제4호 Cardiomyopathy Education and Research Center 2022

심근증교육연구센터 소식

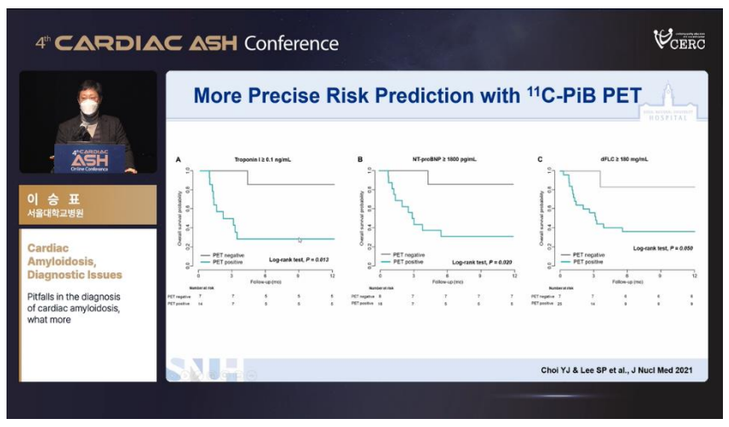
사단법인 심근증교육연구센터는 2022년 2월 5일(토), 6일(일) 양일간 온라인으로 4th Cardiac ASH Conference를 개최하였다, 이는 코로나의 여파로 인해 지난 2020년은 개최 취소, 2021년은 개최 연기되어 약 2년여 만의 학술대회이다.

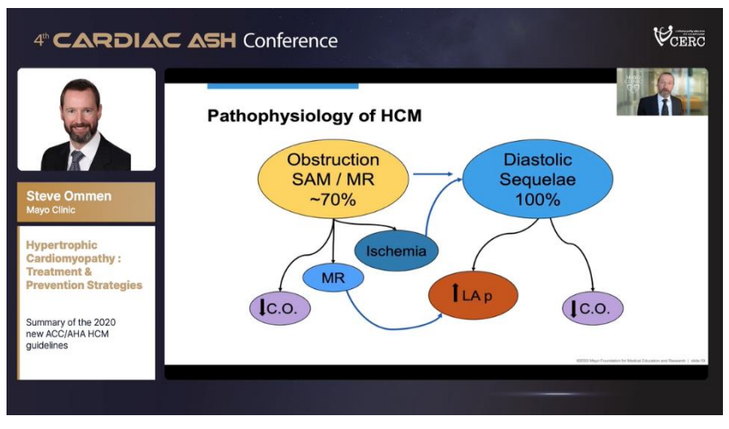
이번 학술대회는 처음으로 개최된 온라인 학술대회로 온라인 학술대회 전문 플랫폼 '닥터빌 컨퍼런스 (www.dvwebinar.co.kr)'를 통해 개최하였다.

닥터빌 컨퍼런스는 데스크톱, 노트북, 태블릿, 모바일 등 원하는 형태로 접속이 가능하다. 강의를 들으며 질문을 남길 수 있는 채팅 서비스와 이를 좌장이 실시간으로 확인해 의견을 나눌 수 있는 토론 시간을 마련했다. 또한 플랫폼을 통해 전자문서 형태의 초록과 발표자료를 열람할 수 있으며, E-booth(온라인 부스, 가상 전시관)를 통해 후원사의 기업 소개 및 제품 설명 등을 볼 수 있도록 준비했다.

4th Cardiac ASH Conference는 첫째 날 5일(토)에 ▲ Hypertrophic Cardiomyopathy: Diagnostic Strategies ▲ Hypertrophic Cardiomyopathy: Treatment & prevention Strategies ▲ Sarcoidosis: Diagnostic & Prognostic Issues ▲ Sarcoidosis: Treatment Strategies, 둘째 날인 6일(일)에 ▲ Cardiac Amyloidosis, Diagnostic Issues ▲ ATTR Cardiac Amyloidosis and More 총 여섯 개 세션으로 나누어 진행했다.

이번 학술대회는 대한내과학회로부터 내과 전문의 평점 및 순환기 분과 전문의 평점을 승인 받았으며, 처음 진행하는 온라인 학술대회임에도 의료 관계자의 많은 관심과 참여로 성황리에 마쳤다.





Program

Day-1 FEB. 05 (Sat) 2022

09:25-09:30	Opening Remarks	
09:30-10:50	Hypertrophic Cardiomyopathy : Diagnostic Strategies	Jae-Kwan Song Asan Medical Center Yong-Jin Kim Seoul National Univ. Hosp.
09:30-09:45	Best Use of The Contemporary Imaging Markers in Hypertrophic Cardiomyopathy	Chi-Young Shim Yonsei Univ. Severance Hosp.
09:45-10:00	Sex Differences in Hypertrophic Cardiomyopathy	Sangchol Lee Samsung Medical Center
10:00-10:15	Practical Advice for The Management of HCM	Yeonyee E. Yoon Seoul National Univ. Bundang Hosp.
10:15-10:30	Pediatric Cases of HCM, Special Considerations	Mi-Kyoung Song Seoul National Univ. Children's Hosp.
10:30-10:50	Discussion	
10:50-11:10	*Coffee Break*	
11:10-12:25	Hypertrophic Cardiomyopathy : Treatment & Prevention Strategies	Goo-Yeong Cho Seoul National Univ. Bundang Hosp. Hyung-Kwan Kim Seoul National Univ. Hosp.
11:10-11:30	Summary of The 2020 New ACC/AHA HCM Guidelines	Steve Ommen Mayo Clinic
11:30-11:45	Surgical Myectomy in HCM	Kyung Hwan Kim Seoul National Univ. Hosp.
11:45-11:55	Hypertrophic Cardiomyopathy Case I	Hyun-Jung Lee Seoul National Univ. Hosp.
11:55-12:05	Hypertrophic Cardiomyopathy Case II	Jang-Won Son Yeungnam Univ. Hosp.
12:05-12:25	Discussion	
12:25-13:25	*Lunch*	
13:25-14:35	Sarcoidosis : Diagnostic & Prognostic Issues	Dae-Won Sohn Seoul National Univ. Hosp. Man-Pyo Chung Samsung Medical Center
13:25-13:40	Diagnosis & Follow-Up of Cardiac Sarcoidosis, an Overview	Jun-Bean Park Seoul National Univ. Hosp.
13:40-14:00	How Imaging Helps to Understand The Disease	Ron Blankstein Brigham and Women's Hospital
14:00-14:15	Extracardiac Manifestions of Sarcoidosis, Pulmonologist's Viewpoint	Jin-Woo Song Asan Medical Center
14:15-14:35	Discussion	
14:35-14:55	*Coffee Break*	
14:55-16:10	Sarcoidosis : Treatment Strategies	Seung-Woo Park Samsung Medical Center Kye Hun Kim Chonnam National Univ. Hosp.
14:55-15:15	Rhythm Disturbances and Sudden Cardiac Death, What We Can Do More	Soryoung Lee Seoul National Univ. Hosp.
15:15-15:30	Treatment of Cardiac Sarcoidosis, When to Move Forward	Jukka Lehtonen Helsinki University Central Hospital
15:30-15:40	Cardiac Sarcoidosis Case I	Hong-Mi Choi Seoul National Univ. Bundang Hosp.
15:40-15:50	Cardiac Sarcoidosis Case II	So-Ree Kim Korea Univ. Anam Hosp.
15:50-16:10	Discussion	

