

Sudden Cardiac Arrest的死亡 以及救活病例的臨床病理的探討

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為了研究心臟病的急性發作的病態以及救活成功條件，本文對90心臟病入院病例作臨床病理的檢討。Sudden cardiac arrest的臨床診斷是虛血性心臟病40例占45%，心肌症18例占20%，弁膜症13例占14%，其他（包括高血壓心疾患、心肌炎等）19例占21%。發現時的不整脈為心室細動（ventricular fibrillation, VF）占46%、心室頻脈（ventricular tachycardia, VT）占19%，徐脈占35%。三分之一是狹心症以及心肌梗塞的發作。其中26例救活例都是在發作一分鐘內作心臟massage以及症狀比較輕者。64死亡例中32例有2條以上的主冠狀動脈有有意義的狹窄（75%），4例可見動脈內血栓。有2例弁膜症因VF發作而突然死亡，其冠狀動脈無狹窄也無血栓，心肌細胞的收縮帶壞死分布在右心室壁層，可能是冠狀動脈的spasm引起一過性動脈閉塞造成心肌細胞的再貫流障害。總之，急死的原因大部分是與心肌梗塞的發作過程有關，其預防方法尙待檢討。

Key Word: Sudden cardiac arrest, Ventricular fibrillation, Mild contraction bands, Severs contraction bands, contraction bands necrosis
(中山醫學 2: 36~40, 1991)

心臟病的急性發作引起病患很快地死亡的病態，尙有許多不明白的地方⁽¹⁾。一般研究心臟急死，都是針對死亡例為主⁽²⁾。為了更了解心臟急死的實態，本文以個人在日本京都大學留學期間的經驗以及收集的資料為主⁽³⁾，對於死亡例以及救活例作檢討。本文的目的是(1)突然死亡的實際情況、基礎疾病、發生狀況、導因以及經過(2)救活成功的條件檢討(3)特殊解剖例的臨床病理的探討。

對象

為京大病院及其關連病院的住院心臟病患者產生sudden cardiac arrest的90病例（60歲以下35%、60歲以上65%）。Sudden cardiac arrest的定義是由於突然產生心室細動（VF）、心室頻脈（VT）、重度徐脈（asystolic arrest, idioventricular rhythm），產生意識消失以至於血壓、脈博無法測定者。在這裡不包括末期病患。

結果

90例中有42例（47%）對於心肺蘇醒術

(CPR)無反應而死亡。其他48例(53%)在CPR之後血壓在短期間內可以測知,其中22例產生腦死,一週以後死亡。26例最後活著可以出院。Sudden cardiac arrest的臨床診斷是虛血性心臟病40例占45%(狹心症8例、急性心肌梗塞回復期13例、陳舊性心肌梗塞19例),心肌症18例占20%,弁膜症13例占14%,其他(包括高血壓心疾患、心肌炎等)19例占21%。發現時的不整脈為VF占46%,VT占19%,徐脈占35%。產生徐脈不整脈的患者被發現的時間較遲,並無救活者。發生的情況以在室內談話、看電視時為多(74%),睡眠中以及在室外活動時比較少。三分之一是心肌梗塞以及狹心症的突然發作(心電圖診斷)。以被護士以及同室者發現為多。CPR後可以救活而出院的病例都是在心臟發作1分鐘以內由護士開始作心臟massage以及心衰竭症狀比較輕者(NYHA class I~II(46%, N=35)比III(18%, N=55), $p < 0.05$)。以及在作CPR時,作血液gas分析發現acidosis程度較輕度者($pH > 7.3$)。10分鐘以上才開始作心臟massage者並無救活者。救活例中其基礎疾患的類別並無有意義的相差。

