Pseudo-Knock Sound in a Patient with Nephrotic Syndrome and Massive Ascites

Hirofumi Ichiyasu, M.D., Shozo Nabeyama, M.D., Masayuki Takasugi, M.D., Yasuhide Nakashima, M.D., and Akio Kuroiwa, M.D.

SUMMARY

A patient with markedly elevated diaphragm due to massive ascites secondary to nephrotic syndrome demonstrated an intense early diastolic sound with low- and medium-pitch. This abnormal sound coincided closely with the "D" point of the anterior mitral valve echogram. This sound remarkably diminished in intensity during inspiration with lowering of diaphragm, and after removing ascites it completely disappeared. Noninvasive study with phonoechocardiograms showed neither constrictive pericarditis nor large pericardial effusion. These findings lead us to believe that the sound may be related to an abnormal ventricular recoil striking the extracardiac structures at the end of the isovolumetric relaxation time. To our knowledge, the fact that the elevated diaphragm itself can produce an early diastolic sound ("pseudo-knock sound") has not been previously reported.

Additional Indexing Words:
Early diastolic sound  Phonoechocardiogram  Jugular venous pulse

One of the less well recognized causes of nephrotic syndrome is the mechanical effect of increased pressure in the renal veins. Reversible nephrotic syndrome with increased venous pressure secondary to constrictive pericarditis has been reported.1) A characteristic early diastolic sound or "pericardial knock sound" is present in many patients with constrictive pericarditis.5) Therefore, it is reasonable to consider that, when an abnormal early diastolic sound is found in a patient with nephrotic syndrome, constrictive pericarditis may be complicated or the primary cause.

We have recently experienced a patient with nephrotic syndrome with massive ascites who demonstrated the abnormal loud early diastolic sound.

From the Second Department of Internal Medicine, School of Medicine, University of Occupational and Environmental Health, Yahata-nishi-ku, Kitakyushu-shi 807, Japan.

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We present here that its abnormal early diastolic sound is not a so-called "knock sound" of constrictive pericarditis, but a "pseudo-knock sound" caused by an abnormally elevated diaphragm. To our knowledge, this is the first case report to discuss that massive ascites can produce the abnormal early diastolic sound in a peculiar case.

**Case Report**

A 48-year-old man was admitted to the UOEH Hospital, Japan, for treatment of ascites. He had 1-year history of nephrotic syndrome having a treatment with diuretics. The patient complained of dyspnea even at rest. There was a history of B type hepatitis.

Physical examination revealed massive ascites, and lung-liver border was

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*Fig. 1. Chest X-ray film showing marked elevation of diaphragm (A). There are no cardiomegaly and no pulmonary congestion. Electrocardiogram of standard leads (B). Low QRS amplitude is present in limb leads.*
on the 4th rib along the midclavicular line. Body weight was 62.5 Kg and body height was 158 cm. There was peripheral edema of lower extremities. The blood pressure was 120/68 mmHg and the pulse rate was 76 beats per minute and regular. The carotid pulses were normally palpable and pulsus paradoxus was not found. Jugular vein was slightly distended and monophasic jugular pulsation which did not show sharp and deep descent was noted. There was no Kussmaul’s sign.

Cardiac examination revealed that apical impulse was not palpated. The first heart sound was normal and second heart sound was physiologically split at the pulmonic area. A fourth or third heart-sound gallop was not audible. A grade 1/6 systolic ejection murmur which peaked in mid-systole was audible at the left sternal border. Immediately following the second sound, a loud and sharp sound with low and medium-pitch was heard around

![Fig. 2. Phonocardiogram showing an abnormal early diastolic sound (X) at apex. The aortic component of the second heart sound (IIA) is followed by the early diastolic sound (X) with the interval of 70 msec. The interval of IIA-IIP is 60 msec. 2L=the second intercostal space along the left sternal border; L=low frequency; M1 (M2)=medium frequency; H=high frequency phonocardiogram; IIP=pulmonic component of the second heart sound.](image-url)
the apex. This was accentuated during expiration.

A chest X-ray film (Fig. 1-A) showed atelectasis in both lower lobes with bilateral pleural effusion and elevation of both right and left diaphragms: the heart appeared normal in size with normal pulmonary vascularity. No calcification of pericardium was present. The electrocardiogram (Fig. 1-B) demonstrated a sinus bradycardia at a rate of 56, with normal conduction times. Low QRS amplitude in limb leads was present.

Renal function was impaired; the serum urea nitrogen was 58 mg per 100 ml; creatinine was 2.0 mg per 100 ml; endogenous creatinine clearance was 33 ml/min. There was 4 Gm of protein in a 24-hour urine specimen. Needle biopsy of the kidney showed membranoproliferative glomerulonephritis. Liver function test indicated hepatic cirrhosis; GOT, 74 IU/L; GPT, 35 IU/L; total protein, 5.4 Gm/100 ml; albumin, 1.7 Gm/100 ml; γ-globulin, 33.2%; TTT, 17.2 K-U; ZTT, 23.2 K-U; ICG, 20% (15 min). An ultrasound study of the liver disclosed nodular granulation of liver with assive ascites.

![Fig. 3. Jugular venous pulses (JVP). A: Jugular venous pulse before removal of ascites shows a monophasic pattern. This pattern is not completely similar to that of vena caval pressure in cardiac tamponade. B: Jugular venous pulse tracing after removal of ascites. There is a small Y descent. The early diastolic sound disappears, and IIP still remains. An early diastolic murmur (DM) develops because of high blood pressure (160/90 mmHg).](image-url)
Fig. 4. M-mode echocardiogram. There is neither mitral valve prolapse (B) nor massive pericardial effusion (C).

