盛岡赤十字病院紀要　Vol. 26, No. 1, 12－19, 2017

原著論文

術後の慢性心嚢液貯留に合併した化膿性心外膜炎の一例

盛岡赤十字病院　循環器科[1)]・岩手県立久慈病院　循環器科[2)]
肥田　親彦[1)]・朴澤麻衣子[2)]・平野　邦夫[2)]・山屋　昌平[2)]・椋木かれら[2)]

Purulent Pericarditis
-A Case Report of Postoperative Chronic Pericardial Effusion-

Chikahiko Koeda PhD[1)], Maiko Hozawa MD[2)], Kunio Hirano MD[2)]
Shohei Yamaya MD[2)], Karera Mukunoki MD[2)]

Morioka red cross Hospital[1)] Department of Cardiology, Iwate Prefectural Kuji Hospital[2)]

Abstract
Nowadays, purulent pericarditis (PP) has become a rare disease with the increased use of antibiotics in clinical practices. The outcome of PP is poor due to severe infection and haemodynamic failure by the inhibition of the ventricle repletion, and no consensus exists on the treatment. We describe a case of PP with postoperative chronic pericardial effusion changing to the abscess. Following literature, the patient was treated with drainage and intrapericardial injection of urokinase. Inflammation and haemodynamic indexes with the transthoracic echocardiography were improved. We report a valuable case with literature discussion.

Key words： Purulent pericarditis, bacterial translocation, sepsis.

【諸　　言】

化膿性心外膜炎（PP）は心嚢内に膿瘍が形成される疾患で，致死率は20-30%とされる[1]．今回，我々は限局性の慢性心嚢液が血行性に膿瘍化し，ドレナージとウロキナーゼの心嚢内投与を行って良好な転帰を辿った症例を経験したので報告する．

【症　　例】

70歳代の男性．50歳代で感染性心内膜炎，大動脈弁閉鎖不全症，僧房弁狭窄症に対して大動脈弁置換術（機械弁），および僧帽弁形成術を施行された．膿瘍は完全に除去され，良好に経過していたが，術後９年目からComputed tomography（CT）と経胸壁心エコー図検査（TTE）で左室後側面に限局性の慢性心嚢液貯留が認められるようになり，経過観察されていた．以後，慢性心不全，慢性腎不全のため当院を通院していたが，入院日の１週間前から悪寒，腹部膨満感を自覚するようになり来院した．受診時の血圧は108/74mmHg，脈拍数は79bpm，体温は38.4℃で，聴診で心膜ノック音，機械弁閉鎖音が聴取された．座位での頸静脈怒張は12cmで，吸気時にさらに増強した．採血結果はWBC13,400/μL，CRP13.2mg/dL，Na132mEq/L，K 4.7mEq/L，Cl98mEq/L，BUN47.3mg/dL，CRE2.1mg/dL，BNP293.9pg/mLであった．胸部レントゲン写真での心胸郭比は79%で，肺紋理陰影の増強と肋骨横隔

膜角の鈍化が認められた. CT上, 左室後側面の慢性心嚢液のサイズは不変であった. TTE, 経食道心エコー図検査では, 左室後側面の心膜の輝度が高く, 心嚢内部でfibrin depositsが確認された（Figure. 1）. 疣贅や弁下膿瘍などの感染性心内膜炎の所見は認められなかった. 三尖弁と僧帽弁での心室流入血流速波形の呼吸性変動は明らかではなかったが, 両心房は拡大しており, 左室拡張早期に後壁が平坦化するとともに, 心室中隔は左室の拡張に合わせて前方と後方に小刻みに運動していた. 肝静脈では, 逆流波形の増高が認められた（Figure. 2a, b, c）.