病理解剖

64位死亡例中有32例(50%)作解剖,其中虛血性心臟病18例、心肌症4例、弁膜症8例、高血壓性心疾患2例。虛血性心臟病中14例

(77%)有二條以上的冠狀動脈有有意義的狹窄($>75%$),其中有4例可見冠狀動脈內血栓。

【特殊解剖例的臨床病理】

Table所示,2例弁膜症疾患,都是由於突然發生VF、CPR無效而死亡者。解剖時可見遠心性左心室腔擴大,平均心重量607gm,冠狀動脈無有意義的狹窄,無血栓。心肌細胞無一般的凝固壞死(coagulation necrosis, CN),但是可以發現有一種特殊的收縮帶壞死(severe type contraction bands=contraction band necrosis, CBN),而且是局限在右心室壁,所占的面積比率比其他心室壁以及正常無心臟病者($<0.5\%$ area)高出許多(Fig. 1&Table)⁽⁴⁾。CBN與一般在心內膜以及心外膜下所見的心筋細胞的contraction bands(mild type)在形態學上不同(Fig. 1)。

討論

在心內膜以及心外膜下的contraction bands與心肌活體生檢切片在形態學上相似⁽⁵⁾,不是一種細胞的壞死。而心肌細胞的CBN有別於一般的CN,形態學上認為是一種細胞的壞死,其產生的原因被認為是心肌細胞的(1)再灌流傷害(reperfusion injury)^(6,7);(2)

Table. Clinical and Pathological Data of the Two Patients.

Case	Age & Sex	Diagnosis	Cause of Death	% Area of Contraction Bands				Fx
				AS S/M	L S/M	P S/M	RV S/M	
1	26/M	MR	VF	0/0	0/0	0/0	6.1/0.1	+
2	43/M	AR, MSR	VF	0/0.2	0.2/0	0.2/0	6.3/0.2	+

MR: mitral regurgitation, AR: aortic regurgitation, MSR: mitral regurgitation and stenosis, M: mild type, S: severe type, VF: ventricular fibrillation. AS: antero-septal left ventricular (LV) wall, L: lateral LV wall, P: posterior LV wall, RV: right ventricular wall. Fx: Fibrosis was localized in the same ventricular wall where severe contraction bands were seen.

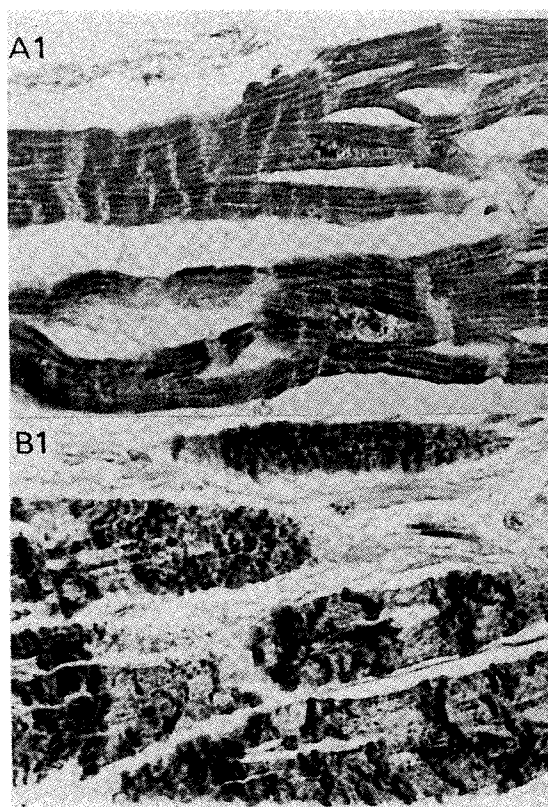


Fig 1. The classification of contraction bands.
 A1: Light microscopic photograph of mild contraction bands. Note regular and wide contraction bands without disruption of myofibril. Granules or vacuoles in the cytoplasm are rarely seen (Masson trichrome stain X400).
 B1: light microscopic photographs of severe contraction bands. Note thin and irregular contraction bands with definite disruption of the myofibril. Granules and vacuoles in the cytoplasm were abundant. (Masson trichrome stain, X400).

