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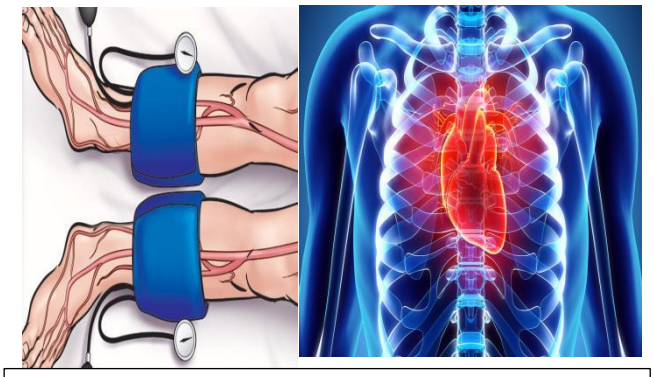
IN THIS ISSUE

Dear Doctor,

Greetings from Micro Labs Limited. We are happy to bring you "Cardio Mag" which contains latest and interesting news in the field of Cardiology

Relationship between steno-risk and subclinical atherosclerotic disease in individuals with type 1 diabetes

INTRODUCTION- Type 1 diabetes (T1D) accounts for 10 to 15% of all the causes of diabetes.[1] Globally, T1D prevalence is rising, and one of the main causes of death for those with T1D was cardiovascular disease (CVD). While acute diabetes consequences, like hypoglycemia and ketosis, are more likely to result in death among younger T1D patients, as individuals age, cardiovascular disease CVD becomes more common.[2] Effective primary preventions of CVD in this relatively young population depends on early diagnosis of the condition at the preclinical stage. Though the pathophysiology of CVD and risk factors in these two types of diabetes are not exactly the same, international recommendations for the prevention and management of CVD in T1D are derived from the extrapolation of data on type 2 diabetes. There were not many methods for early CVD screening that can identify subclinical atherosclerosis, such as evaluating the ankle brachial index (ABI) or the carotid intima media thickness. To predict the first fatal or non-fatal CVD event in the T1D population, other techniques in the form of cardiovascular risk scores have been developed. One such tool was the Steno Type 1 Risk Score (Steno-Risk). This score has a strong prognostic value for cardiovascular events at five and ten years in T1D, but prospective longitudinal studies were needed to validate it in other groups.[1] The aim of the current study was to determine the association between steno risk and subclinical atherosclerotic disease identified by the ABI and to assess the incidence of these abnormalities in T1D patients.



Ankle brachial index in assessing cardiovascular risk

METHODS-This was a single-center, cross-sectional study conducted among 312 T1D patients. The patients underwent a clinical examination to measure their body mass index (BMI), waist circumference (WC), systolic blood pressure (SBP), and diastolic blood pressure (DBP), in addition to thorough investigation regarding their personal and family histories of cardiovascular disease. For these patients, an Ankle-Brachial Index assessment was performed. A metabolic evaluation was carried out to measure HbA1c, total cholesterol, HDL cholesterol, and triglycerides as well as albuminuria. Individuals were classified according to their ABI values: an ABI of less than 0.9 signified peripheral artery disease (PAD), an ABI of more than or equal to 1.3 indicated arterial calcification (AC), and an ABI of between 0.9 and 1.3 was considered normal. In 277 patients, the 10-year CVD risk was estimated using the Steno Risk equation, which sets the risk at less than 10%, 10%–20% moderate, and more than or equal to 20% high. Heart failure, peripheral artery disease, ischemic heart disease, and ischemic stroke were among the composite CVD end-points used by this instrument.