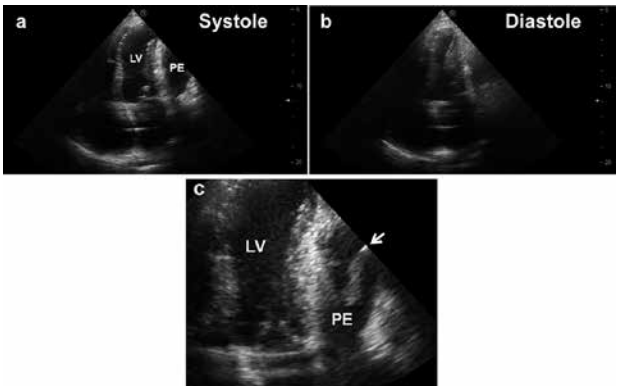

Figure. 1　経胸壁心エコー図での四腔断面像. (a, b) 左室後側面に限局性の心嚢液貯留が認められる. 心嚢内にfibrin depositsが認められる (c).

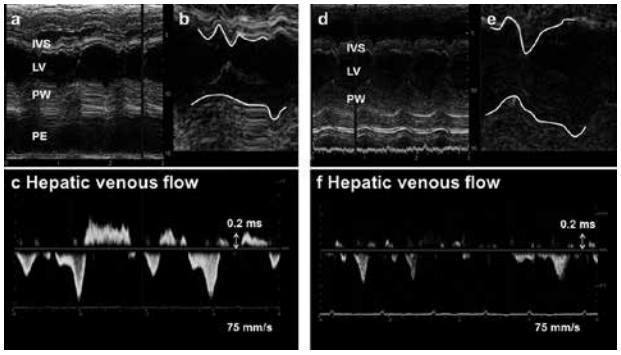

Figure. 2　左室拡張早期に後壁は平坦化し, 心室中隔は左室の拡張に合わせて前方と後方に小刻みに運動していた (b). 肝静脈では逆流波形の増高が認められた (c). d, e, fは同部位でのドレナージ後の波形である. 左室は十分に進展し, 後壁の早期平坦化の所見は消失している. 肝静脈の逆流波は減高した.

入院時から炎症巣が判然とせず, 血液培養を採取したのちにタゾバクタム・アンピシリンで加療を開始して経過を観察した. また, 理学所見とTTEで収縮性心膜炎に合致した所見がみられたことから, 腹部症状は開心術後の収縮性心膜炎による右心不全徴候と判断し, トルバプタンでの体液調節を開始した. 血液培養から腸球菌が検出され, 第８病日からγグロブリンを併用しながら抗菌薬をメロペネムに変更したが, 炎症反応は横ばいに経過した. 第18病日に全身ガリウムシンチグラフィを行ったところ, 心嚢に淡い集積が確認された. 同部位はMRI拡散強調画像, およびT2強調画像で高信号を示し, 臨床経過と併せて膿瘍と考えられた. 第21病日にCTガイド下で膿瘍へのドレナージ術を施行した. 心嚢内より180mlの排液が得られ, その性状は無臭で, カフェオレ様に混濁していた（Figure. 3a）. 心嚢液培養結果はno growthだったが, 排液中の白血球数は256×10^3mmと高値であった. 排膿後に生食での心嚢内洗浄とウロキナーゼの心嚢内投与（12万単位/回, ３回/日, ２日間）を行ったところ, 順調に消炎傾向が得られた. 洗浄液にはフィブリンの沈着物が確認された （Figure. 3b）.

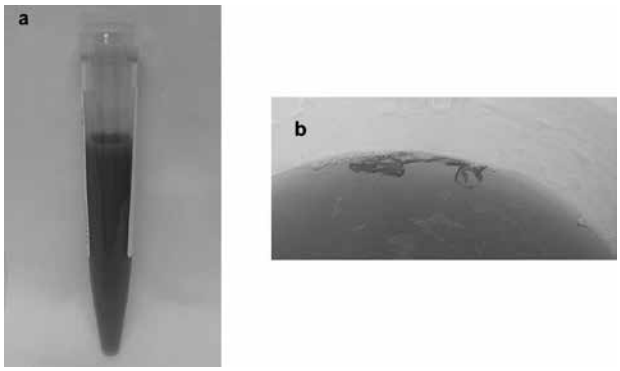

Figure. 3　心嚢内からカフェオレ様に混濁した膿汁が得られた (a). 心嚢内を生理食塩水で洗浄しつつ, ウロキナーゼを心嚢内に投与したところ, フィブリン様の沈着物が吸引された (b).