Day-2 FEB. 06 (Sun), 2022

09:00-10:25	Cardiac Amyloidosis, Diagnostic Issues	Ho-Joong Youn The Catholic Univ. of Korea Seoul St. Mary's Hosp. Sung-Soo Yoon Seoul National Univ. Hosp.
09:00-09:15	Diverse Manifestations of Cardiac Amyloidosis	In-Chang Hwang Seoul National Univ. Bundang Hosp.
09:15-09:35	Diagnostic Strategy of Cardiac Amyloidosis: Guides and Pitfalls	Mazen Hanna Cleveland Clinic
09:35-09:50	ATTR Cardiac Amyloidosis	Darae Kim Samsung Medical Center
09:50-10:05	Pitfalls in The Diagnosis of Cardiac Amyloidosis, What More	Seung-Pyo Lee Seoul National Univ. Hosp.
10:05-10:25	Discussion	
10:25-10:45	*Coffee Break*	
10:45-12:10	ATTR Cardiac Amyloidosis and More	Eun-Seok Jeon Samsung Medical Center Jong-Won Ha Yonsei Univ. Severance Hosp.
10:45-11:00	Transplantation in Cardiac Amyloidosis, Clinical Issues	Hyun-Jai Cho Seoul National Univ. Hosp.
11:00-11:20	Promising Therapeutics in ATTR Cardiac Amyloidosis	Mathew Maurer Columbia University
11:20-11:30	Cardiac Amyloidosis Case I	Seung-Ah Lee Asan Medical Center
11:30-11:40	Cardiac Amyloidosis Case II	Woo-Baek Chung The Catholic Univ. of Korea Seoul St. Mary's Hosp.
11:40-12:10	Discussion	
12:10-12:15	Closing Remarks	

2 A.S.H - Amyloidosis

Amyloidosis

Definition and Prevalence

Seung-Pyo Lee
MD, PhD
Seoul National Univ. Hosp.

Cardiac amyloidosis is caused by abnormal deposits of non-native, insoluble proteins in the heart mainly in the interstitial space of the myocardium but also in the vessels, valves and the pericardium. Approximately 10 proteins are known to cause cardiac amyloidosis with light chain immunoglobulin, wild type transthyretin (TTR) or mutant TTR being the most com-mon. An amyloid protein is noted with a letter 'A' in front of the amyloid protein of interest, for example, an amyloid form of TTR as ATTR and the amyloid light chain as AL. Cardiac amyloidosis is rare but because of the unique morphologic and also, the possibility of cure, is conceived as treatable disease. The prevalence of AL type is approximately 1/10 million in the US and is almost undoubtedly associated with plasma cell dyscrasia. The prevalence of ATTR type has been reported to be up to 25% in those with 80 years or older by autopsy and certain reports have demonstrated that wild type TTR (wtTTR) cardiac amyloidosis might be underesti-mated in the elderly population. Considering these fin dings, the prevalence of cardiac amyloidosis may be underestimated than expected.

Diagnosis of Cardiac Amy-loidosis(Table 1)

The gold standard method of diagnosing cardiac amyloidosis is to verify the presence of amyloidogenic proteins in the heart that may explain the organ dysfunction. However, the diagnosis of cardiac amyloidosis can be made without endomyocardial biopsy if the amyloid is verified in an organ other than the heart and if the ventricular thickening or the rise of serum N-terminal pro-B-type natriuretic peptide (NT-proBNP) cannot be explained otherwise. But the use of the invasive tools for definite diagnosis, it is more important not to miss certain clues that leads to the clinical suspicion of cardiac amyloidosis. Cardiac amyloidosis is often diagnosed late because of vague symptoms that range from exertional dyspnea, (pre)syncope, chest pain or even sudden cardiac arrest, which may sometimes seem unclear and vague, leading to significant delays in the appropriate diagnosis and even, treated inappropriately for the wrong disease. The following are some typical findings that may be encountered in noninvasive tests and give clues to the diagnosis of cardiac amyloidosis. Importantly, the clinical presentation of cardiac amyloidosis is very diverse as stated previously and therefore, the absence of the listed findings does not rule-out its absence.