RESULTS-This study results showed that 138 (44, 2%) had normal ABI, 112 (35, 9%) had PAD, and 62 (19, 9%) had AC. Men experienced AC at higher rates than women (p = 0,006). Both the PAD and AC groups had a high prevalence of a family history of stroke (p = 0,042 and p = 0,046 respectively). In comparison to patients with normal ABI, those with AC were more often older (36 ± 13 vs. 30 ± 12; P =0.012), had a longer duration of diabetes (20 ± 11 vs. 14 ± 9; P < 0,001), had a higher prevalence of hypertension (25,8% vs. 14,5%, P = 0,05), smoked more frequently (19,3% vs. 9,4%, P = 0,046), had a lower estimated glomerular filtration rate (egger) (81 ± 30 vs. 92 ± 28, P = 0,014), and had more DR (diabetic neuropathy) (50 % vs. 22,4%, P <0,001) and pain neuropathy (22,6% vs. 6,5 %, P = 0,001). Compared to individuals with a normal ABI, patients with PAD exhibited higher SBP (131 ± 16 vs. 127 ± 19; P = 0,006), MBP (mean blood pressure) (98 ± 10 vs. 95 ± 12; P = 0,03), and a lower estimated glomerular filtration rate (egger) (84 ± 24 vs. 92 ± 28; P = 0.017). Table 1 depicted a direct correlation between the Steno-Risk score and the AC objective at the ankle brachial index. The results indicated that there were higher numbers of participants with AC in the Steno-Risk 10–20% and ≥20% groups compared to the Steno-Risk <10% group (37, 5 vs. 15, 5; 29 vs. 15, 5 p = 0, 0106), respectively. Nevertheless, there were somewhat fewer patients with PAD in the Steno-Risk 10–20% than in the Steno-Risk <10% (22, 5 vs. 36, 4) and slightly more subjects with PAD in the Steno-Risk ≥20% than in the Steno-Risk <10% (38, 7 vs. 36, 4).

N (%) P= 0,0106		ABI category normal	PAD	AC	Total n (%)
STENO-RISK category	Steno-Risk <10%	99	75	32	206 (74,4)
	Steno-Risk 10–20%	16	9	15	40 (14,4)
	Steno-Risk =20%	10	12	9	31 (11,2)
Total		125	96	56	277 (100)

Table 1. Association between ABI anomalies and StenoRisk category

DISCUSSION-The current study showed that ABI could be an easy-to-use method for T1D for screening atherosclerosis and ABI > 1,30 indicating the presence of arterial calcification, was substantially linked to moderate and high cardiovascular risk as determined by stenorisk.[1] Similar to this, Lima A et al. showed that ABI is a simple and accurate technique that has shown to be effective in determining the degree of subclinical atherosclerosis in people with type 1 diabetes. Subclinical atherosclerosis in T1D patients was linked to vascular calcification and the ABI.[3]

CONCLUSION-The current study concludes that, ABI > 1, 30 was prevalent in older type 1 diabetic patients and was related to factors such as male gender, smoking, hypertension, long-term diabetes, and a history of stroke in the family. Furthermore, in patients with type 1 diabetes, ABI > 1,30, indicating the presence of arterial calcification, was substantially correlated with moderate and high cardiovascular risk as determined by stenorisk.

Ankle Brachial index(ABI) might be an easy tool for screening atherosclerosis in people with type 1 diabetes.

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Relationship between coronary collaterals and myocardial salvage determined by Serial Cardiac Magnetic Resonance Imaging in post acute myocardial infarction

INTRODUCTION-Coronary collaterals provide an alternate source of blood supply to the heart, reducing myocardial ischemia in the event of flow-limiting stenosis or obstruction. In acute and stable coronary artery disease, angiographically visible collaterals have been linked to decreased infarct size, better left ventricular function and a lower risk of future unfavourable cardiac events. In ST-segment elevated myocardial infarction (STEMI) patients having successful primary percutaneous coronary intervention (PCI), well-developed collaterals to the infarct-related artery (IRA) minimize myocardial infarct size and enhance myocardial salvage.[1] Patients with coronary artery disease have been found to have reduced mortality rates when using coronary collateral flow in angiography. However, the underlying mechanism importance was limited. Thus, this study aimed to investigate the significant coronary collateral flow linked to more salvaged myocardium and a decreased risk of heart failure in individuals suffering from acute myocardial infection (AMI).[2]

