Journal of the Tsuruma HEALTH SCI. MED. KANAZAWA UNIV. Vol. 27
137～140 2003

Fibrillation of the articular cartilages of the human sternoclavicular joint. a report of 20 autopsy cases

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KEY WORDS

human sternoclavicular joint, osteoarthrosis, articular cartilage, fibrillation histopathology

Introduction

Recently, the patients who suffer from the osteoarthrosis (OA) increase, with rapid progression of longevity. The pain of the joint progresses rapidly from their forties, and they are prominent especially in the women. The pain of the joint gets behind in the daily life, and causes a big trouble for QOL[1-3]. There are articular capsule, articular cartilage, and synovial membrane as main components of the synovial joint. Nobody feels pain in the cartilage itself because a vessel and a nerve do not exist in the articular cartilage. However the damage of the cartilage proceeds, pain appears in the surrounding of the joint and synovial membrane[4].

It is well known that articular surfaces of human synovial joints are mostly formed by hyaline cartilage. As exceptions to this, surfaces of the sternoclavicular joint (SCJ), acromioclavicular joint and temporomandibular joint are described to be of dense fibrocartilage[5]. But histological details of the human SCJ are still unclear[6-9]. To our best knowledge, there is no histopathological report about the OA changes of the SCJ. The cartilage fibrillation appears in all joints suffered from OA[1-3]. The aim of this study is to evaluate OA changes, especially the degree of cartilage fibrillation of the human SCJ.

Materials and Methods

Human SCJs were collected from twenty autopsy cases, carried out from April 2000 to July 2001, at Kanazawa university graduate school of medicine. Ante-mortem diagnosis of the musculoskeletal disease containing OA was not given in every case. There were 11 males and 9 females and the age ranged from 17 to 88 yr (mean±SD : 65.1±17.9). Clinical features of the 20 cases containing age, sex, main disease, and cause of death are summarized in Table. 1. In each case, SCJ was selected at random from either the left or the right one, and obtained in the routine autopsy technique of thoracotomy. The specimens were fixed in 10% neutral buffered formalin, decalcified in the Plank-Rychlo's solution for 1 day, then cut and sampled from coronal plane of the SCJs and embedded in paraffin. Sections of 3 μm thicknesses were cut from each paraffin block, and were stained with hematoxylin and eosin.

Results

There were forty articular cartilages available because SCJ had the sternal and clavicular surfaces in each. Semi-quantitative analysis was done histologically about the degree of cartilage fibrillation. In 11 cartilages (clavicular surface of case 2, 5, 6, 8, 14, 19, 20 and sternal surface of case 6, 11, 18, 20), most on the surface of cartilage showed apparent fibrillation. In 16 cartilages (clavicular surface of case 10, 12, 15, 16, 17, 18 and sternal surface of case 1, 7, 8, 9, 10, 14, 15, 16, 17, 19), a part on the surface

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Table 1. Clinical Summary of 20 Cases Used in this Study

Case No.	Age	Sex	Sampling Side	Main Disease	Cause of Death
1	67	F	R	SLE	MOF
2	83	M	L	DIC	SL
3	17	M	R	Myelodysplstic Syndromes	Sepsis
4	40	F	L	AML	Pneumonia
5	81	F	L	Mitral Regurgitation	Pneumonia
6	88	F	R	Myocardial Infarction	SL
7	74	F	R	Acute Cardiac Failure	Cardiac Failure
8	76	M	R	Myocardial Infarction	Cardiac Failure
9	66	M	L	Angiosarcoma of Heart	SL
10	81	F	L	Dissecting Aneurysm	SL
11	73	F	R	Infectious Peritonitis	SL
12	58	M	L	Liver cirrhosis	Liver Failure
13	75	M	L	Gastric Carcinoma	Renal Failure
14	49	M	R	Vasculitis Syndrome	unknown
15	71	M	L	Lung Carcinoma	SL
16	58	M	L	Hepatocellular Carcinoma	Liver Failure
17	54	M	R	Hepatocellular Carcinoma	Liver Failure
18	39	F	L	CML	MOF
19	78	F	L	Liver Failure	Liver Failure
20	73	M	R	Carcinomatous Meningitis	SL