Modality /Method	Parameter	Diagnosis of organ involvement
1. Endomyocardial biopsy		Proof of amyloid deposit in the heart on pathologic examination
2. Biopsy of an organ other than the heart		Proof of amyloid deposit in other organs(fat pad, kidney, etc.) on pathologic examination
3. Echocardigraphy	Mean wall thickness	>12mm with no other cause and identification of amyloid deposit in other tissue or organ
4. NT-pro-BNP		>332ng/L in the absence of renal failure or atrial fibrillation

Table1. Diagnostic criteria for cardiac amyloidosis

For diagnosis of cardiac amyloidosis, the following criteria suffices. 1 only; 2 + 3 or 4

Electrocardiogram

There may be any abnormal 12-lead electrocardiogram (ECG) finding in up to 90% of patients with cardiac amyloidosis. A typical finding is the low QRS voltage in the limb leads despite ventricular thickening (Figure 1), which is extremely rare in patients with other disease that present as ventricular hypertro-phy. A QS wave in any two consecutive leads, especially in the septal leads, is called 'pseudoinfarction' as it is not a real 'infarction'. The pseudoinfarction pattern is also a 12-lead ECG finding that can be commonly seen in cardiac amyloidosis patients (Figure 2). However, the prevalence of the typical electrocardiographic findings is <50% and therefore, the low QRS voltage nor the pseudoinfarction pattern may not be seen in a majority of cardiac amyloidosis cases.

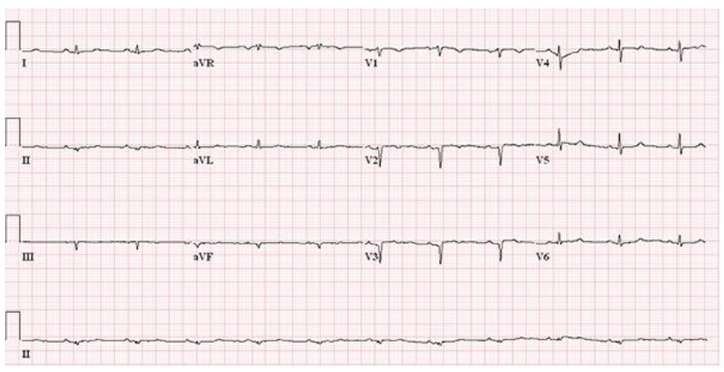


Figure 1. A typical low QRS voltage finding in a cardiac amyloidosis patient.

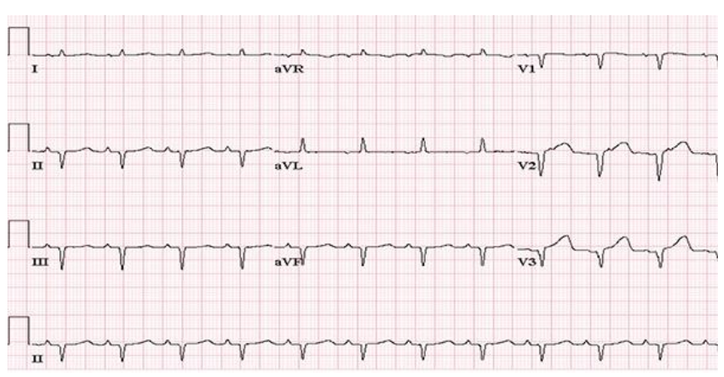


Figure 2. A typical pseudoinfarction pattern in lead V1 and V2.

Echocardiography

Cardiac amyloidosis should be always suspected in patients with symptoms or signs of heart failure, especially when the ventricu-lar wall is thick (Video 1) and when there is prominent diastolic dysfunction (Video 2). Cardiac amyloidosis may present as con-centric hypertrophy in contrast to hypertrophic cardiomyopathy where localized ventricular thickening is more common. Howe-ver, there have been papers that describe cardiac amyloidosis without ventricular thickening.

Echocardiography is very useful for patients with cardiac amyloidosis because it serves as a gatekeeper to the diagnosis of cardiac amyloidosis in most institutions and it can evaluate not only the structure of the heart but also, the hemodynamic consequences associated with the disease. It is not uncommon to notice findings other than the ventricular thickening such as valvular thickening and pericardial effusion. Biatrial enlargement that reflects the chronic diastolic dysfunction is seen in a majority of the patients. One of the most striking finding may be the 'apical sparing' pattern on the bull's eye plot of the global longitudinal strain using speckle tracking echocardiography (Figure 3). Although in a single center study, this apical sparing pattern has a 80~90% sensitivity and specificity for the diagnosis of cardiac amyloidosis.

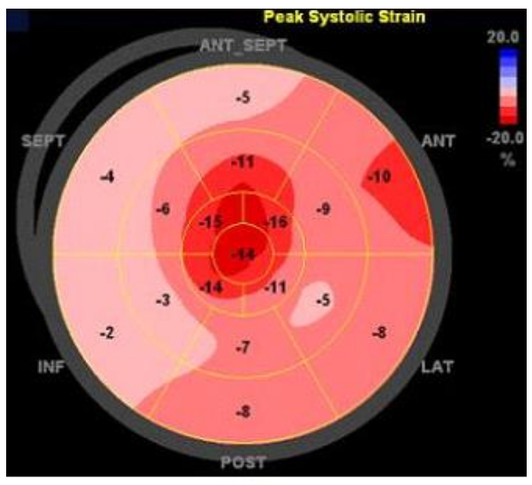


Figure 3. Apical sparing pattern on strain imaging that is typical for cardiac amyloidosis.

Cardiovascular magnetic resonance (CMR)

With the advent of new sequences and clinical experiences, CMR is now used popularly in patients with cardiac amyloidosis. It is useful not only for evaluation of the wall thickness and the overall structure associated with the disease but is unique for evaluation of the myocardial change itself, which cannot be fully evaluated with any other technology.

Using gadolinium-based contrast, the discovery of the difference in the gadolinium kinetics in the myocardium between the norm-al versus the amyloidosis patients has been seminal. The pattern of late gadolinium enhancement (LGE) in the myocardium is may be diverse, ranging from the global transmural or patchy focal LGE to suboptimal myocardial nulling (Figure 4), which has been shown to match the deposition pattern of the interstitial amyloid. Incidental findings such as intracardiac thrombus may also be detected on CMR, because of its superiority in signal-to-noise ratio over that of the echocardiography.