METHODS-In this retrospective analysis, a total of 36 patients (81% men, mean age 56 ± 9 years) were included. According to standard clinical practice, all patients had primary PCI. Aspirin, unfractionated heparin, and P2Y12 inhibitors were given to all patients. Patients with their first AMI who had a percutaneous coronary intervention within 24 hours of symptom onset were visually characterized by giving a Cohen-Rentrop Score (CRS) ranging from 0 (no collaterals) to 3 (full retrograde filling of the occluded artery). Based on the lack of antegrade flow and the change in flow following PCI, the infarct-related artery and culprit lesion were identified. The TIMI classification of perfusion was used to assess the antegrade flow: grade 0, no perfusion; grade 1, penetration without perfusion; grade 2, partial perfusion; and grade 3, complete perfusion. The intraclass correlation value suggested that the CRS evaluation was reproducible between two observers (ICC=0.872, $p<0.001$). After myocardial infarction, cardiac magnetic resonance imaging was performed on all patients within 3–5 days and again after 12 weeks of PCI. Based on the CRS, student's t-test was used to compare patients who presented only with STEMI, had TIMI flow prior to PCI 0-3, and had a culprit lesion in the right coronary artery.

RESULTS-This study showed the culprit lesion was exclusively discovered in the right coronary artery in patients with CRS 2-3, however, it was primarily discovered in the left anterior descending artery in the group with inadequate collateralization. Patients with CRS 2-3 had notably reduced infarct sizes at baseline and week 12. The I/R edema in the CRS 0–1 and CRS 2-3 groups was comparable. Significantly greater MSI was also correlated with appropriate collateral flow. In CRS 2-3 compared to CRS 0-1, there was a statistically significant decrease in infarct size from baseline to 12 weeks. All patients experienced a substantial decrease in infarct size (−4.3%, CI −5.9 to −2.6%, p < 0.001). Additionally, the left ventricular ejection fraction of both groups improved early after AMI (Figure 1), but the recovery was higher in CRS 2-3 patients than in CRS 0-1 patients (+8 ± 5% vs. +3 ± 5%, p = 0.015). In patients with CRS 2-3, NT-proBNP dropped in 12 weeks significantly more (−616 pg/mL [213–1547] vs. −384 pg/mL [231–709], p = 0.313). There were not significant variations between the two groups in terms of the clinical parameters used to classify the severity of heart failure (NYHA) or the clinical score for angina severity (CCS). A substantial correlation was observed between CRS 2-3 and a reduced final infarct size, even following the inclusion of patients with a TIMI flow ≥ 2 (p = 0.013). Likewise, after excluding patients who presented with non-ST elevation myocardial infarction, a statistically significant correlation (p < 0.001) was found between CRS 2-3 and a smaller final infarct size. A sensitivity analysis was carried out including patients (CRS 0–1 n = 5; CRS 2–3 n = 8) with a culprit lesion exclusively in the right coronary artery in order to account for the unequal distribution of infarct location. The results demonstrated a strong association between CRS 2–3 and smaller final infarct size (p = 0.052).

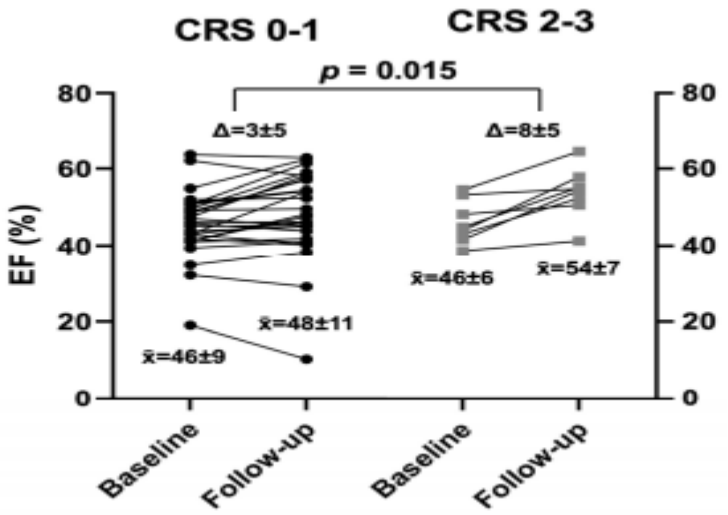

Figure 1. Baseline and follow-up left ventricular ejection fraction based on Cohen Rentrop Score (CRS 0–1: black: CRS 2–3: grey). Δ: change

DISCUSSION-The current study findings showed collateral circulation has a cardio-protective effect on myocardial salvage following acute myocardial infarction. Similar to this, Kim E et al. showed a positive effect of early collateral blood flow in individuals suffering from ST-segment elevation myocardial infarction.[3]

CONCLUSION-The current study concluded that relevant collateral flow to the infarct-related artery was linked to a reduced final infarct size at 12 weeks in patients who had their first AMI. This finding translated into a higher improvement in systolic left ventricular function.

