions were benign. A significant difference was detected in the shape ($p = 0.002$) and the location ($p = 0.041$) between HCCs and benign lesions on the first dynamic CT examination. If the non-wedge shaped and intrahepatic hypervascular lesions were assumed to be HCCs, the positive and negative predictive values (PPV, NPV) for diagnosing HCC were 21% and 85%, respectively.

Besides the 10 HCCs immediately diagnosed by increased serum alpha fetoprotein levels, arterial hypervascularity in the dynamic CT and hepatic arteriography, and sustaining accumulation of iodized-oil on follow-up CT

studies after transcatheter arterial chemoembolization, 159 lesions in 59 patients had multiple comparable serial follow-up CT data (range of length of last follow-up, 12-60 months; mean, 24 months). On the final follow-up CT examinations, 20 lesions were increased in size, 8 remained stable, and 131 were decreased in size or no longer visible (Fig. 3 and 4). Eighteen (90%) of 20 lesions with interval growth during the follow-up period were HCCs and displayed a washout appearance on portal and/or delay phase images of the enlarged lesions (> 1cm in long dimension) (Fig. 1 and 2). In 18 HCCs with interval growth, the mean interval time from the initial baseline CT scan and the follow up CT examination showing subsequent washout appearance for the first time was 6.2 months (range of interval time, 2-14 months). Six of 18 HCCs displayed both interval growth and subsequent washout at the first follow-up CT examination less than 3 months after the initial baseline examination. In ten of 18 HCCs with interval growth, the subsequent washout appearance was concurrently noted at the second follow-up CT examination less than 6 months after the initial examination. Two angiographically verified APSs showing centrally located irregular hypervascularity on the initial CT were also increased in size on the final follow-up dynamic CT scan, but did not show a washout appearance on portal or delay phase images (Fig. 5). The PPV and NPV for diagnosis of HCC on the basis of lesion growth were 90% and 93%, respectively, and the mean volume doubling time of HCCs that had multiple follow-up CT examinations was 4 months (with a range of 1-14 months).

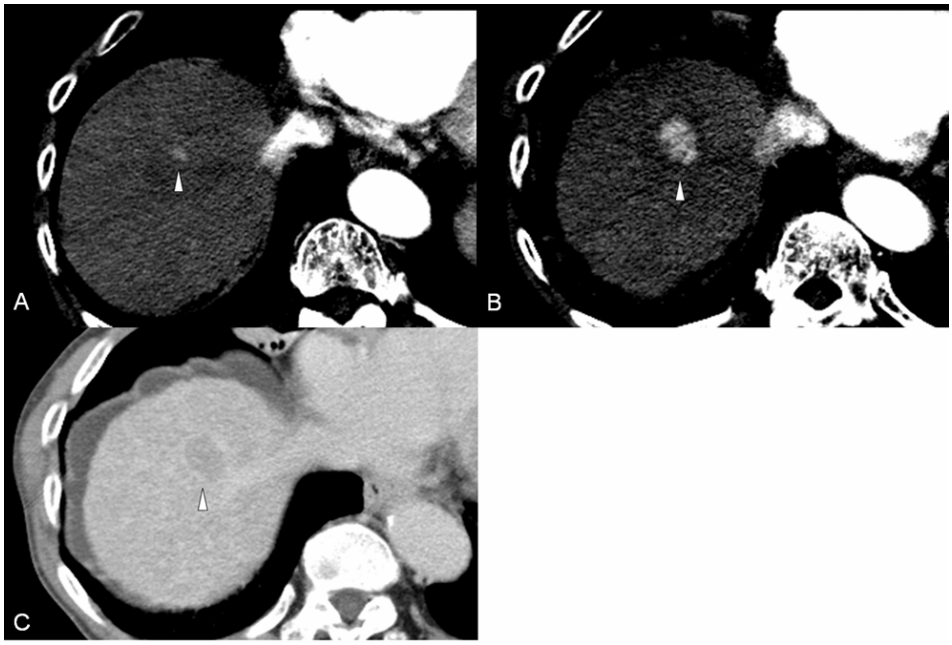


**Fig 1**.—A 71-year-old man with a hepatocellular carcinoma that increased in size during a 12-month follow-up.

**A**, Arterial phase contrast enhanced dynamic CT scan obtained at the initial CT examination shows a 0.6-cm-diameter, irregular early enhancing lesion (arrow head) in the dome of cirrhotic liver.