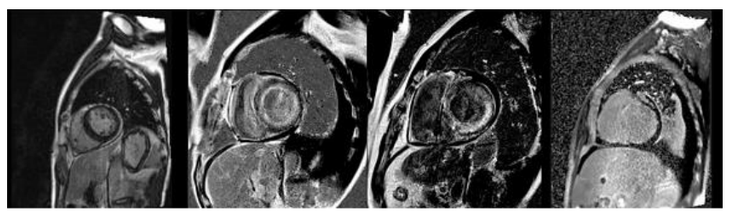


Figure 4. LGE-CMR findings, ranging from normal(far left), subendocardial diffuse LGE(middle left), patchy transmu-ral LGE(middle right) and suboptimal myocardial mulling (far right).

The parametric CMR has also been investigated for the assess-ment of cardiac amyloidosis and is now used as adjunct methods in the evaluation of the disease. This is based on the histologic findings that the extracellular space is significantly expanded in cardiac amyloidosis than any other myocardial disease and that this finding is reflected by the lengthening of the myocardial T1 relaxation time. Using specific sequences for mapping the T1 relaxation time such as modified look-locker inversion recovery (MOLLI) sequence or shortened MOLLI (shMOLLI) sequence, the measurement of native T1 or the calculation of extracellular volu-me fraction from both pre- and post-contrast T1 mapping results may also help in the diagnosis of cardiac amyloidosis (Figure 5).

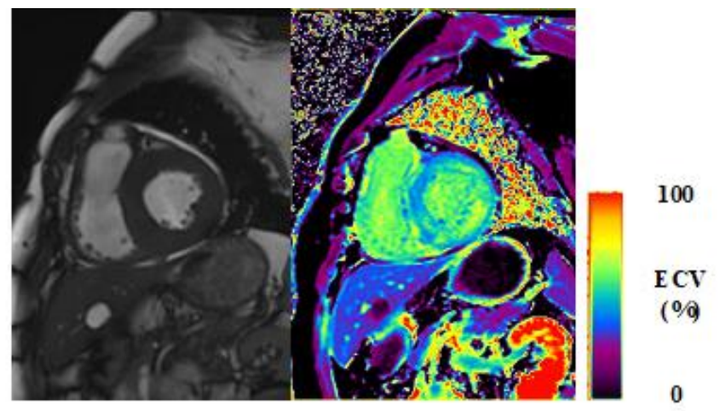


Figure 5. A patient with thick ventricle in cardiac amy-loidosis (left panel) and a representative ECV map (right panel). The average ECV of the LV myocardium was approximately 50%

Nuclear imaging

Single photon emission com-puted tomography(SPECT)

Bone imaging SPECT tracers based on diphosphonates, such as 99mTc-pyrophosphate (99mTc-PYC), 99mTc-methylene diphos-phonate (99mTc-MDP), 99mTc-hydroxymethylene diphosphonate (99mTc-HDP) and 99mTc-3,3-diphosphono-1,2-propanodicarbox-ylic acid (99mTc-DPD) can be used for the diagnosis of cardiac amyloidosis, especially the ATTR type (Figure 6). Of these, the 99mTc-DPD has been most extensively investigated and it does not seem to differentiate between the wtTTR versus the mtTTR. Although accurate quantitation of the tracer retention is difficult using SPECT, the degree of tracer retention in the myocardium is a marker of the myocardial involvement severity.

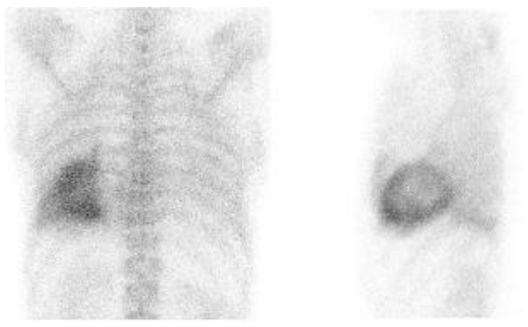


Figure 6. A representative 99mTc-DPD scan in a patient with wtTTR cardiac amyloidosis

Continued on page 3

Positron emission tomography (PET)

The recently developed PET tracers have been made to directly target the amyloidogenic proteins itself. Building on the fact that the cause of Alzheimer's disease is amyloid accumulation in the brain parenchyme, several investigators have reported the clinical utility of amyloid PET tracers in cardiac amyloidosis such as 11C-Pittsburgh B (PiB) compound in imaging cardiac amyloidosis. Others have also used 18F-tagged radiotracers such as 18F-florbetapir or 18F-florbetaben because of the limited half-life of the 11C (t1/2≈20minutes) and the relatively longer half-life of the 18F (t1/2≈110minutes). These 18F-tagged radiotracers have been shown to accumulate specifically to the sites of amyloid deposits in the myocardium. The major caveat with the PET agents is that there is a shortage of outcome studies that show its clinical utility.

Pathologic diagnosis

The visualization of amyloid deposit directly from pathologic specimens is the cornerstone of cardiac amyloidosis diagnosis. A typical myocardial specimen of the cardiac amyloidosis patient would show expansion of the extracellular matrix in a conventional hematoxylin and eosin staining (Figure 7). This amyloid deposit is confirmed by a typical apple-green birefringence on Congo red staining (Figure 7). However, some institutions advocate the use of adjunct methods such as sulfated Alcian blue or amyloid P immunostaining as the sensitivity may be low and the difficulty in quantification of amyloid deposit in the Congo red staining. The type of amyloid deposit can also be confirmed with immunohistochemical staining. The pattern of amyloid deposit may vary, from a typical diffuse subsarcolemmal pattern to 'nodular' patterns where amyloid deposits can be seen in the form of clumps in the interstitial space.