Fig. 5. Two-dimensional echocardiogram. There is no massive pericardial effusion.
Phonocardiogram

Phonocardiogram showed wide splitting of the first and second heart sounds (Fig. 2). The second heart sound presented the normal respiratory splitting (40–60 msec). An intense low and medium-pitched sound, "pseudo-knock sound" (X) at apex was displayed at a variable interval after second heart sounds (70–90 msec). The Q-X intervals average 420–440 msec. The intensity of pseudo-knock sound diminished with inspiration and it disappeared with deep inspiration. The apex cardiogram was unable to record. The jugular pulse was of monophasic pattern. However, this did not resemble the pattern of vena caval pressure in cardiac tamponade1) (Fig. 3-A).

Phonoechocardiogram

A M-mode echogram (Fig. 4) did not reveal any sudden anterior displacement followed by a brisk posterior rebound which usually appeared in constrictive pericarditis.8),9) The posterior left ventricular endocardium did not exhibit flat motion during diastole. The presence of mild echo-free space behind the left ventricular posterior wall was suggested on M-mode echogram. However, it was not a definite sign of pericardial effusion and it was difficult to differentiate from pleural effusion. Abnormal motion of various

![Fig. 6. Phonoechocardiogram showing that the early diastolic sounds coincide with the D point of anterior mitral valve.](image-url)
structures as a consequence of the free swing of the heart which was characteristic finding in massive pericardial effusion\(^\text{10}\) was not noted. There were no findings of mitral prolapse. A two-dimensional echogram (Fig. 5) confirmed these findings of the M-mode echogram. Pseudo-knock sound (X) coincided with the point of D of anterior mitral leaflet (before E point) on phonoechocardiogram (Fig. 6). This abnormal sound also occurred around the point of D of tricuspid leaflet.

Clinical course

Clinical diagnosis was primary nephrotic syndrome with hepatic cirrhosis. The patient received diuretics and human plasma protein fraction. One month after admission, his body weight decreased from 62.5 to 53.0 Kg and his ascites remarkably decreased. Chest X-ray film showed that elevated diaphragm was lowered by one intercostal space. The patient became free of dyspnea and peripheral edema. Liver function test showed no remarkable change.

On auscultation, the early diastolic sound ("pseudo-knock sound") disappeared completely. This was confirmed by serial phonocardiograms (Fig. 2-B). An early diastolic murmur developed at apex when the blood pressure of the patient was high (160/90 mmHg) (Fig. 2-B). The jugular pulse showed that Y descent slightly developed compared with last jugular pulse tracing (Fig. 2-A). The M-mode echogram still presented a small echo-free space behind left ventricular wall.

Discussion

The loud early diastolic sound in our patient occurred at the time of D points of anterior mitral valve. The opening snaps from either side of the heart occur at exactly the time of maximal opening of the respective atrioventricular valve in early diastole.\(^\text{11}\) The third heart sound occurs after the E point of the anterior mitral valve.\(^\text{12}\) Therefore, it is obvious that this abnormal sound in our patient is neither the opening snap nor the third heart sound.

The early diastolic sound in our patient is very similar to the "pericardial knock sound" of constrictive pericarditis. However, it is unlikely that this sound in our patient originates from pericardial sac because our patient has no clinical and phonoechocardiographical signs of constrictive pericarditis. Jugular venous pulse did not disclose a pattern of deep X or Y descent characterizing constrictive pericarditis.\(^\text{13}\) This view is supported by the evidence of disappearance of this abnormal sound after decrease in
volume of ascites with lowering of diaphragm. An unusual, high-pitched, early diastolic sound in a patient with a large chronic pericardial effusion has recently been reported.\textsuperscript{14,15} Sakamoto et al\textsuperscript{14} postulated that pulse and sound were due to early diastolic filling analogous to the pulse and sound of pericardial constriction. On the other hand, Bonner et al\textsuperscript{15} believe that the sound and pulse are caused by the ballistic effect of the apex of the heart swinging up and striking the anterior pericardium and chest wall.

In our patient, large pericardial effusion was absent. There should be another factor responsible for early diastolic sound. We believe that this abnormal sound originated from extracardiac structure. This unusual early diastolic sound occurred at the end of isovolumetric relaxation time (D point of anterior mitral valve). It seems that the mechanism of the production of this sound is not the same as the "pericardial knock sound" in pericardial constriction with sudden restriction of ventricular filling. In early diastole, ventricular recoil may be restricted between elevated diaphragm and lungs in our patient. This possible mechanical abnormalities may produce the unusual ventricular rotation during isovolumetric relaxation time. We assume that some parts of cardiac structure struck the diaphragm during early diastole. Although the real mode of production of this sound remains obscure, this possible hypothesis may be supported by the fact that this abnormal sound disappeared during inspiration when the diaphragm positioned at the lower level.

Therefore, we would propose, even if there was no massive pericardial effusion nor pericardial constriction, the extracardiac compression, for example, by markedly elevated diaphragm, could produce the unusual early diastolic sound. We should keep in mind that there were more than 3 factors contributing to the production of the early diastolic sounds in nephrotic syndrome. Firstly, it is pericardial constriction, secondly, large pericardial effusion and the third factor is massive ascites.

\textbf{REFERENCES}


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