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ORIGINAL ARTICLE

Growth of Asymptomatic Intracranial Fusiform Aneurysms

Incidence and Risk Factors

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Abstract

Purpose Growth of intracranial fusiform aneurysms (IFA) may become clinically problematic through a mass effect or rupture. We investigated the growth rate and factors contributing to growth in asymptomatic untreated IFA.

Method As a retrospective review, we assessed patients diagnosed with asymptomatic IFA between August 2000 and September 2014, all untreated. No acute or symptomatic dissecting lesions were considered. Clinical and serial angiographic follow-up data were analyzed, defining growth as expansion > 2 mm in one or more dimensions. A binary logistic regression model and Kaplan-Meier method were applied for statistical analysis.

Results The mean follow-up in the 82 eligible patients was 47.7 months (range 12–190 months). Among them, 7 aneurysms (8.5%, 2.1% per aneurysm year) demonstrated growth (in any dimension). In univariate analysis, height and multiplicity of aneurysms emerged as significant factors in terms of growth. Height remained an independent risk factor in the binary logistic regression model, with receiver operating curves indicating a threshold of 6.9 mm initial height in determining IFA growth (area under the curve 0.804). Of the patients six (except one who underwent endovascular treatment) were observed during continued follow-up monitoring. All six lesions were stable in serial imaging tests, without further detectable growth or rupture (mean 33 months).

Conclusion Most (91.5%) of the asymptomatic and untreated IFAs studied proved to be stable, with no continued growth; however, because aneurysm height proved to be independently predictive of growth (lesions > 6.9 mm being at risk), periodic imaging is required in those left untreated. Growing but still asymptomatic aneurysms call for the utmost caution and care in decision-making.

Keywords Aneurysm · Fusiform · Unruptured · Growth · Follow-up

Introduction

Progress made in non-invasive screening modalities has aided in the detection of asymptomatic unruptured intracranial aneurysms (UIAs). Improved operative and endovascular techniques have also contributed to more active treatment of UIAs. Unruptured aneurysms affect 2–8% of the general population [1–3] but because the incidence of subarachnoid hemorrhage is only 10–30 per 100,000 people, the treatment must be selective [4]. A crucial factor in the decision to treat is growth. Findings of one meta-analysis indicated that stable aneurysms have a 0.1% rupture rate, as opposed to a rate > 3% with demonstrable growth [5]. A growing aneurysm then becomes an object of active treatment [6, 7].

Growth of UIAs ranges from 2–5% per year of follow-up [8–12]. It also appears that the growth rate in non-saccular aneurysms exceeds that of saccular aneurysms [5]. Various reports on non-saccular aneurysms cite a growth rate of 46% for 8.5 years follow-up, with 52% mortality [13–17]; however, even in studies addressing non-saccular aneurysms, inclusion and exclusion criteria vary widely, so it is difficult to generalize results [17]. In addition, the treatment of non-saccular (vs. saccular) aneurysms differs

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technically, given the distinctive angiographic shapes and configurations of fusiform lesions [18] and morbidity and mortality rates are high. It is therefore beneficial to accurately discern their natural course and properly define therapeutic indications [19, 20].

The clinical course of growing yet asymptomatic and untreated intracranial fusiform aneurysms (IFAs) is sparsely documented. As such, this study was conducted to analyze growth rates and factors contributing to growth in IFAs, which were expected to differ from saccular aneurysms and from symptomatic fusiform lesions in terms of natural course.

Material and Methods

Study Population

The cohort was drawn from a consecutively recorded database of clinical patients, conducting an extensive electronic search of corresponding imaging studies and medical records for a retrospective review.

An IFA was defined as fusiform arterial ectasia at a nonbranching site of an intracranial cerebral artery (ICA). All IFAs in this study were identified through prior diagnostics using conventional angiography, magnetic resonance angiography (MRA) or computed tomography angiography (CTA). All participants had unruptured lesions, with no related clinical symptoms or signs and the study endpoint being growth of asymptomatic, untreated, and unruptured IFAs during follow-up monitoring. Patients with acute dissecting aneurysms (including ruptured lesions), symptomatic fusiform aneurysms and histories of mycotic, traumatic, vasculitis or previously treated aneurysms were excluded from the study. Symptomatic presentation was considered the seeking of medical attention as a direct consequence of an aneurysm, for example, mass effect, stroke and transient ischemic attack in a lesion's vascular distribution, or acute pain.