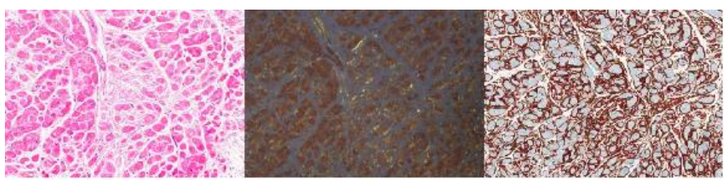


Figure 7. Myocardial specimen examination by pathology. H&E (left), Congo-red (middle), amyloid P (right) staining results.

Typing cardiac amyloidosis

It is important to determine the type of cardiac amyloidosis as this significantly impacts the treatment and also the prognosis. The typing of AL cardiac amyloidosis depends on the analysis of the serum protein, for example, protein immunoelectrophoresis and serum free light chain assay. However, there are some important caveats to consider with these 'indirect' methods. First, these 'indirect' methods of typing are only useful in systemic AL amyloidosis and furthermore, there may be some AL cardiac amyloidosis without a definite rise in the serum concentration of the AL protein. Second, although the majority of cardiac amyloidosis which are not the AL type by serum light chain assay may be the ATTR type, a minority of cardiac amyloidosis patients are neither the AL nor the ATTR type. Therefore, it would be too impetuous to directly determine the type of cardiac amyloidosis without using either the immunohistochemical or the proteomic analysis or even both.

Pathologic examination are very useful for determining the type but in contrast to the expectation, the immunohistochemical staining results may not be always clear-cut and there can always be false-positive or -negative results. A lot of laboratories may support these pathology results with appropriate proteomic analysis, mainly mass spectrometry. Additionally, in those who are suspected to have cardiac amyloidosis due to mtTTR, the genotyping of the TTR gene is also a must-do item.

Treatment

Treatment of heart failure symptoms

Besides the treatment of amyloidosis infiltration itself, the mainstay of cardiac amyloidosis treatment is directed towards control of heart failure symptoms. Loop diuretics, either stand-alone or in combination with mineralocorticoid antagonist, is often used for volume control. However, because a significant proportion of those of with cardiac amyloidosis have concomitant autonomic dysfunction, diuretics should be used with caution so as to avoid excessive volume depletion. Likewise, based on the same reason, medications that are standard for heart failure, such as renin-angiotensin system blocker or beta-blocker, should be used prudently.

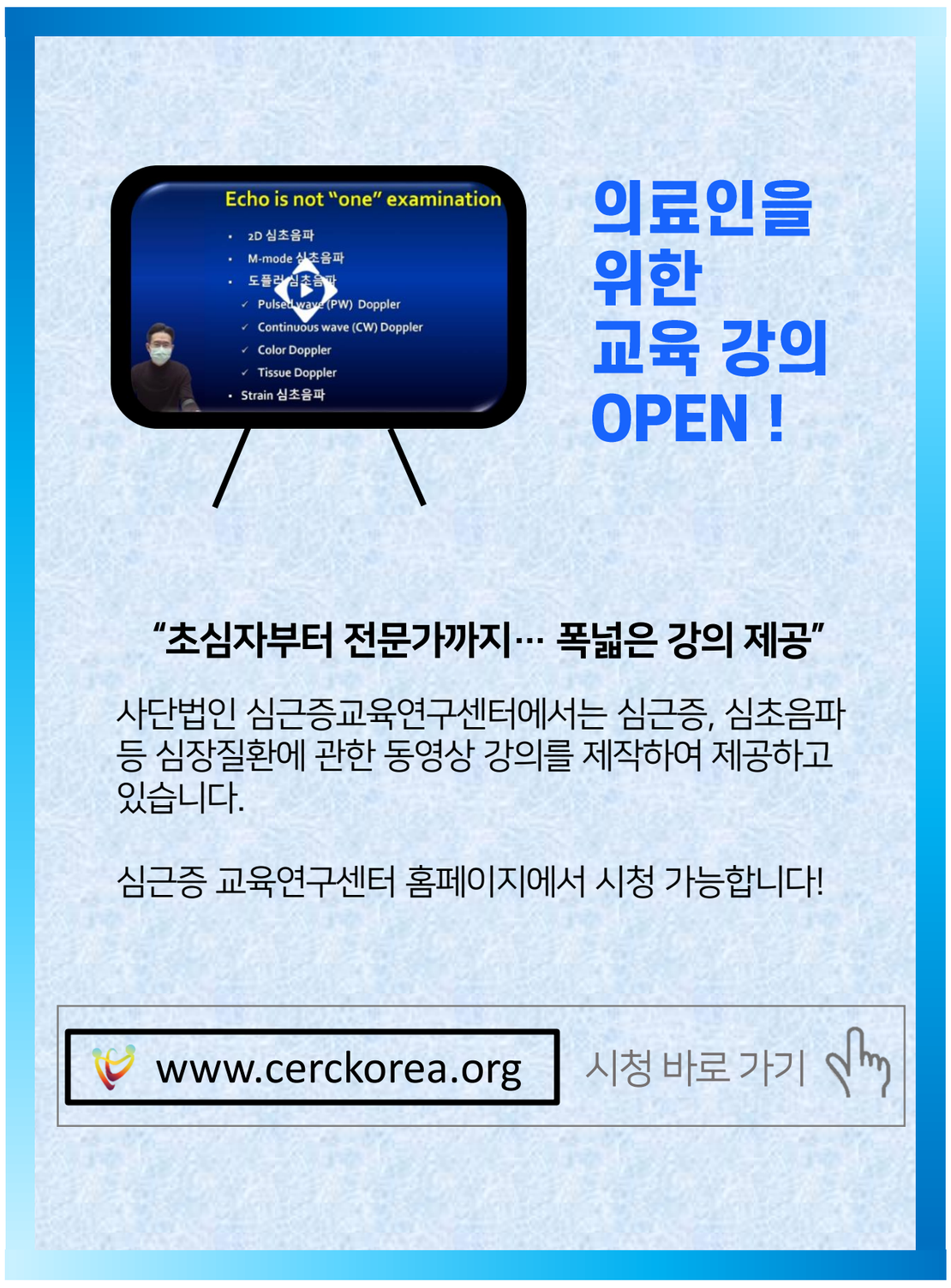
The clinical evidence on the use of cardiac resynchroniza-tion therapy has not been proven, partially because of the limited survival in cardiac amyloidosis patients.