代謝性傷害 (metabolic cell injury) catecholamine, corticoid intoxication 以及電解質不平衡等^(8~10); (3)機械性傷害 (mechanical cell injury) 如internal electric shock⁽¹¹⁾; (4)急性

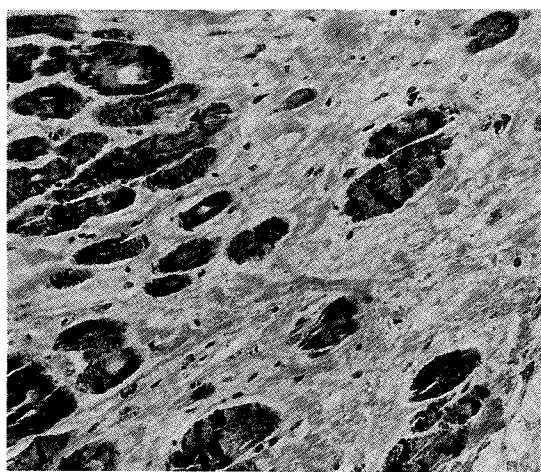


Fig 2. Histological findings of the two exceptional cases. Fibrosis and the myocytes with severe contraction bands are seen in the same tissue area. (Masson trichrome stain, X200).

出血性休克 (acute hemorrhagic shock)⁽¹²⁾。本3例病例的CBN分布在心室壁層，而且周圍有纖維化組織 (Fig·2)，而且臨床上並無上述(2)~(4)的可能。因為冠狀動脈無狹窄，也無血栓，所以斷定是冠狀動脈的spasm引起一時的冠狀動脈閉塞以及再開通所造成的心肌細胞的再灌流傷害，是一種小型無症候性的心肌梗塞 (subclinical infarct)。因為reperfusion arrhythmia的出現，VF產生造成死亡。

結論

(1)心臟病患的急性發病造成死亡的基礎病因中以冠狀動脈病患占45%為最多，但比西洋人 (約70%) 為少。發生的狀況以安靜時為多，三分之一是由於心肌梗塞以及狹心症的突然發生。(2)冠狀動脈硬化造成內腔狹窄以及血栓形成是重要原因以外，spasm也是原因之一。(3)發作時都出現不整脈，心電圖的監視非常重要。(4)CPR成功例都是早期發現，1分鐘以內開始作心臟massage為多，護士的角色非常地

重要。

Sudden cardiac arrest的預防

因為大多數病例的發生原因與急性心肌梗塞發作過程有密切的關係，所以這個問題未解決的話，基本上無法預防。突然死亡的預防除了心臟病的治療與管理以外，high risk病人（運動能力低下、心衰禍嚴重者）要特別注意。另外在醫院以外的地方所發生的情況只有依賴一般人的急救知識以及訓練了。

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Running Title "Sudden Cardiac Arrest in Hospital"

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In order to study the pathogenesis of sudden cardiac arrest of cardiac disease patients, clinicopathological studies were performed in 90 admitted cardiac patients. There were 40 ischemic cardiac patients (45%), 18 cardiomyopathy patients (20%), 13 valvular patients (14%) and 19 others (hypertensive cardiac disease and myocarditis, 21%). The arrhythmia was ventricular fibrillation 46%, ventricular tachycardia 19% and bradycardia 35%. Most of the patients were found at rest. One thirds were due to sudden attacks of angina pectoris and/or myocardial infarction. 26 cases response to cardiac massage done immediately within one minutes after the attack and could be discharged. Their clinical symptoms were less severe (NYHA class I-II/III=46%/18%, $p < 0.05$, $N=35$ and 55 , respectively). Patients died if cardiopulmonary resusciation done more than 10 minutes. Autopsies were done in

32 cases (50%). There were 18 ischemic cardiac diseases. In there, 14 patients had significant coronary artery stenosis in more than two main arteries. Coronary arterial thrombi were found in 4 patients. Interesting, there were two valvular diseased patients died suddenly due to attacks of ventricular fibrillation. At autopsy, contraction bands necrosis surrounding with fibrosis were found in the layers of right ventricular wall. There were no coagulation necrosis. The coronary artery revealed no significant stenosis, nor thrombi found. The pathogenesis was considered to be due to reperfusion injury of myocytes. So, recurrent coronary artery spasms were the possible mechanism. In conclusion, most cause of sudden cardiac death was due to the process of acute myocardial infarction. Its prevention is still controversial.

(CSMJ 2: 36-40, 1991)