Relevant collateral flow to the infarct-related artery was linked to a reduced final infarct size at 12 weeks in patients who had their first AMI.

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Correlation between blood pressure (BP) control and long-term major adverse cardiovascular and cerebrovascular event (MACCE)

INTRODUCTION-The term "obstructive sleep apnea" (OSA) refers to the episodes of hypopnea or apnea that result in a partial or total obstruction of the airways. On the other side, an increase in systemic arterial blood pressure above a specific threshold was known as hypertension. Numerous pathophysiological variables, including gender, obesity, an unhealthy lifestyle, poor sleep quality, the renin-angiotensin system, and enhanced fluid distribution, were shared by both OSA and hypertension.[1] Comorbid OSA affects between 30 and 50 percent of hypertensive patients. Further, it was clear that in patients with OSA who also have hypertension, the blood pressure (BP) can only be slightly lowered (3 mmHg) following therapy with continuous positive airway pressure (CPAP). In addition to hypertension, OSA was linked to coronary heart disease, heart failure, stroke, and other cardiovascular risks. Moreover, valsartan was found to cause a four-fold reduction in mean 24-hour blood pressure compared to CPAP in untreated hypertensive patients with OSA in a previous 8-week randomized controlled trial. This suggested that antihypertensive medications were more beneficial for controlling blood pressure in OSA patients with hypertension; however, the long-term benefits of blood pressure control for cardiovascular disease (CVD) outcomes in OSA have not been assessed.[2] Thus, this study aimed to investigate the potential purpose of blood pressure control by assessing the relationship between blood pressure control and the long-term cardiovascular effects among individuals having both OSA and hypertension.

METHODS-This was a retrospective study, included total of 3267 patients and divided into 3 groups by the follow-up BP. The first onset major adverse cardiovascular and cerebrovascular event (MACCE) after enrolment during the follow-up period, including cerebrovascular and cardiovascular events that were fatal or non-fatal, were the primary outcomes. Every endpoint was established using the suggested definitions provided by the Standardized Data Collection for Cardiovascular Trials Initiative. Cases of fatal illness were categorized using the

international classification of diseases (ICD-10) classification code. Kaplan-Meier survival curves were utilized to assess the cumulative incidence of primary outcomes, and the log-rank test was employed to estimate the variation between the various degrees of blood pressure control. To investigate the relationship between the levels of blood pressure management in the entire cohort and OSA patients and the outcomes of CVDs (such as MACCE, cardiac event, and cerebrovascular event), Cox proportional hazards models were conducted.

RESULTS- The current study showed that at baseline, 77.6% of patients had an OSA diagnosis, with a mean age of 48.6 years. During a median follow-up of 7.0 years, 379 out of 3267 patients had MACCE. When blood pressure was not maintained below 140/90 mmHg, the incidence of stroke considerably decreased (log-rank test, P=0.022, Figure 1). Additionally, when the three groups were compared pairwise, the incidence of stroke was as follows: ≥140/90 mmHg vs. 120–139/80–89 mmHg ($\chi2$=3.44, P=0.064), ≥140/90 mmHg vs.<120/80 mmHg ($\chi2$=10.37, P=0.001), and 120–139/80–89 mmHg vs.<120/80mmHg ($\chi2$=10.36, P=0.001). However, there was no discernible difference between cardiac events and MACCE and BP control. Only BP management between 120 and 139/80 and 89 mmHg was linked to the lowest risk of cerebrovascular incident after complete risk adjustment (HR: 0.53, 95%CI: 0.35–0.82). The lowest HR of the incident MACCE and cerebrovascular event was connected, separately, to the SBP control range of 120–139 mmHg. Patients who managed their blood pressure below 80 mm Hg had nearly 50% decreased risk of a cerebrovascular incident [(HR:0.51, 95% CI: 0.29–0.88)] as compared to those whose DBP ≥90 mm Hg. Nevertheless, there were no discernible advantages to control SBP and DBP for cardiac events. In patients with OSA, the HR was fairly comparable in the 120-139 mmHg and <120 mmHg groups, and the risk of cerebrovascular event was significantly lower in patients with BP control <140 mmHg compared to the subjects with SBP ≥140 mmHg. Using DBP >90 mmHg as a reference, when DBP <80 mmHg, the risk of a cerebrovascular event might drop by 54%, but the risk of MACCE decreased by 29%. In the non-OSA group, there was a decrease in the incidence of MACCE and cerebrovascular event when SBP control <140 mmHg or DBP control <90 mmHg.