Between August 2000 and September 2014, a total of 115 patients with fusiform aneurysms were observed without any specific treatment because all the aneurysms were asymptomatic and unruptured lesions; however, 22 patients lost to follow-up or lacking the required 12 months of monitoring were excluded, as were 11 patients with acute symptomatic dissection. Therefore, 82 patients who were observed and followed at the neurovascular outpatient clinic of our facility for >12 months, finally qualified for the study. During the same period, 94 patients with non-saccular aneurysms were treated, 2 by direct open surgery and 92 by endovascular intervention. Stent-assisted coiling was the primary endovascular treatment ($n=61$) but coil trapping was performed in 26 and stenting alone was used in 4 patients. There was one instance of using flow diverter. Acute dissections occurred in 71 patients, including various hemorrhagic presentations, 4 large fusiform aneurysms, and 8 secondary interventions in previously treated lesions. The remaining 11 aneurysms were asymptomatic fusiform lesions with specific clinical imperatives for treatment (young age, large size, severe anxiety, doctor/patient preference, etc.). An algorithm for patient selection is shown in Fig. 1.

Therapeutic alternatives, including surgical clipping, endovascular coiling or observation and follow-up measures were formulated by both neurological, neurosurgical and neurointerventional teams in a multidisciplinary decision-making process and then discussed with all patients and family after careful consultation. This study complied with the principles outlined in the Declaration of Helsinki and was approved by our institutional review board.

Clinical and Radiographic Data

Baseline clinical characteristics, including age at diagnosis, gender, follow-up period, and modes of presentation, were extracted from electronic records. We also searched available records for risk factors predisposing to IFAs, such as hypertension, diabetes mellitus, dyslipidemia, smoking, other cerebral vascular lesions, and known connective tis-

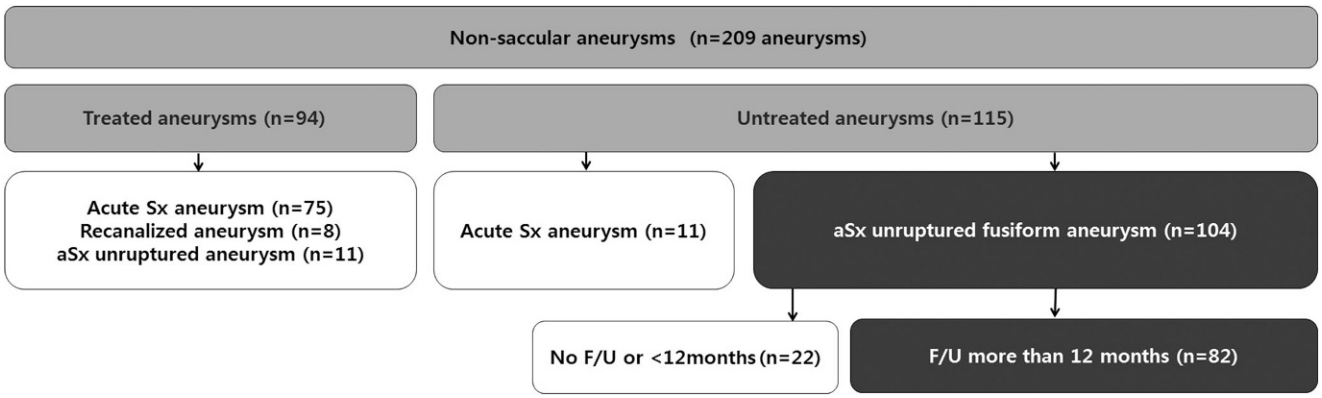

Fig. 1 Algorithm for the patient population selected in the study (*Sx* symptomatic, *F/U* follow-up)

sue diseases. From imaging at first presentation to serial follow-up studies, aneurysm size (maximum length and height), location, and multiplicity (saccular or non-saccular) of lesions were also investigated. All aneurysms were categorized as atherosclerosis-related or non-atherosclerotic lesions, according to the classification of Sacho et al. [21].

