Case Reports

Three Cases of Traumatic Pericarditis Following Transseptal Left Heart Catheterization: with Special Reference to Atrial Injury Current

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The chance of inadvertent puncture of the free wall of the right atrium upon transseptal left heart catheterization seems to be not so rare. So far as we know, however, the traumatic pericarditis attributable to the puncture of the right atrial wall solely by the transseptal needle has not been reported. The changes of the P-Ta segment of electrocardiogram produced by atrial injury current in cases of atrial infarction have been noted from old.1,2) Whereas, the tracing of atrial injury current caused by puncture of the right atrial wall during the procedure has not been reported.

The purpose of this paper is to describe 3 cases of traumatic pericarditis following the inadvertent puncture of the free wall of the right atrium by the transseptal needle, in which the recording of atrial injury current was made during their course. The procedure modified by Brockenbrough and associates3,4) was employed in all 3 cases.

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Case 1. A 58-year-old diabetic, hypertensive man with a 8-year history of xanthoma of eyelids was admitted complaining of diplopia with diminution of vision for the last 2 months. On admission, the blood pressure was 172/96 mm. Hg; the pulse rate was 60 per min. and regular; cardiothoracic ratio was 0.56. Funduscopic examination revealed hypertensive and sclerotic changes of grade 2 with 2 small spots of rather old retinal hemorrhage. The electrocardiogram showed normal contour of the S-T segment and T wave. Urinalysis was negative except glycosuria. Serum total cholesterol was 265 mg./100 ml., and fasting blood sugar level 146 mg./100 ml. Renal function showed a moderate impairment; RBF was 647 ml./min., RPF 343 ml./min., GFR 93.7 ml./min., and FF 0.27.

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Transseptal left heart catheterization was carried out on Oct. 22, 1962. Several minutes after the presumed puncture of the atrial septum by the transseptal needle resulted in failure twice, a marked depression of the P-Ta segment in lead II appeared, which continued about 15 min. (Fig. 1-B). Twenty minutes after the last puncture, the patient complained of oppression of the chest with cold sweat and bradycardia of 52 per min., and developed shock with a fall of systolic blood pressure to 35 mm. Hg. The electrocardiogram at that time revealed transient left

Fig. 1. Lead II of the electrocardiogram in case 1.
A: Immediately before the transseptal left heart catheterization. B and C: Several minutes, and 25 min. after the last puncture, respectively.

Fig. 2. Electrocardiogram in the same case as in Fig. 1.
A: Before the transseptal left heart catheterization. B, C, and D: One hour, 3 days, and 15 days after the puncture, respectively.
bundle branch block pattern (Fig. 1-C). Following the recovery of normal conduction, S-T depression in leads I, II, III and aVF was observed for many hours (Fig. 2-B). Intravenous drip infusion of norepinephrine was started and 20 mg. of prednisolone was administered intravenously. The shock continued for 9 days and required intravenous drip infusion of norepinephrine for 2 days. Leukocytosis of 15,300, slight fever and tachycardia were present for a few days following the puncture. Upward S-T displacement in electrocardiogram occurred on Oct. 23, and persisted for 10 days; on Oct. 24, the electrocardiogram revealed transient atrial fibrillation and increased elevation of the S-T segment in leads I, II, aVL, aVF and V_6 associated with the evidence of slight cardiac enlargement (Fig. 2-C); on Oct. 29, the erythrocyte sedimentation rate became elevated to 84 mm. in one hour; serum glutamic oxaloacetic transaminase levels were less than 34 units. Finally a slight inversion of the terminal portion of the T wave developed in V_3 through V_5 on Nov. 6 (Fig. 2-D), and then the patient improved gradually.

Case 2. A 23-year-old man diagnosed to have aortic insufficiency with a 5-year history of palpitation, shortness of breath and anginal pain was catheterized by transseptal procedure on Dec. 17, 1962. Before the procedure, the blood pressure was 165/0 mm. Hg; the pulse rate was 82 per min. and regular; cardiothoracic ratio was 0.49. The patient had neither basilar rales, liver swelling, pitting edema, nor any history of treatment with digitalis.

One hour after 4 unsuccessful punctures of the atrial septum, the patient developed hypotension of 80/0 mm. Hg and diminution of pulse rate to 74 per min. associated with chest pain on inspiration, nausea, pallor and cold sweat. Though 20 min. later the blood pressure recovered to 140/0 mm. Hg, chest pain with anxiety remained severe. Two and a half hours after the last puncture, the electrocardiogram revealed a downward displacement of the S-T segment in leads I, V_4 and V_5, and low or biphasic T wave in V_3 through V_6 (Fig. 3-B). As an interesting finding, depression of P-Ta segment was noted in V_1 through V_6. The next day, pericardial friction rub occurred in association with slight fever of 37.7°C, leukocytosis of 14,500, erythrocyte sedimentation rate of 17 mm. in one hour, and tachycardia of 100 per min. The electrocardiogram showed upward S-T displacement in leads I, II, III and precordial leads (Fig. 3-C). The P-Ta segment was depressed distinctly in leads I, II, aVF and V_4 through V_6 and slightly in leads III, V_2 and V_3. X-ray findings of the heart and lungs were negative; serum glutamic oxaloacetic transaminase levels were less than 26 units. Finally a slight inversion of the T wave in V_4 through V_6 developed 22 hours after the last puncture (Fig. 3-D). From Dec. 18, intramuscular administration of prednisolone in a daily dose of 20-10 mg. was continued for 18 days with improvement.