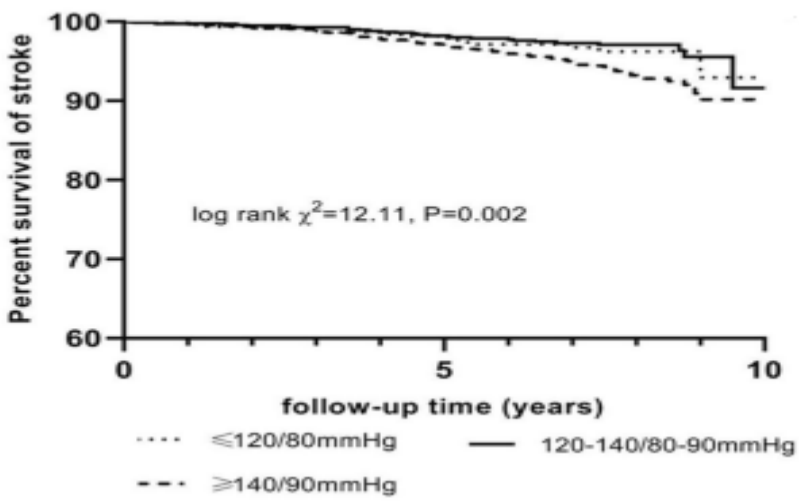

Figure 1. Kaplan-Meier curve of cardiovascular outcomes: Proportion of stroke survivors at various blood pressure management levels

DISCUSSION-The current study suggested that the risk pattern of SBP and DBP may differ by clinical outcomes in hypertension with OSA.[2] Similar to this, Itoga NK et al. showed that based on the clinical outcomes, SBP and DBP have different risk patterns.[3]

CONCLUSION-The current study concluded that anti-hypertensive medication-induced blood pressure regulation at 120–139/80–89 mmHg may have a favourable effect on MACCE incidents.

Anti-hypertensive medication-induced blood pressure regulation at 120-139/80-89 mmHg in the office and at home may have a positive impact on MACCE incidents

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Case report on transcatheter aortic valve infolding resulting in cardiac arrest

INTRODUCTION-Prosthetic valve infolding is an uncommon but serious consequence of self-expanding transcatheter aortic valve implantation (TAVI).[1] A common and accepted treatment for individuals with severe aortic stenosis was transcatheter aortic valve implantation, which was carried out using a balloon-expandable or self-expandable device. Self-expanding valve frames can break down, which might result in complications. An uncommon and probably underreported consequences of these devices is valve infolding, which occurs at an incidence rate of around 3%. Because of this complication was uncommon, there was a lack of information about it, and it is frequently overlooked, which can result in severe cases of paravalvular regurgitation and even hemodynamic compromise.[2] This case report presented a case of a male patient, age 61, who had a history of heart failure with reduced ejection fraction (HFrEF), low-flow, low-gradient severe aortic valve stenosis of a bicuspid aortic valve who presented to undergo TAVI.

CASE PRESENTATION-A 61-year-old man had a history of class III obesity, low-flow, low-gradient severe aortic valve stenosis, obstructive sleep apnea (OSA), diabetes, hypertension, hyperlipidemia, coronary artery disease (CAD) with prior percutaneous coronary intervention (PCI), HFrEF, which was recently estimated to be between 10% and 15%, ventricular tachycardia status following the implantation of an implanted cardioverter-defibrillator (ICD), and coronary artery disease (CAD) with prior percutaneous coronary intervention (PCI). Considering his elevated Society of Thoracic Surgery (STS) score, which indicated a 4.98% chance of operative death, the choice was taken to pursue TAVI rather than surgical aortic valve replacement. His computed aortic valve area (AVA) dropped to 0.72 cm^2