We also analyzed the radiological follow-up for each aneurysm, aimed at patients undergoing two or more follow-up imaging examinations more than 1 year apart. Each aneurysm was measured in terms of height (diameter at widest point by arteriography or cross-sectional imaging) and length (extent of involved vessel segment; [16, 20]), defining growth as >2 mm change in diameter at more than one point [13]. All follow-up diagnostics were reviewed at random. The imaging diagnostics were independently assessed by two experienced neurointerventionists, blinded to pertinent clinical and radiological information: MRA (or CTA) and conventional angiography. In the event of disagreement, a consensus was established by a third interventional neuroradiologist.

We also studied all clinical developments, specifically checking for evidence of subarachnoid hemorrhage, mass effect or related stroke and transient ischemic attacks. Any patient undergoing procedures or surgery during follow-up of IFAs was included in such investigations.

Statistical Analysis

Descriptive statistics were used to summarize patient demographics, clinical risk factors and comorbidities, and characteristics of IFAs (location, size, multiplicity). Continuous variables were expressed as mean ± standard deviation (SD) and range. Comparison of growth and non-growth subsets was achieved using Fisher's exact test, χ^2-test or t-test. To assess correlations between clinical factors and aneurysm growth, binary logistic regression was applied. Receiver operating characteristic (ROC) curve analysis was also invoked to determine the cut-off point for IFA height and area under the curve (AUC). Kaplan-Meier survival estimates were then plotted, based on heights of aneurysms in growth and non-growth subsets. All tests were 2-tailed, relying on standard software (SPSS Statistics v20.0 for Windows; IBM, Chicago, IL, USA) for calculations and setting significance at $p < 0.05$.

Results

Study Population

For all aneurysms discovery was prompted by nonspecific neurological complaints, such as mild headaches, dizziness or at medical check-ups. The mean age at time of aneurysm detection in the 82 patients studied (male 35, female 47) was 56.7 years (range 32–78 years). Of the aneurysms 31 (37.8%) involved the anterior circulation (see supplementary table) and in addition to IFAs, second aneurysms (all saccular) were observed in 19 patients (23.2%). In 19 patients (23.2%), the IFAs were classifiable as atherosclerotic. Clinical and imaging follow-up studies were maintained for a mean of 47.7 months (range 12–190 months; Table 1).

Follow-up of Growing Aneurysms

Using our criteria 7 aneurysms (8.5%, 2.1% per aneurysm year) displayed features of growth. The mean age in this subset (male 4, female 3) was 53.1 years. The mean interval from initial discovery to detection of growth was 50.1 months (range 5–127 months). Of the lesions two involved anterior circulation, the others were situated in posterior vessels. The mean size of these IFAs at time of discovery was 14.5 × 10.8 mm. At the time of detection the

Table 1 Univariate analysis of patients with fusiform intracranial aneurysms, stratified by presence or absence of growth

Variables	Total ($n = 82$)	Stable ($n = 75$)	Growth ($n = 7$)	p-value
Female	47 (57.3%)	44 (58.7)	3 (42.9)	0.453
Age, years (mean)	56.7 ± 11.7	57.0 ± 11.8	53.1 ± 11.3	0.408
Hypertension	51 (62.2%)	48 (64.0)	3 (42.9)	0.417
Diabetes	5 (6.1%)	5 (6.7)	0 (0)	0.644
Smoking	11 (13.4%)	10 (13.3)	1 (14.3)	1.000
Total F/U period (months)	47.7 ± 34.3	44.3 ± 30.1	83.7 ± 55.1	0.003
Period of growth from detection (months)	–	–	50.1 ± 42.6	–
Anterior location	31 (37.8%)	29 (38.7)	2 (28.6)	0.705
Multiplicity	19 (23.2%)	15 (20.0)	4 (57.1)	0.026
Atherosclerotic	19 (23.2%)	17 (22.7%)	2 (28.6%)	0.723
Length (mm; mean)	12.1 ± 6.4	11.8 ± 6.2	14.5 ± 8.5	0.390
Height (mm); mean)	6.0 ± 2.9	5.54 ± 1.8	10.8 ± 6.9	<0.0001