**B**, Arterial phase contrast enhanced dynamic CT scan obtained 6 months later shows that the lesion (arrow head) has increased in size.

**C**, Portal phase contrast enhanced dynamic CT scan obtained 6 months later shows a low-attenuation lesion (arrow head), which is indicative of hepatocellular carcinoma.

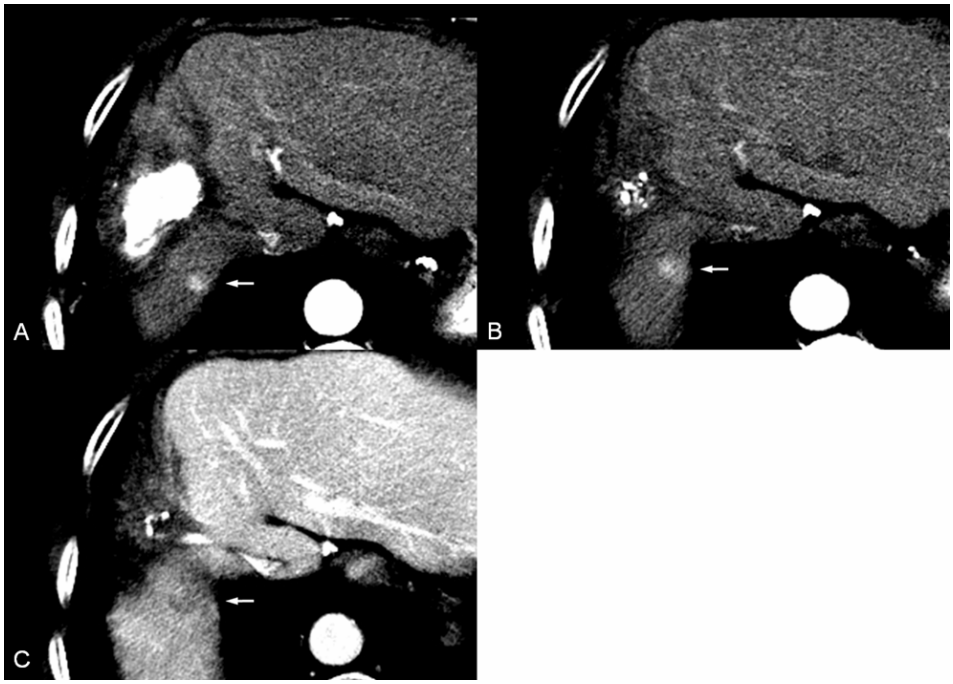


**Fig 2**.—A 55-year-old man with a hepatocellular carcinoma that increased in size during a 12-month follow-up.

**A**, Arterial phase contrast enhanced dynamic CT scan obtained at the initial CT examination shows a 0.9-cm-diameter, nodular early enhancing lesion (arrow) in the subcapsular portion of cirrhotic liver.

**B**, Arterial phase contrast enhanced dynamic CT scan obtained 12 months later shows that the lesion (arrow) has increased in size.

**C**, Portal phase contrast enhanced dynamic CT scan obtained 12 months later shows a low-attenuation lesion (arrow), which is indicative of HCC.