Treatment directed against the amyloid protein

As AL cardiac amyloidosis is almost always invariantly associated with plasma cell dyscrasias, treatment of AL cardiac amyloidosis is directed against clearance of the plasma cells as well as the prevention of target organ damage, including the myocardium. The standard treatment regimen consists of a combination of chemotherapeutic agents, such as alkylating agent (melphalan, cyclophosphamide) and/or proteasome inhibitor (bortezomib) or immunomodulatory agent (thalidomide, lenalidomide). After a significant reduction of the plasma cell burden, the chemotherapy is usually followed by autologous hematopoietic cell transplantation, if eligible. However, as the chemotherapeutic agents have minimal effect on eliminating the AL protein deposited in the end organ, antibodies that direct the misfolded proteins are being actively developed.

As for the ATTR, treatment is directed towards disruption of ATTR, reduction of TTR transcription or stabilization of the TTR protein. Following the promising results of doxycycline, together with tauroursodeoxycholic acid, in reducing amyloid aggregation in mice by stabilizing the TTR protein, a phase II trial has also shown promising results in cardiac amyloidosis. Anti-sense oligonucleotides directed against the transcription of TTR protein has also shown to be safe in phase II trials and are now being escalated into phase III trials. Tafamidis and diflunisal binds to the thyroxine binding sites of the TTR, thereby inhibiting the dissociation of TTR into monomers. Again, both have shown promising results in those with ATTR cardiac amyloidosis and in the midst of phase III clinical trials.

Although the cardiac involvement by amyloid protein was once considered a contraindication of heart transplantation, this belief is changing in centers where multidisciplinary team approach is possible. Heart transplantation is currently considered an option for cardiac amyloidosis in many hospitals nowadays and the outcomes comparable to any other restrictive cardiomyopathies.



4 건강 이야기

건강을 지키는 식습관

당신의 식탁은 건강하신가요? – 심혈관질환을 예방하는 식사

현대 사회의 발달에 따라 우리의 식탁도 과거에 비해서 더욱 풍요롭고 다채로워 졌습니다. 경제적 풍요 및 세계화에 따라 다양한 음식을 접하게 되고 국내 식습관은 빠르게 서구화 되었습니다. 바쁜 일상으로 인해 인스턴트 식품과 배달이 발달하게 됐습니다. 최근에는 COVID-19의 영향으로 비(非)대면을 선호함에 따라 배달 음식, 인스턴트와 같은 음식이 우리 식탁에 이전보다 더 자주 오릅니다. 이러한 식습관은 비만, 당뇨, 고혈압 등 각종 질환의 원인이 되기도 합니다. 식탁에 적색 불이 켜졌습니다.

잘 알다시피 포화지방이 많은 음식과 나트륨, 당분 함량이 높은 음식을 많이 섭취하면 여러 질환의 위험이 높아지게 됩니다. 미국에서는 혈압을 낮추기 위해 국립보건원과 심폐혈관연구소 에서 DASH 식단을 만들었는데요, 고혈압 환자의 혈압을 낮추기 위해 만들어진 식단이나, 여러 질환에서 효과를 보이게 되어 현재는 널리 알려진 인기있는 식단 중 하나입니다.

DASH는 'Dietary Approached to Stop Hypertension'의 약자로 우리말로 풀이하면 '고혈압을 막기 위한 식이요법'이라 할 수 있습니다. 이 식단은 포화지방, 콜레스테롤 및 총 지방의 섭취를 줄이고 과일, 채소, 무지방 및 저지방 유제품 섭취를 강조한 식단입니다. 또한 전곡류, 생선, 가금류, 견과류를 식사에 포함시키고 붉은 색 육류, 당류, 설탕 함유 식품의 섭취를 줄이도록 권장합니다.

<그림1>은 미국의 DASH 식단입니다. 아래의 <표1>은 보건복지부에서 작성한 '질환 관리를 위한 바른 식생활 가이드'에 소개된 DASH 식사 구성의 예입니다.

DASH 식단은 임상적으로 그 효과가 입증된 것으로, 식단과 함께 생활습관 을 개선하면 혈압을 낮추고 고혈압을 예방하는데 도움이 됩니다.

<표1> DASH 식사 구성의 예

출처: 보건복지부 '질환 관리를 위한 바른 식생활 가이드'

식품군	섭취횟수	식품의 예 및 1회 섭취량
곡류 및 그 제품	6~8/일	빵 1쪽, 밥 1/2공기, 삶은 국수 1/2공기
채소류	4~5/일	잎 채소 생것 1컵, 익힌 채소 1/2컵
과일류	4~5/일	야구공 크기 과일 1개, 과일주스 1/2컵
저지방 또는 무지방유제품	2~3/일	저지방우유 1컵, 무가당요구르트 1컵
육류, 가금류 및 생선	6 이하/일	익힌고기 30g, 생선 작은 것 1토막, 달걀 1개
견과류, 종실류 및 말린 콩류	4~5/주	익힌 콩 1/2컵, 견과류 1/3컵
지방과 기름	2~3/일	기름 1작은술, 마요네즈 1큰술
당류	5 이하/주	설탕 1큰술, 잼 1큰술

심혈관 질환의 적… 나트륨

세계보건기구(WHO) 조사 결과에 따르면 전 세계 사망원인 1위는 심혈관 질환입니다. 고혈압, 고지혈증, 당뇨 등 은 심혈관질환을 일으키는 주요 요인 이며, 여기에 직접적으로 영향을 미치는 것이 당과 나트륨이기에 세계보건기구 (WHO) 및 각 나라에서는 이를 줄이 고 자 노력하고 있습니다.