排膿直後から腹部症状が消失したが, 頸静脈怒張の所見は残存した. TTE所見では, 拡張早期の左室後壁の平坦化所見が消失するとともに, 左室拡張期径は拡がり, 肝静脈血流波の拡張期逆流の減弱が確

認された（Figure.2 d, e, f）. 左室内容積の拡張を反映し, 推定一回拍出量は46mlから86mlに増加した. また, 来院時のBNP値が293.9pg/mLであったのに対し, 慢性期のBNP値は95.1pg/mLに低下した. 第50病日に独歩退院となった.

【考　　察】

PPは予後不良な細菌感染症である. 抗菌薬が普及する以前は, 肺炎を原発とすることが多く, かつ急性収縮性心膜炎の40%を占める疾患であったが, 近年は血行感染が主となり, 非常に稀な疾患となった[1]. 起因菌としてStreptococci, pneumococci, staplylococciが多いとされているが[2], 少数ながらE. coliなども散見される[3]. 本疾患は心タンポナーデに陥る致死的疾患でもあることから, Cronierらは心エコー図での評価が望ましいと報告している[4].

本症例は敗血症の症例で, 全身ガリウムシンチグラフィで心嚢に炎症所見が確認された. 同部位はMRIで膿瘍と合致する結果を示したことから, PPの可能性が強く疑われた. 心嚢液の培養結果は陰性であったが, 心嚢液から多数の炎症性細胞が認められた点や, 過去に21例のPPのうち, 16例がno growthだったとする報告もあるために[5], 培養結果が陰性であってもPPの可能性は否定できない.

PPは血行動態に影響を与える疾患であると同時に, 膿瘍による慢性炎症性疾患としての側面を有する. その治療に関するコンセンサスは十分ではないが, 多くの報告で心嚢内ドレナージと血栓溶解薬の心嚢内投与法が有効だったとされており[1,6], ウロキナーゼの心嚢内投与の有効性を調査したランダム化比較試験では, 心膜内面を線維化させるフィブリンの形成がウロキナーゼ投与群で抑制され, 有意に心膜肥厚と癒着が減り, 収縮性心膜炎の発生率が劇的に抑えられたとされている[6]. 一方で, 血栓溶解薬の心嚢内投与法による重篤な副作用についてはほとんど報告が無い.

収縮性心膜炎のTTE上の特徴として心室への流入血流速波形の呼吸変動や肝静脈血流波の変化, 拡張早期の左室後壁運動の平坦化などが知られているが[7], 本症例では心嚢内ドレナージ後にこれらの諸指標の改善と推定心拍出量の増加が確認された. このことから, 膿瘍を機械的にドレナージすることが, 心室充満を向上させ, 血行動態の安定化と感染巣の除去を一元的に図る最善の方法である可能性があると考えられた. その一方で, 本症例は頸静脈怒張が完全に消失しなかった点から, 既に開心術遠隔期に成立した収縮性心膜炎を下地にしていると考えられ, ウロキナーゼの心嚢内投与による慢性期の収縮性心膜炎予防効果がどの程度本症例の予後に寄与するか慎重に経過をみていく必要がある.

本症例の感染経路に関しては, 心嚢液の培養結果がno growthのため確定に至らないものの, 血液培養にてbacterial translocationの起因菌として最多である腸球菌が血液培養で検出されたこと[8,9], および浮腫を有する慢性心不全患者では腸管免疫能が低下していることから[10], 慢性の右心不全で腸管粘膜の防御機構が破綻し, bacterial translocationの機序で敗血症に陥り, 血行性に限局性心嚢液が膿瘍化したものと考えられた. もともと嚢胞となっていた部位が膿瘍化した例は過去になく, 稀少な症例と思われたため報告する.

【結　　語】

慢性の限局性心嚢液が膿瘍化したPPの症例を経験した. PPに対して心嚢内へのドレナージを行ったところ, 炎症と血行動態の改善がみられ, 良好な転帰を得ることが出来た.

【謝　　辞】

本論文を作成するにあたり, 岩手医科大学医学教育学講座地域医療学分野の伊藤智範先生に添削を行っていただきました. 謝辞とかえさせていただきます.