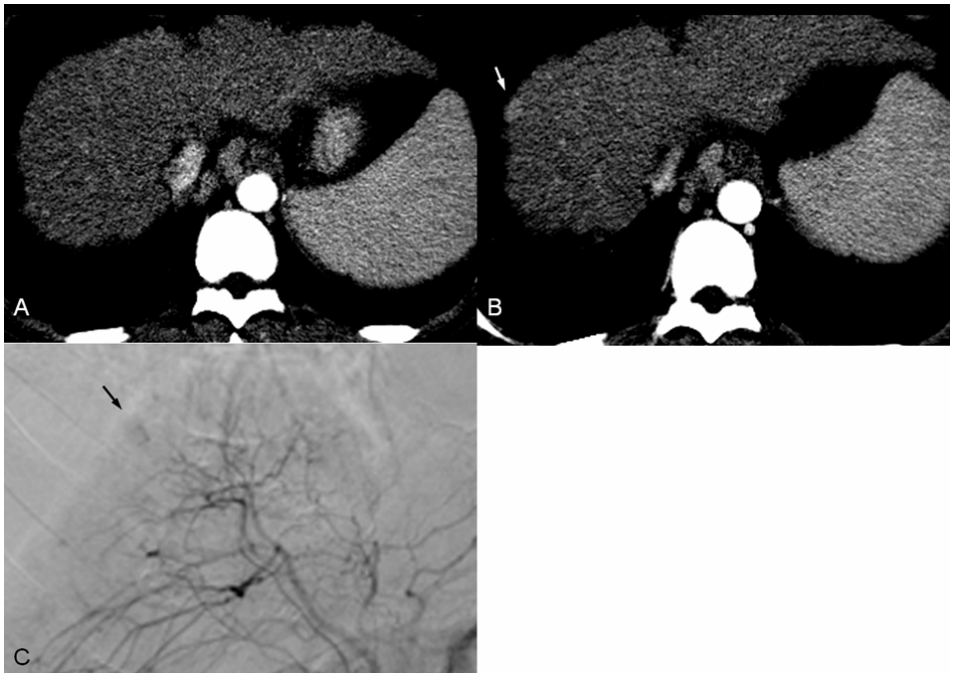


**Fig 3.**—A 40-year-old man with a non-tumorous arterioportal shunt verified angiographically.

**A,** Arterial phase contrast enhanced dynamic CT scan obtained at the initial CT examination shows a nodular lesion (arrow) in the subcapsular portion of the liver.

**B,** Hepatic arteriography obtained 2 weeks later shows branching or dotlike vascular structures (arrow) that appeared early in the arterial phase followed by focal parenchymal contrast enhancement in the tumor-free area, which is compatible with the arterioportal shunt.

**C,** A follow-up dynamic CT scan obtained 1 month later shows the lesion has disappeared.

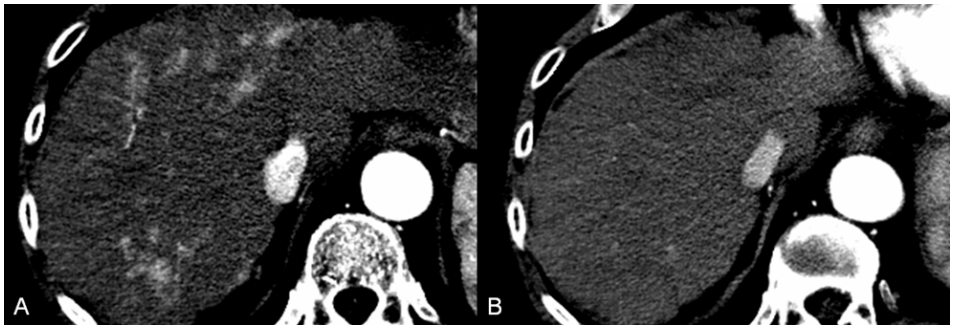


**Fig. 4.**—A 70 year-old man with small early enhancing hepatic lesions that disappeared during a 12 month follow-up.

**A,** Arterial phase contrast enhanced dynamic CT scan obtained at the initial CT examination shows irregular or nodular early enhancing lesions at the central portion of liver.

**B,** Arterial phase contrast enhanced dynamic CT scan obtained 12 months later shows that previously detected abnormalities have disappeared, which is indicative of a perfusional pseudolesion.