following a dobutamine stress echocardiography performed prior to the operation. Before the procedure, transesophageal echocardiography (TEE) was also used for three-dimensional (3D) imaging. After being prepped and draped, the patient was taken to a hybrid operating room. Following a time-out, anaesthetics were used to sedate and intubate the patient. TEE was done concurrently with the surgery. A right femur cut down tooks place. Using ascending aortography, the coronary cusps were identified. A 34-mm Medtronic Evolut FX self-expanding valve (Minneapolis, MN, USA) was the preferred valve. The artificial valve was deployed over the native valve to 80% at 120 bpm while the heart was being paced, when it was noticed to be excessively deep. The valve was 100% redeployed after being recaptured and repositioned. The repeat valve position was satisfactory, but there was substantial prosthetic valvular regurgitation (Figure 1) and extensive valve infolding. The patient had cardiac arrest and developed step by step more hypotensive. Return of spontaneous circulation (ROSC) was achieved in two minutes after the start of cardiopulmonary resuscitation (CPR). A 24-mm balloon was pushed across the infolded prosthetic valve and post-dilated with rapid pacing after the left femoral artery was accessible. A well-expanded valve was seen by repeat imaging, and there was no discernible gradient, paravalvular leak, or valvular regurgitation. Later that day, the patient's extubation went smoothly, and the next day, patient had been discharged from the hospital in good health.

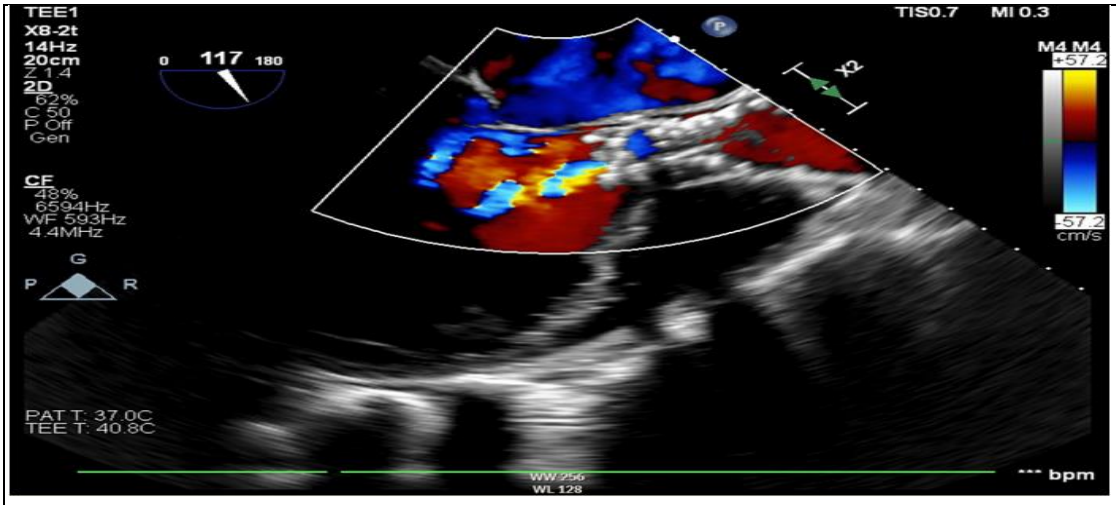

Figure 1. Intraprocedural transesophageal echocardiography(TEE) of the aortic valve with color doppler, mid-esophageal long axis view indicating significant prosthetic valvular regurgitation due to infolding

DISCUSSION-Severe aortic regurgitation is a dreaded consequence of valve infolding, and it can lead to hemodynamic compromise in patients with dilated cardiomyopathy. According to Ancona et al., 29% and 12% of cases had significant aortic regurgitation in TAVI and led to hemodynamic collapse and cardiac arrest.[1]

CONCLUSION-The current study concluded that a rare, subtle, but deadly consequence that needs more attention was prosthetic valvular infolding during TAVI. With a lack of available data, more research was required to identify the risk variables that might put patients

at the risk for this consequence. It was predicted that the prevalence of valvular infolding would rise as the list of TAVI indications grows and the treatment becomes more common.

To minimize patient morbidity and death, structuralists and echocardiographers must exercise extra caution in order to enable early detection and immediate treatment of the condition.

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