文　献

1. Augustin P, Desmard M, Mordant P, et al. Clinical review: Intrapericardial fibrinolysis in management of purulent pericarditis. Critical Care 2011 ; 15 : 220.
2. Sagristà-Sauleda J, Barrabés JA, Permanyer-Miralda G, et al .pericarditis: review of a 20-year experience in a general hospital. J Am Coll Cardiol. 1993;22(6):1661-5.
3. Parikh, SV, Memon N, Echols M, et al. Purulent Pericarditis: Report of 2 Cases and Review of the Literature. Medicine 2009;88:52-65.
4. Cronier P, Eugène B, Passefort S, et al. A pneumococcal purulent pericarditis revealing a pneumonia and complicated by an acute cardiac tamponade. Journal of Cardiology Cases. 2012;5:61—64.
5. Liem NT, Tuan T, Dung le A. Thoracoscopic pericardiectomy for purulent pericarditis: experience with 21 cases. J Laparoendosc Adv Surg Tech A. 2006;16:518-521.
6. Cui HB, Chen XY, Cui CC, et al. Prevention of pericardial constriction by transcatheter intrapericardial fibrinolysis with urokinase. Chin Med Sci J. 2005;20(1):5-10.
7. Oh JK, Hatle LK, Seward JB, et al. Diagnostic role of Doppler echocardiography in constrictive pericarditis. J Am Coll Cardiol. 1994;23(1):154-62.
8. Berg RD, Garlington AW. Translocation of certain indigenous bacteria from the gastrointestinal tract to the mesenteric lymph nodes and other organs in a gnotobiotic mouse model. Infect Immun. 1979;23(2):403-11.
9. O' Boyle CJ, MacFie J, Mitchell CJ, et al. Microbiology of bacterial translocation in humans. Gut. 1998;42:29-35.
10. Niebauer J, Volk HD, Kemp M, et al. Endotoxin and immune activation in chronic heart failure : a prospective cohort study. Lancet. 1999 ; 353 (9167) : 1838-42.

Purulent Pericarditis
-A Case Report of Postoperative Chronic Pericardial Effusion-

Chikahiko Koeda PhD, Maiko Hozawa MD※, Kunio Hirano MD※
Shohei Yamaya MD※, Karera Mukunoki MD※

Morioka red cross Hospital　※ Department of Cardiology, Iwate Prefectural Kuji Hospital

Abstract
Nowadays, purulent pericarditis (PP) has become a rare disease with the increased use of antibiotics in clinical practices. The outcome of PP is poor due to severe infection and haemodynamic failure by the inhibition of the ventricle repletion, and no consensus exists on the treatment. We describe a case of PP with postoperative chronic pericardial effusion changing to the abscess. Following literature, the patient was treated with drainage and intrapericardial injection of urokinase. Inflammation and haemodynamic indexes with the transthoracic echocardiography were improved. We report a valuable case with literature discussion.

Key words： Purulent pericarditis, bacterial translocation, sepsis.

Introduction

Purulent pericarditis (PP) is a serious disease characterized with formation of abscess in the pericardial sac with the mortality rate of 20-30 % [1]. We describe a case of PP with localized chronic pericardial effusion changing to the abscess hematogenously, and had a good outcome by drainage and intrapericardial injection of urokinase to pericardial effusion.

Case report

A 70-year-old male was admitted to our hospital for chills and abdomen distension. His past medical history were infective endocarditis, and chronic kidney disease. In his 50’s, he had enforced aortic valve replacement and mitral valve plasty with no surgical complications and the empyema was completely eliminated. Nine years after surgery, he had a localized chronic pericardial effusion in a left ventricle (LV) backside with computed tomography (CT) and transthoracic echocardiography (TTE) without any findings of inflammation.