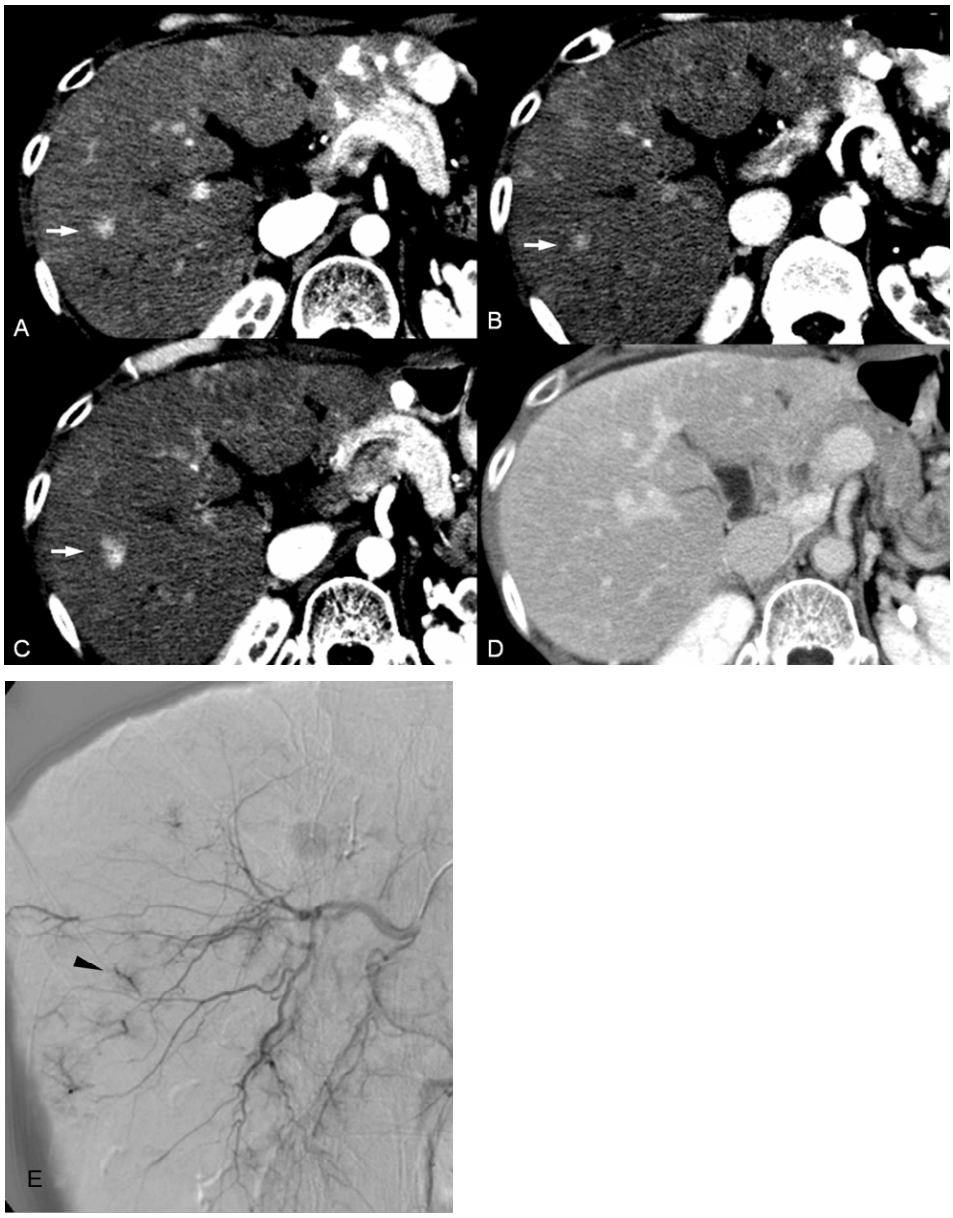


**Fig 5.**—70-year-old man with a non-tumorous arterioportal shunt showing instability of size during follow-up the period.

**A,** Arterial phase contrast enhanced dynamic CT scan obtained at the initial CT examination shows an irregular early enhancing lesion (arrow) at the central

portion of cirrhotic liver.