특히 우리나라 성인 나트륨 섭취량은 약 3700mg으로 세계보건기구(WHO) 권고상한치 2000mg을 초과하고 있습니다. 2000mg이 커피 티스푼으로 한 스푼 정도인 걸 생각하면 우리가 얼마나 과다섭취하고 있는지 알 수 있는 부분입니다.

우리나라 사람들의 주요 나트륨 섭취는 김치, 라면, 간장 및 된장을 사용하는 찌개/전골, 국/탕 등의 음식을 통해 이뤄지고 있습니다. 전체 섭취의 60% 이상을 위의 음식을 통해 섭취하는 만큼 이들 음식을 통한 나트륨 섭취량을 집중적으로 조절할 필요가 있습니다. 조리할 때는 저염 소금, 저염 간장 을 사용하거나, 국물 음식은 건 더기 위주로 섭취하는 것도 좋은 방법 입니다.

우리 정부도 바른 식생활을 통한 질환 관리를 위해 노력하고 있는데요, 보건복지부 홈페이지를 통해 각종 질환과 식사 원칙, 식단 등을 확인할 수 있습니다.

한번 발생하면 이전으로 되돌리기 힘들고 관리가 힘든 질환들이지만 평소 생활 습관, 특히 식습관 개선 시 효과를 볼 수 있기 때문에 반드시 개선하는 것 이 좋습니다.

<표2> 고혈압 관리를 위한 표준 식단의 예

출처: 보건복지부 '질환 관리를 위한 바른 식생활 가이드'

끼니	1일	2일	3일
아침	현미밥(230g) 콩나물국(콩나물 50g) 조기구이(50g) 양상추무침(70g) 저염물김치(70g)[1]	굴죽(굴 40g, 표고 · 양파 · 당근 · 호박 40g, 쌀 60g) 장조림(쇠고기 20g)[2] 쑥갓나물(70g) 저염물김치(70g)	쌀밥(230g) 양배추국(양배추 50g) 모듬전(동태전 50g, 양파전(40g) 돌나물무침(70g) 무초절이(50g)
점심	보리밥(230g) 무채국(무 50g) 북어보푸침(15g) 두부부침(80g) 호박나물(70g) 배추김치(20g)	콩밥(230g) 아욱국(아욱 50g) 꽁치생강구이(50g) 달걀찜(50g) 오이생채(70g) 무초절이(50g)	새싹비빔밥 (보리밥 230g, 새싹 채소 70g, 쇠고기 30g) 실파김국(실파 50g, 김 1장) 두부조림(80g) 백김치(20g)
저녁	팥밥(230g) 근대국(근대 50g) 닭살겨자냉채(닭살 80g) 도라지나물(50g) 깻잎찜(20g) 양배추초절이(50g)	보리밥(230g) 미역국(건미역 5g) 해물볶음 (물오징어 50g, 새우 100g/양파, 당근 30g) 감자샐러드(감자 100g, 오이, 당근 40g) 미나리나물(70g) 배추김치(20g)	콩밥(230g) 시금치국(시금치 50g) 삼치레몬구이(100g) 상추/치커리무침(70g) 저염물김치
간식	저지방우유(1컵) 사과(160g) 무가당호상요구르트(100g)	두유(1컵) 무지방우유(1컵) 귤(240g)	저지방우유(2컵) 토마토(350g) 키위(80g)

끼니	4일	5일	6일
아침	차조밥(230g) 근대국(근대 50g) 연두부찜(150g) 가지나물(70g) 양배추초절이(50g)	통밀빵(70g) 스크램블(달걀 1개, 양파 · 호박20g) 채소스틱(오이 · 당근 · 샐러리 70g) 무지방우유(1컵)	쌀밥(230g) 열무사골국(열무 50g) 닭불고기(닭고기 40g) 무나물(70g) 김무침(20g) 저염물김치(70g)
점심	보리밥(230g) 연포탕(낙지 100g) 돼지고기 장조림 (돼지고기 40g) 청경채가쯔오볶음(70g) 백김치(20g)	완두콩밥(210g) 쇠고기두부전골 (쇠고기 40g, 두부 80g , 각종 채소) 감자조림(130g) 무생채(70g) 배추김치(20g)	강된장 비빔밥 (보리밥 230g, 두부 40g, 쇠고기 20g, 양파, 호박, 고추 40g) 달걀말이(1개) 우무무침(100g) 배추김치(20g)
저녁	현미밥(230g) 버섯국(70g) 불고기(쇠고기 80g, 양파 20g) 모둠채소쌈(100g) 물미역무침(50g) 배추김치(20g)	쌀밥(210g) 무다시마국(무 50g) 쇠고기 마늘종볶음 (쇠고기 40g/ 마늘종 30g) 꼬막찜(70g) 시금치나물(70g) 저염물김치(70g)	보리밥(230g) 미역국(건미역 5g) 갈치조림(50g) 꽈리고추돼지고기조림 (돼지고기 40g, 꽈리고추 50g) 마늘초절이(50g) 저염물김치(70g)
간식	저지방우유(1컵) 바나나(100g) 무가당호상요구르트(100g)	저지방우유(1컵) 포도 160g	저지방우유(1컵) 무가당호상요구르트(100g) 배(220g)