His clinical characteristics were: height 165 cm, weight 75.5 kg, blood pressure 108/74 mmHg, heart rate 79 beats per minute, body temperature 38.4° C, and heart sound was pericardial knock. The jugular venous distention was 12 cm, and it showed a paradoxical rise during inspiration. Blood test results were as follows: White blood cell 13.4×10^3 /μL, C-reactive protein 13.2 mg/dL, blood urea nitrogen 47.3 mg/dL, serum creatinine 2.1 mg/dL, brain natriuretic peptide (BNP) 293.9 pg/mL. A chest x-ray showed a cardiothoracic ratio of 79 % with lung congestion and pleural fluid. The size of the chronic pericardial fluid of the LV backside was constant with CT. TTE and transesophageal echocardiography revealed a high brightness on posterolateral pericardium, and fibrin deposits in cardiac sac (Fig. 1). The findings of infective endocarditis such as vegetation and abscesses were not found. The respiratory fluctuation of two ventricle inflow velocity patterns was not clear. Two atriums were large, and the posterior wall flattened in mid diastolic LV wall motion. The interventricular septal wall showed the early diastolic notch. The reversal hepatic venous flow in diastolic phase increased (Fig. 2a, b, c).

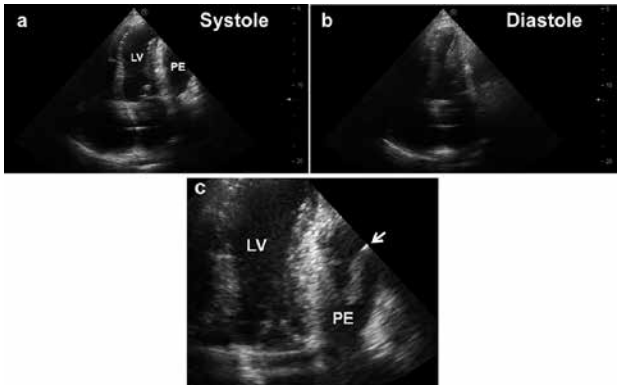

Fig. 1　Four alveus section images with the transthoracic echocardiography (a, b). Localized pericardial effusion (PE) is found in a left ventricle (LV) backside. A fibrin deposits are found in a cardiac sac (c).

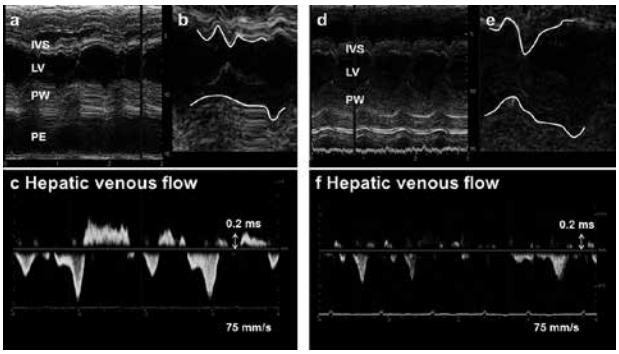

Fig. 2　The interventricular septal wall (IVS wall) showed the early diastolic notch in conjunction with the expansion of left ventricular (LV) (b). The reversal hepatic vein flow in diastolic phase increased (c). Whereas (d), (e), (f) are wave form after the drainage pericardial effusion (PE) in the site. The LV dilatation is enough, and the flattening findings of the posterior wall (PW) in early phase disappear. The hepatic vein flow decreased.

Although an inflammatory focus was not clear after hospitalization, we started intravenous antibiotic treatment with Tazobactam-Piperacillin hydrate after having blood cultures (Fig. 3). In addition, the abdominal symptom was judged as a right heart failure sign due to constrictive pericarditis after the open heart surgery because physical examination and TTE findings were concordant with constrictive pericarditis, and we added Tolvaptan for right heart failure.

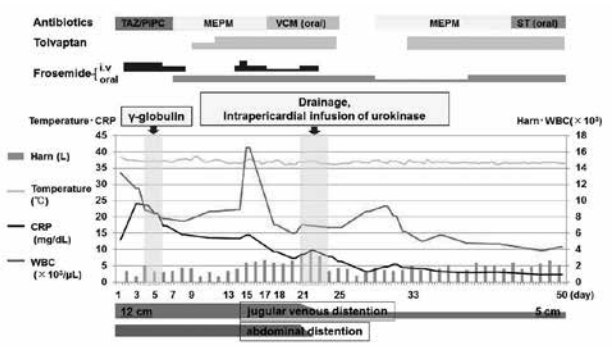

Fig. 3　Clinical course. The inflammatory reaction decreased from the first contact, but it passed without any improvement. After drainage, an inflammation had the tendency to alleviation of fever, and the physical symptom of right heart failure got the improvement. TAZ/PIPC = Tazobactam-Piperacillin hydrate, MEPM = meropenem, VCM = vancomycin, ST = sulfamethoxazole/ trimethoprim.