Data are expressed as mean ± SD or number (percentage), *F/U* follow-up

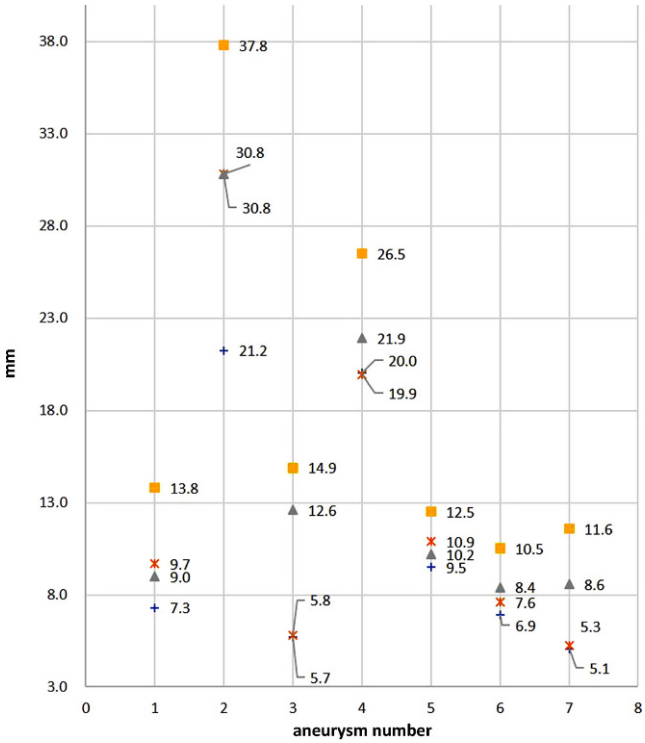

Fig. 2 Changes in height and length of growing fusiform intracranial aneurysms (*triangle* initial length, *square* length after growth, *cross* initial height, *star* height after growth)

mean size was 17.8 × 13.0 mm. The length increased by an average of 28.3% (up to 53.3%) and height by an average of 15.4% (up to 45.0%; Fig. 2) and 4 patients had additional aneurysms (≥ a total of 2 aneurysms). No patient suffered a subarachnoid hemorrhage during follow-up, but one patient suffered cerebral infarction, without permanent deficits.

In one female patient with an enlarging IFA (cranial nerve palsy due to mass effect) of the right distal ICA (9.0 × 7.3 mm → 13.8 × 9.7 mm), endovascular stent-assisted coiling was performed; however, she suffered delayed infarction 2 months later. In the other six patients, watchful observation was elected (without treatment), given the continued lack of symptoms. All lesions were stable, without any symptoms developing or further growth for the duration of follow-up (mean 33 months; maximum 63 months), once enlargement was detected.

Among the remaining 75 patients without growth, 3 patients suffered cerebral infarction developed at the IFA-correlated territory, but they achieved complete recovery.

Analysis of Risk Factors in Growing Aneurysms

Growth and non-growth patient subsets were compared, examining factors potentially implicated in aneurysm growth, such as gender, age, hypertension, diabetes, regular smoking, extent of follow-up, and lesion characteristics (location, multiplicity, length, and height; Table 1). In the growth (vs non-growth) subset, size of IFAs at time of detection was comparatively larger (14.5 × 10.8 mm vs. 11.8 × 5.5 mm), and height was significantly greater ($p < 0.001$). Univariate analysis indicated a significant association between multiplicity of aneurysms and growth (57.1% vs. 20.0%; $p = 0.026$). Based on established criteria, 19 atherosclerotic IFAs were identified. Among them, only 2 aneurysms showed growth, but atherosclerosis per se was not associated with growth of aneurysms.

Table 2 Binary logistic regression analysis of factors predicting growth in fusiform intracranial aneurysms

Variables	Odds ratio	95% Confidence interval	*p*-value
Multiplicity	2.365	0.341–16.395	0.384
Height	1.471	1.021–2.120	0.038

Binary logistic regression analysis was performed to determine if multiplicity or height were important independent risk factors for growth in asymptomatic IFAs (Table 2); however, height alone showed statistical significance (OR = 1.471, 95% CI 1.021–2.120; $p = 0.038$). In the receiver operating characteristic curve generated, the threshold for height as a determinant of growth in IFAs was 6.9 mm (AUC = 0.804; $p = 0.008$; Fig. 3).

Kaplan-Meier survival curves for growing IFAs at baseline height of 6.9 mm are presented in Fig. 4. The 60-month estimated cumulative survival (without growth) was 90%, with height > 6.9 mm as risk factor (size ≤ 6.9 mm, 95.7%; log rank $p = 0.044$).