**B,** Arterial phase contrast enhanced dynamic CT scan obtained 3 months later shows that the lesion (arrow) has decreased in size, which is indicative of a pseudolesion.

**C,** Arterial phase contrast enhanced dynamic CT scan obtained 12 months later shows the lesion (arrow) has increased in size.

**D,** The lesions was not distinguished from background hepatic parenchyma on the equilibrium phase of dynamic CT scan obtained 12 months.

**E,** Based on hepatic arteriography obtained 12 months later, this lesion (arrow head) is diagnosed as a non-tumorous arterioportal shunt.

**Table I** **Initial CT Features of 169 Early-Enhancing Lesions in 67 Patients**

| Final Diagnosis, No.of Cases(%) | Diameter (mm) | | Shape | | | Location | |
|---|---|---|---|---|---|---|---|
| | <10 | 10-30 | Nodular | Irregular | Wedge | Central | Subcapsular |
| HCC, 28 (17) | 18 (64) | 10 (36) | 21 (75) | 7(25) | 0 | 17 (61) | 11 (39) |
| Benign, 141 (83) | 93 (66) | 48 (34) | 62 (44) | 36 (26) | 43 (30) | 41 (29) | 100 (71) |
| Total, 169 (100) | 111 (66) | 58 (34) | 83 (49) | 43 (25) | 43 (25) | 58 (34) | 111 (66) |

Note.- HCC = hepatocellular carcinoma

Numbers in the parentheses are percentages of the each diagnosis out of all 169 lesions

## IV. Discussion

The goal of screening patients with chronic liver disease is to detect HCCs while they are small, asymptomatic, and potentially curable.[19] HCC is one of the representative hypervascular tumors and imaging diagnosis is mostly dependent upon the arterial hypervascularity detected by dynamic imaging studies. Unfortunately, however, small areas of enhancement seen only on arterial phase imaging are common and rather nonspecifically demonstrated, especially in the patients with cirrhotic liver.

Liver transplantation is feasible in patients with HCCs (stage I or II disease), since it can be curative.[20] According to the American Liver Tumor Study Group Modified Tumor-Node-Metastasis staging classification, stage I tumors are those with one tumor nodule less than 2.0 cm in diameter. Stage II tumors are those with either one nodule 2-5cm in diameter or up to three nodules each less than 3cm in diameter. Therefore, if one mass less than 5cm in diameter or up to three masses each less than 3cm in diameter are present in the liver without evidence of extrahepatic metastases, transplantation can be performed.[20] The problem of small HCC detection is particularly important when the patient is in the waiting list for liver transplantation.

There have been several reports mainly regarding MR imaging of small hypervascular pseudolesions in cirrhotic liver.[10,11,21] Ito, et al[21] reported that hypervascular pseudolesions tend to show prolonged enhancement during the arterial phases compared with hypervascular HCCs on multiarterial phase contrast-enhanced dynamic MR imaging. For CT, however, the excessive radiation exposure and the use of a large amount of contrast material at CT make limitation of multiple arterial phase imaging of the whole liver. There was only one report regarding small hypervascular nodules on dynamic CT.[22] However, the previous report only dealt with nodular lesions and could make a bias for evaluation of all small hypervascualr lesions on CT by omitting the irregular or amorphous lesions as well as the wedge-shaped lesions.[22]

For the mechanism of arterial hypervascularity, locally activated transvasal or transplexal communications between the small hepatic arteries and portal veins have been suggested to make hypervascular enhancing pseudolesions. This activation depends on the intrinsic changes related to cirrhosis itself or traumatic arterioportal fistula from extrinsic factors in addition to congenital variations.[23-25] Early-enhancing lesions on arterial phase images showing a wedge-shaped appearance in the subcapsular portion commonly suggested non-tumorous APS in prior

reports.[6,7,9] Besides the typical APSs of wedge-shaped subcapsular lesions, 28 (22.2%) of 126 non-wedge-shaped foci were diagnosed as HCCs and 11 (9.9%) of 111 subcapsular early-enhancing lesions seen on CT scans were confirmed to be HCCs in the present study. However, many of the subcapsular arterial hypervascular lesions were smaller than 10 mm on the initial CT, which indicated that it would be rather difficult to recognize the wedge-shaped appearance even after carefully observing several contiguous CT images[24] Moreover, regardless of the location of the lesions, somewhat nodular or irregular lesions can develop due to the parenchymal distortion in the advanced cirrhotic liver.[7]