Enterococcus was detected in blood cultures, and we changed the antibiotics to the meropenem while using a gamma globulin together from the eighth day, but the inflammatory reaction passed without any improvement. On the 18th day, systemic gallium scintigraphy revealed accumulation in cardiac sac. This site showed a high signal with a magnetic resonance imaging (MRI) diffusion weighted image and a T2-weighted image. Hence, it had diagnosis of an abscess with clinical course. The patient underwent drainage of pericardial effusion guided by a CT on the 21st day. After draining 180 ml of effusion, it was odorless and turbidity like a café au lait (Fig. 4a). In this fluid, bacterial culture result showed no growth, but white blood cell was high at 256.0×10^3 /μL. The patient was treated with transcatheter intrapericardial saline and urokinase (120,000 IU each, three times a day for two days) and the tendency for the inflammatory reaction had disappeared. A fibrinous deposit was shown in washings (Fig. 4b).

Soon after the drainage, an abdominal symptom disappeared, but the jugular venous distention was remained. On the TTE findings, the flattening finding of the posterior LV wall in the early phase of diastolic filling had disappeared. Then, the LV diastolic diameter spread, and the diastolic phase reflux wave form in the hepatic vein has decreased

(Fig.2 d, e, f). There was reflected expansion of the capacity in the LV and the estimated stroke volume increased from 46 ml to 86 ml. The BNP value at the chronic stage decreased to 95.1 pg/mL whereas the BNP value at the admission was 293.9 pg/mL, and the patient was discharged on the 50th day.

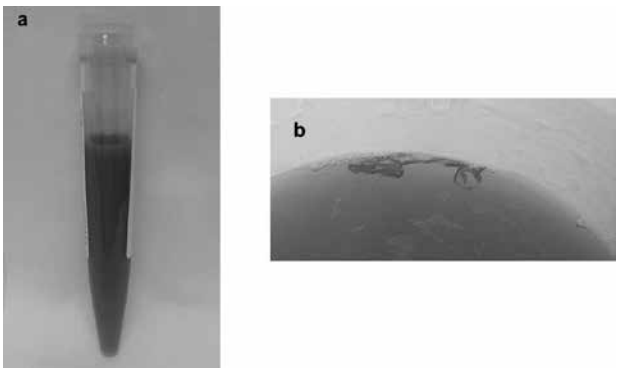

Fig. 4　The coloring matter like a cafe au lait was obtained from the cardiac sac (a). A fibrin deposit was aspirated when the cardiac sac of the patient was rinsed with saline and urokinase irrigation (b).

Discussion

PP is a serious bacterial infection with a high mortality rate. The primary source of PP in the pre-antibiotics era was pneumonia, and accounts for 40 % of acute constrictive pericarditis [1]. In the 1960s, some PP cases were reported frequently as much as a review of 425 cases [2], but PP cases have become rare and hematogenous infection in recent years, and the epidemiological surveys have become less and small-scale. Although the most common microorganisms were streptococci, pneumococci, and staplylococci [3,4], several reports have listed escherichia coli as the causative bacteria [5].

This case was sepsis, and the inflammatory focus was confirmed to a cardiac sac by systemic gallium scintigraphy. Based on the result of MRI which confirmed the features of abscess, we strongly suspected the patient with PP. The culture results of the pericardial fluid were negative, but we should not ignore the likelihood of the PP because several reports have demonstrated that 16 out of 21 PP cases were no growth in the past [6], and there was detected many inflammatory cells in pericardial fluid.

PP affects the hemodynamics and it also has other factors as the chronic inflammatory disease due to the abscess. There is no consensus about its treatment, but many reports have investigated the efficacy of intrapericardial fibrinolysis with urokinase and pericardiocentesis [1,7]. In the controlled trial where cardiac sac of patients were rinsed with saline and they were treated with urokinase irrigation, fibrinous formation to make intrapericardial surface become fibrotic was inhibited in patients who were treated with urokinase, and it significantly reduced the thickness and adhesion of pericardium [7]. On the other hand, there are few reports about serious complication by the intrapericardial fibrinolysis.