M, Male; F, Female; L, Left; R, Right; SL, Same as in the Left
MOF, Multiple Organ Failure; CML, Chronic Myeloid Leukemia
SLE, Systemic Lupus Erythematosus,; AML, Acute Myeloid Leukemia
DIC, Disseminated Intravascular Coagulation

Table 2. Semi-quantitative analysis of the 40 Articular Surfaces

Case No.	Clavicular Surface	Sternal Surface
1	−	+
2	++	−
3	−	−
4	−	−
5	++	−
6	++	++
7	−	+
8	++	+
9	−	+
10	+	+
11	−	++
12	+	−
13	−	−
14	++	+
15	+	+
16	+	+
17	+	+
18	+	++
19	++	+
20	++	++

++, fibrillation is apparent in most on the surface of cartilage
+, fibrillation is observed in a part on the surface of cartilage
−, none

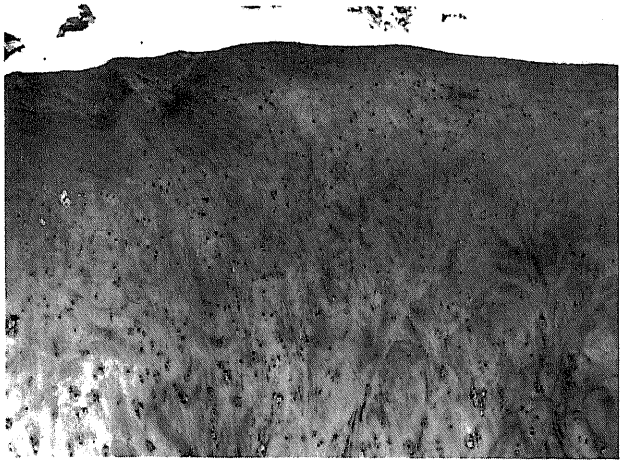

Fig. 1. The example of an articular cartilage of the SCJ without fibrillation.

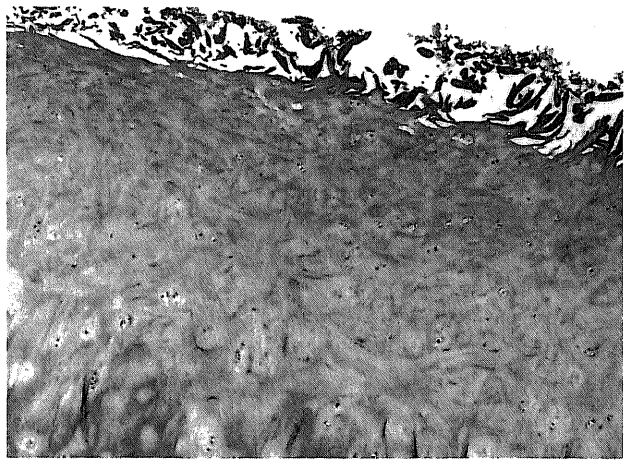

Fig. 2. The example of an articular cartilage of the SCJ with apparent fibrillation.

of the cartilage showed fibrillation. And in the residual 13 cartilages (clavicular surface of case 1, 3, 4, 7, 9, 11, 13 and sternal surface of case 2, 3, 4, 5, 12, 13), apparent fibrillation was not shown (Table. 2). There was no significant difference though the degree of fibrillation showed a tendency to become worse with age.

Discussion

OA is one of the most common and disabling disorders in the general population. It is a slowly progressive disease, characterized morphologically by destruction of cartilage, formation of bone cysts, sclerosis of subchondral bone and presence of osteophytes at the joint margin. Histologically, OA is characterized by significant changes in the composition of cartilage. The earliest structural change in OA is fibrillation at the articular surface[11]. It is possible that OA attacks the joint of the whole body, and that the cartilage fibrillation appears in all joints suffered from OA[1-3]. We deduced that the SCJ is not an exception to it. The results described above support our expectations, though all cases used in this study had no ante-mortem diagnosis of the musculoskeletal disease including OA. It may indicate that the human SCJs are involved frequently in OA without clinical sign, or symptoms of their OA are pretermitted.

As far as we know, this is the first histopathological description of cartilage fibrillation of the human SCJ.

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ヒト胸鎖関節関節軟骨の fibrillation，20剖検症例の報告

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