Several authors[9,26,27] have suggested that most subcapsular enhancing lesions in cirrhotic livers were pseudolesions rather than true HCCs. On the basis of our study results, however, we realized that besides the typical wedge-shaped subcapsular pseudolesions, the contour or the location of early-enhancing lesions on CT scans could not reliably differentiate HCCs from other pseudo- or benign lesions, or vice versa, in the cirrhotic liver. Other studies[10, 11] dealing with the small (< 20 mm) early-enhancing nodules on contrast-enhanced MR imaging have previously reported that the majority of these lesions were pseudolesions rather than hypervascular HCCs. Because of the difficulties in

confidently diagnosing small HCCs based on imaging studies, the follow-up of small arterial phase-enhancing lesions has been recommended to assess tumor growth. Follow-up MR imaging data from these studies[10, 11] of small HCCs were similar to the data in the present study using CT, which is more generally used in patients undergoing surveillance for cirrhosis. On the follow-up CT, most of the HCCs showed a washout appearance of relative hypoattenuation on the portal or delayed phase images.

Due to the small number of hypervascular pseudolesions that finally enlarged in size, the growth of the lesion was helpful in distinguishing the small HCCs from the pseudolesions in the present study (PPV, 90%; NPV, 93%). Regarding the nature of the reciprocal communications between the artery and portal vein in APSs, however, the extent of the arterial enhancement of the hepatic parenchyma is variable depending on the arterial perfusion pressure at the timing of image acquisition.[7] This hemodynamic instability could cause the size variations of the APS-induced hypervascular foci observed on the serial follow-up dynamic imaging studies. From this viewpoint, the simple enlargement of hypervascular lesions on follow-up imaging cannot be enough to characterize HCC in the cirrhotic liver.

For the lesions that showed no hypervascular staining or shunt vessels on hepatic arteriography or those that immediately disappeared on the follow-up CT, there would still be a possibility of arterial hypervascularity on the initial CT studies. Somewhat localized small hepatic venous or portal venous thrombosis could have developed due to the sluggish flow from portal hypertension. The temporarily occluded hepatic vein could increase the sinusoidal pressure and reverse the pressure gradient between the sinusoidal and portal veins resulting in a kind of transsinusoidal APS. In such cases, the portal vein branches cannot be well demonstrated during the early phase of hepatic arteriography. In the thrombotic cases of small portal venules, the compensatory increase of arterial flow can also be visible as a hypervascular lesion on the dynamic CT, but not delineated as a direct APS on the hepatic arteriography. The small temporary benign venous thromboses might be easily resolved and tend to be noted inconstantly on the follow-up CT. Another possible explanation is the theory of small subclinical inflammation suggested by Kamura, et al[28], in which small portal or hepatic venules could be obliterated temporarily due to inflammatory cell infiltrations around the pyogenic abscess[29] or along the portal tract.[30] The inflammation-induced increase in arterial

hypervascularity would be the only finding indicative of the inflammatory lesions which would then easily disappear after the inflammation was resolved.[30, 31]

This study had several limitations. Due to the retrospective nature of this study, pathologic proofs of HCC were not available and angiographic evidence of pseudo- or benign lesions were insufficient in many of the cases. In the evaluation of small hypervascular lesions exclusively seen on arterial phase images, we routinely recommend follow-up CT or MR imaging rather than an immediate additional confirmative study. After verification of the growth of the lesions and the washout appearance on portal or equilibrium phase images, hepatic arteriography is generally performed for additional imaging information and TACE. A percutaneous biopsy is reserved for the gradually enlarged and hypovascular lesions in the cirrhotic liver. Although the majority of pseudo- or benign lesions would create a non-tumorous condition verified by means of follow-up imaging, the possibility of an extremely slow-growing small hypervascular tumor still exists. However, most of the non-HCC lesions were decreased in size or even disappeared, and only a limited number of stable lesions were detected during the follow-up imaging studies. We believe that the limited possibility of the extremely slowly growing

hypervascular HCC could not change our results.