Discussion

Non-saccular intracranial aneurysms are less common than saccular aneurysms [22]. Although there are many studies on the natural history of saccular aneurysms, studies on non-saccular aneurysms have lacked study population uniformity, and research methodologies have been diverse [17]. Unlike saccular lesions, non-saccular aneurysms produce symptoms of ischemic stroke or exert mass effects rather than hemorrhaging [13, 17, 21, 23]. Non-saccular aneurysms are often subcategorized as dissecting or non-dissecting lesions, but these terms overlap broadly with what is considered classical dissection or are variably defined in each publication [14–17, 21, 23, 24]. Even Brinjikji et al. viewed saccular aneurysms with lobulated or daughter sac features as non-saccular aneurysms [5].

Based on histopathologic findings (i.e., disruption of internal elastic lamina and secondary intimal reaction), Mizutani et al. assigned aneurysms originating from arterial trunks (not branching zones) to classical dissection, segmental ectasia, dolichoectatic dissection, and saccular

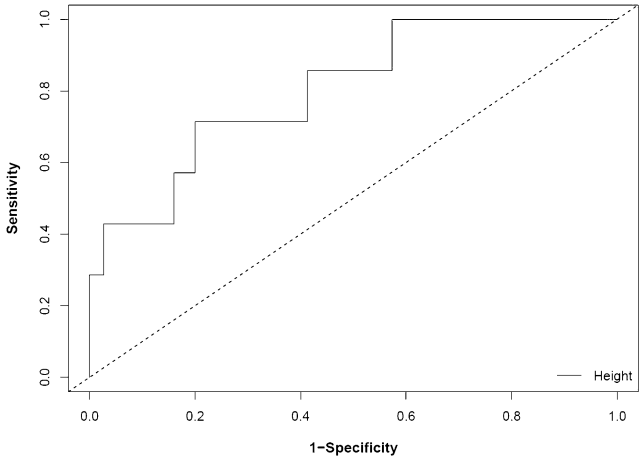

Fig. 3 Receiver operating characteristic curve for height as a determinant of growth in fusiform intracranial aneurysms (cut-off point for height, 6.89; area under the curve, 0.804 [95% confidence interval 0.384–0.808]; sensitivity 80.0%; specificity 71.4%; $p = 0.008$)

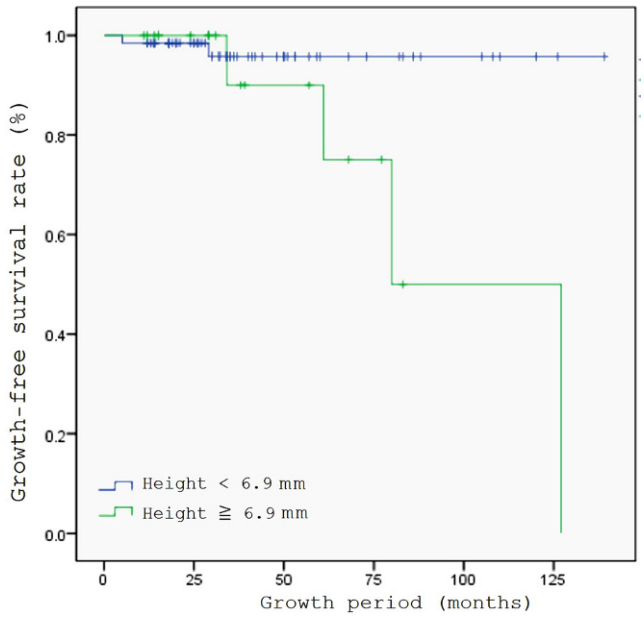

Fig. 4 Kaplan-Meier estimate of cumulative survival in patients with fusiform intracranial aneurysms (height > 6.9 mm as risk factor)

subcategories [25]. Alternatively, Flemming et al. separated non-saccular vertebrobasilar aneurysms without clearly definable necks into fusiform, dolichoectatic, and transitional aneurysms, based on the degree of dilation and arterial segment involved [14]. Intramural hematoma is clearly linked to the growth of fusiform aneurysms, having shown an association with aneurysm size [23]. Although we could not offer pathologic substantiation of our findings, it is still reasonable to presume a dissecting etiology, given the clinical presentations alone [17]. It is sometimes difficult to distinguish between chronic fusiform aneurysms and dolichoectasia, even with clinical or imaging support [23], but we made every effort to exclude dolichoectasia, as detailed in the classification of Flemming et al.[14].