The respiratory-dependent change of the hepatic venous flow, ventricle inflow velocity patterns and the flattening of mid and late diastolic LV wall motion were known as the characteristics of constrictive pericarditis [8], and the improvement of those indexes and stroke volume were confirmed in this case. The drainage of an abscess mechanically may be the best method for haemodynamic stabilization with improvement of the ventricular filling and removal of the infection focus. On the other hand, this case was considered as basis on constrictive pericarditis that was already formed for long term after an open heart surgery, because jugular distention did not completely disappear. It is necessary to follow clinical course carefully to see how the protective efficacy of the prevention of pericardial constriction for chronic phase by the intrapericardial fibrinolysis with urokinase affects the prognosis of this case.

Although the primary source of infection in this case was undetected because the culture results of the pericardial fluid were no growth, the enterococcus which was the main pathogen as origin bacteria of bacterial translocation was detected by blood cultures [9,10]. Furthermore, intestinal immunocompetence decreases with the patients of chronic heart failure with edema [11]. Thus, immunomechanism of the intestinal mucosa in this case has failed due to chronic right heart failure and fell into sepsis by a mechanism of bacterial translocation, and it was considered that a localized pericardial fluid caused the abscess hematogenously. Since there is no previous paper

that reported chronic pericardial effusion changing to the abscess, this investigation was considered as a rare case.

In conclusion, we experienced a case of PP with localized chronic pericardial effusion that caused the abscess. After performing the drainage and injection of urokinase in the cardiac sac for PP, inflammation and haemodynamic system improved, and we were able to obtain a good outcome.

Acknowledgement

We are deeply grateful to Cardiovascular Research Center, Division of Cardiology, Department of Internal Medicine, and Division of Community Medicine, Department of Medical Education, Iwate Medical University, Tomonori Itoh M.D., FACC, FJCC for correction and advice.

Conflict of interest

The authors declare that they have no conflict of interest.

References

1. Augustin P, Desmard M, Mordant P, et al. (2011) Clinical review: Intrapericardial fibrinolysis in management of purulent pericarditis. Critical Care 15 : 220
2. Boyle JD, Pearce ML, Guze LB (1961) Purulent pericarditis: review of literature and report of eleven cases. Medicine 40:119-144
3. Sagristà-Sauleda J, Barrabés JA, Permanyer-Miralda G, et al. (1993) Purulent pericarditis: review of a 20-year experience in a general hospital. J Am Coll Cardiol 22:1661-5
4. Hirokazu Tokuyasu, Yuhei Saitoh, Tomoya Harada, et al. (2009) Purulent Pericarditis Caused by the Streptococcus milleri Group:A Case Report and Review of the Literature. Inter Med 48:1073-1078
5. Parikh, SV, Memon N, Echols M, et al. (2009) Purulent Pericarditis: Report of 2 Cases and Review of the Literature. Medicine 88:52-65
6. Liem NT, Tuan T, Dung le A (2006) Thoracoscopic pericardiectomy for purulent pericarditis: experience with 21 cases. J Laparoendosc Adv Surg Tech A 16:518-521
7. Cui HB, Chen XY, Cui CC, et al. (2005) Prevention of pericardial constriction by transcatheter intrapericardial fibrinolysis with urokinase. Chin Med Sci J 20:5-10
8. Oh JK, Hatle LK, Seward JB, et al. (1994) Diagnostic role of Doppler echocardiography in constrictive pericarditis. J Am Coll Cardiol 23:154-162
9. Berg RD, Garlington AW (1979) Translocation of certain indigenous bacteria from the gastrointestinal tract to the mesenteric lymph nodes and other organs in a gnotobiotic mouse model. Infect Immun 23:403-11
10. O' Boyle CJ, MacFie J, Mitchell CJ, et al. (1998) Microbiology of bacterial translocation in humans. Gut 42:29-35
11. Niebauer J, Volk HD, Kemp M, et al. (1999) Endotoxin and immune activation in chronic heart failure: a prospective cohort study. Lancet 353:1838-1842