## V. Conclusion

In conclusion, although the majority of hypervascular lesions exclusively defined on the arterial phase imaging of dynamic CT were pseudo- or benign lesions, due to the limited value of the PPV of centrally located hypervascular lesions in the diagnosis of HCC, we could not reliably differentiate HCCs from benign conditions by means of the initial CT findings of the shape or locations except for the typical wedge-shaped subcapsular pseudolesions. Interval lesion-growth is highly predictive of HCC, however, due to the instability of the APS-induced pseudolesions, a temporary enlargement in the size of a hypervascular lesion itself would have a limited value to confirm the diagnosis of HCC. We suggest that lesion growth should be monitored in conjunction with the washout appearance on portal or equilibrium phase images on the follow-up CT studies to confirm the imaging diagnosis of HCC. In the present study, the mean volume doubling time of small hypervascular HCCs that initially showed no washout appearance on portal or delayed phase imaging was 4 months, which would be a suitable interval to arrive at the correct diagnosis and management of

subcentimeter HCCs. When small hepatic arterial phase-enhancing lesions on dynamic CT scan are observed, a serial, close follow-up of the lesions is still essential for correct diagnosis.

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## < Abstract in Korean >

간경변증 환자의 다중위상 전산화 단층 촬영시 동맥기에서
과혈관성을 보이는 작은 병변들의 임상적 의의

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**목적:** 간경변증 환자를 대상으로 시행한 역동적 전산화 단층 촬영시 동맥기에서만 과혈관성을 보이는 작은 병변들의 악성빈도와 관련된 영상소견의 의의를 알아보았다.

**방법:** 총 67명의 간경변증 환자에서 시행한 역동적 전산화 단층 촬영에서 동맥기 과혈관성을 보이는 작은 병변들 (직경 > 5mm 그리고 < 30mm) 중 문맥기와 평형기에 주변 간조직과의 감쇄밀도차이를 보이지 않는 169개를 연구대상으로 하였다. 이들 병변들을 최종 진단에 따라 구분하고 각각의 위치, 경계, 그리고 크기변화를 후향적으로 비교분석 하였다.

**결과:** 총 169개의 병변들 중 28개(17%)가 간세포암이었고 나머지는 양성 혹은 거짓 병변들 이었다. 간실질 중심부에 위치한 병변들

(n=58) 중 악성은 17개(29%)였고 소결절성 또는 불규칙한 모양의 병변들 (n=126) 에서 악성은 28개(22%)였다. 피막하에 위치한 쐐기모양의 병변들 (n=43)은 모두 양성이었다. 이런 간세포암과 양성 병변들 사이에는 병변의 모양 (p=0.002) 및 위치 (p=0.041)에서 유의한 차이가 있었다. 하지만 간실질 중심부에 위치한 비쐐기모양의 병변을 간세포암으로 간주하였을 때 양성 예측도는 21%, 음성 예측도는 85%에 그쳤다. 병변의 크기 증가 여부를 기준으로 간세포암을 진단하였을 때 양성 예측도는 90%, 음성 예측도는 98%였다.

**결론:** 간경변증 환자의 역동적 전산화 단층촬영시 동맥기에 과혈관성을 보이는 작은 병변들 중 간실질 중심부에 위치한 결절성 또는 불규칙한 모양을 보이는 병변들을 악성으로 가정시 악성 예측도는 높지 않다. 추후 연속적인 추적 검사를 통한 병변의 크기증가 확인이 보다 정확한 진단을 위해 중요하다.

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핵심되는 말 : 다중위상 역동적 전산화 단층촬영, 간경변증, 간세포암, 동맥문맥단락