In one particular review, it is noted that ~30% of patients with non-saccular vertebrobasilar aneurysms lack symptoms and are discovered incidentally [17]. The overall mortality of all non-saccular vertebrobasilar aneurysms reported was 40% at 7 years, whereas in patients without symptoms, overall mortality was 5.0% at 11.8 years [17]. Other studies likewise documented that the prognosis of intradural non-saccular aneurysms without symptoms is relatively favorable, compared with symptomatic aneurysms [14, 16, 21, 24, 26]; however, studies addressing non-saccular aneurysms vary widely in terms of eligibility criteria, making it difficult to generalize results [17]. Hence, the natural course of asymptomatic IFAs, especially as a function of growth, is still uncertain.

Our study of asymptomatic IFAs showed a growth rate of 2.1% per aneurysm-year. This result is comparable to the 2–5% per aneurysm-year reported for saccular aneurysms in earlier publications [8–12]; however, vertebrobasilar IFAs exerting mass effect have been associated with a hazard ratio of 9.4, compared with asymptomatic counterparts [12]. Sacho et al. also attached a significantly higher odds ratio (16.0) for growth to unruptured IFA symptomatic at clinical presentation, as opposed those without symptoms [21]. Although a poor outcome resulted in one of our patients receiving endovascular treatment for an enlarging IFA, the other 6 growing lesions proved to be stable when followed for another 33 months (max 63 months) after first detecting growth. Recently, a flow diverter has been applied even in asymptomatic IFA, but there is controversy over its usage in terms of safety. Bhogal et al. [27] reported a mortality of 11.1% in patients with asymptomatic posterior circulation IFA that was treated with a flow diverter. Thus, if an asymptomatic IFA remains asymptomatic during follow-up, the approach to patient management must weigh up the risk of a procedure against an otherwise naturally benign course.

It is acknowledged that large aneurysms are prone to growth, whether they are saccular or fusiform [16, 21, 23, 28]. Both fusiform and saccular aneurysms have shown increased risk of growth at sizes > 7 mm [21]. Our findings similarly indicated the significant impact of height, revealing greater risk in lesions > 6.9 mm at time of detection (binary multivariable analysis). The fundamental principle here may be that growth of aneurysms is a manifestation reflecting mechanical instability of the vessel wall [21].

Recently, there have been reports that the prognosis of atherosclerotic IFAs is worse [15, 17, 21], perhaps due to symptoms or large size at time of detection [13, 21]. Sacho et al. have noted that in incidentally discovered atherosclerotic fusiform aneurysms, 50% (3/6) showed growth on follow-up, compared with 37.5% in non-atherosclerotic lesions [21]. Despite use of the criteria of Sacho et al.

atherosclerosis and growth of asymptomatic IFAs (22.7% vs. 28.6%) were unrelated in our study. This may be explained by our small patient population or by differences in age or comorbidities of the patients included.

This study has several distinct limitations, one being that the data were collected retrospectively at a single institute. Thus, there is potential for bias in the choice of conservative treatment; however, if an aneurysm was large at the time of detection, and treatment (without follow-up) was preferred, the patient was excluded from participation. On the other hand, such restrictions may well represent the clinical course of IFAs in actual practice. Another limitation is the lack of histopathologic corroboration and given the retrospective design, a unified imaging protocol could not be implemented for patient follow-up (although the same modality was advised). Inconsistent imaging intervals among patients also left gaps in the analysis. Further prospective studies are needed, providing more conclusive evidence on such lesions through broader sampling of asymptomatic IFAs.

Conclusion

In our patient population, most (91.5%) asymptomatic and untreated IFAs remained stable, showing no further growth. Even so, the risk of complications with continued growth during follow-up was minimal. Because height proved to be an independent predictive of growth in FIAs (lesions > 6.9 mm being more susceptible to growth), serial imaging of larger untreated aneurysms is essential. Ultimately, patient management decisions must be made cautiously in this setting.

Compliance with ethical guidelines

Conflict of interest J. Moon, Y.D. Cho, D.H. Yoo, J. Lee, H.-S. Kang, W.-S. Cho, J.E. Kim, L. Zhang and M.H. Han declare that they have no competing interests.

Ethical standards This study complied with the principles outlined in the Declaration of Helsinki and was approved by our institutional review board (Seoul National University Hospital).

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