<그림1> 미국의 DASH 식단

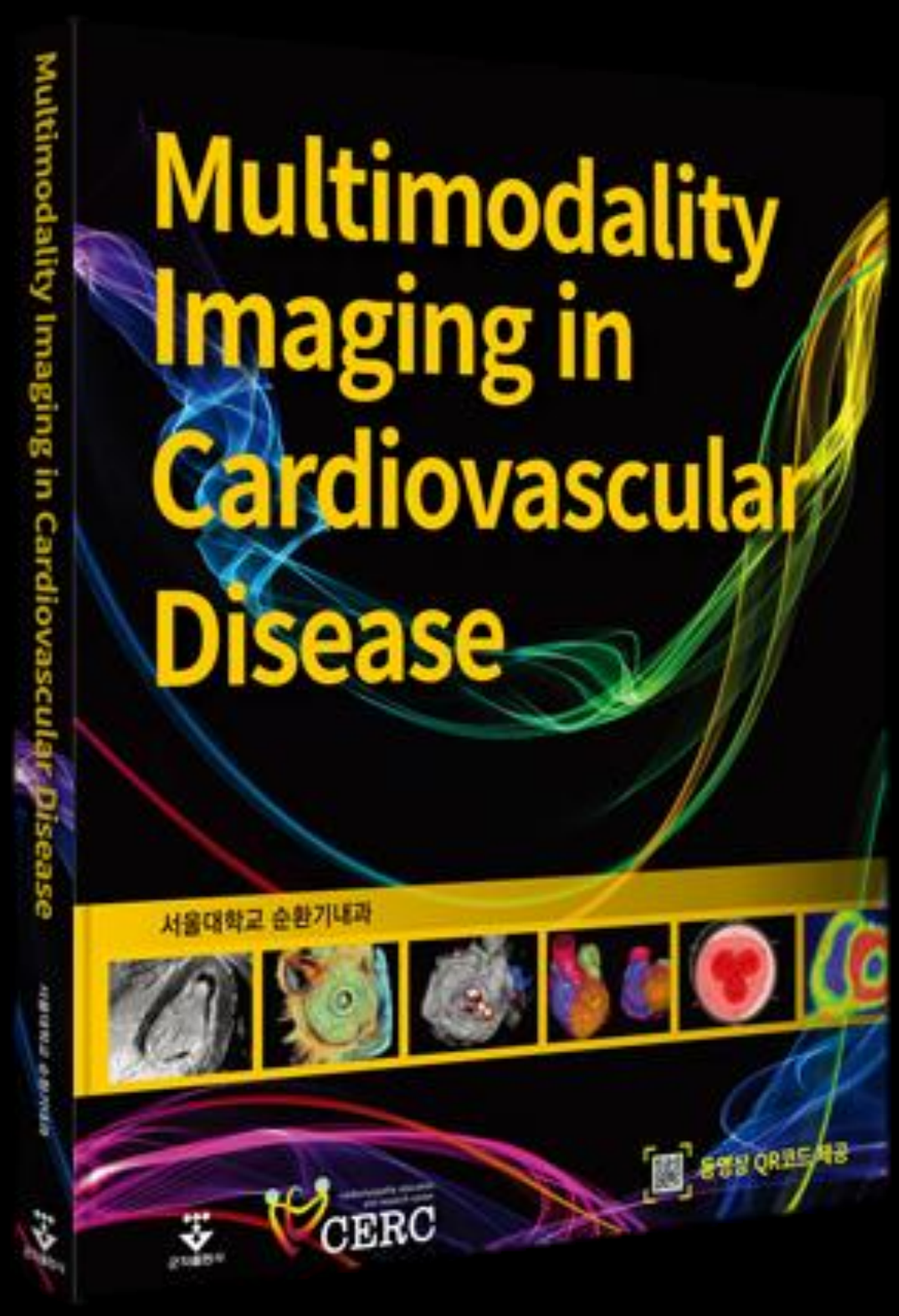
Multimodality Imaging In Cardiovascular Disease

순환기 질환의 다양한 증례!

Multimodality Imaging이 왜 필요한지
진단하는 과정과 치료는 어떻게 하는지

근거 중심 및 실질적 경험을 바탕으로 접근하여
실제 환자 치료에 도움을 받을 수 있는 모든 것!

CERC



Multimodality Imaging in Cardiovascular Disease

의료진이 직접 엄선한

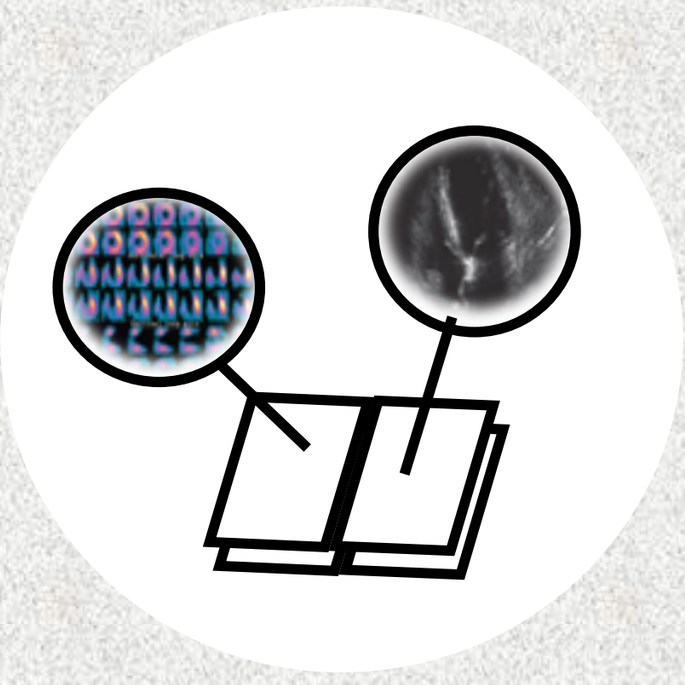
임상 사례

소견 마다 영상을
확인할 수 있는

QR 코드

CT, MRI, PET 등 470개의

다양한 영상자료



심근증 환자의
보다 나은 삶을 위해
최선을 다하겠습니다.

심근증은 의료계의 관심에서도
벗어나 있어 현대의학의 혜택을
누리지 못하는 질환입니다.

다양한 교육 및 지원을 통한
전문가 양성과 대국민 홍보사업을 위해
여러분의 관심과 도움이 필요합니다.

신한 100-034-774312 | (사)심근증교육연구센터

Johann semens

소식 제4호

2022년 8월 5일

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