Case 3. A 30-year-old woman, who was diagnosed as mitral stenosis complaining of palpitation and shortness of breath of one year's duration was catheterized by transseptal procedure on Feb. 4, 1963. She had no history of rheumatic fever, pitting edema and orthopnea. Before the procedure, blood pressure was 100/55 mm. Hg; pulse rate was 114 per min. and regular; cardiothoracic ratio was 0.56. The patient had liver swelling of 3 cm. below the right costal margin, but neither basilar rales, pitting edema nor previous treatment with digitalis.

Immediately after the unsuccessful puncture of the atrial septum, the patient began to complain of chest pain on inspiration. One and a half hour later, the
Fig. 3. Electrocardiogram in case 2.
A: Before the transseptal left heart catheterization. B, C, and D: Two and a half hour, 15 hours and 22 hours after the puncture, respectively.

patient developed hypotension and shock associated with bradycardia, cold sweat, pallor, nausea and vomiting. The drip infusion of norepinephrine and 40 mg. of prednisolone was administered intravenously. Four hours after the puncture, pericardial friction rub was heard and transient nodal rhythm developed (Fig. 4-B). Eight hours after the puncture, T wave became lowered in leads I, II, aVF and V3 through V6, and the upward S-T displacement was recorded. The next day, leukocytosis of 13,000 was present without abnormality of chest roentgenogram, body temperature and erythrocyte sedimentation rate. The administration of
prednisolone in a daily dose of 40 mg. was continued. On Feb. 6, the upward S-T displacement in leads II, III, aVF and V2 through V6 became maximum, and the height of T wave in the same leads began to increase (Fig. 4-C). As an interesting finding, depression of P-Ta segment in leads I, II, aVF and V2 through V6 was noted. Within several days, chest pain and pericardial friction rub completely disappeared. On Feb. 12, the upward S-T displacement decreased and, on the other hand, the change of T wave in leads II, III, aVF and V2 through V6 became maximum without any abnormality of blood potassium levels (Fig. 4-D). At the time of commissurotomy on Feb. 25, a small amount of blood was found in the pericardial space facing the anterior wall of the right atrium.

**Discussion**

The complication of shock and traumatic pericarditis caused by inadvertent puncture of the free wall of the right atrium solely by the
transseptal needle has not been reported so far as we know. However, the chance of the puncture of the right atrial free wall by the transseptal needle is not so rare. Adrouny and associates, who have emphasized the appearance of the unique pressure curve for the diagnosis of the puncture of the right atrial free wall by the transseptal needle, reported such inadvertent puncture in 9 instances out of 191 consecutive transseptal catheterization. Fortunately, as observed in their report, the puncture of the right atrial free wall by the transseptal needle seems to be usually uneventful. McIntosh and associates reported one case, in which air was aspirated through the transseptal needle penetrating the atrial wall, and yet no ill effects occurred after the withdrawal of the needle. Further support is contributed by the findings that even several patients who had undergone uneventful transseptal catheterization were found to have a small amount of blood in the pericardial space at the time of operation. Our cases which developed shock and traumatic pericarditis after the puncture of the atrial free wall may be rather exceptional. Shock in our 3 cases was produced probably by autonomic imbalance resulting from vasovagal reflex.

Gordon and associates, who made the recording of endocardial electrocardiograms through the Ross needle during transseptal puncture of the left atrium, observed that surface leads failed to change even when the S-Ta elevations of atrial injury were huge. Adrouny and associates reported 2 cases of hemopericardium caused by the entry of the Lehman catheter into the pericardial space through the atrial wall. They, however, did not refer to atrial injury current. Though the change of the P-R segment in case 1 might be due to abnormality of the P wave, yet it is rather considered to be due to depression of the P-Ta segment representing atrial injury current attributable to the puncture. The clinical and experimental observations have revealed that lesions of the left atrium produce a positive displacement of P-R segment in lead I and lesions of the right atrium give rise to a negative displacement in the same lead, and that in both cases this segment is displaced downward in lead II and III. Liu and associates have stated in their review that, when plotted vectorially, P-Ta segment is directed toward the area of atrial infarction. In case 1 the localization of injury by means of electrocardiogram was difficult because only the record of lead II was available.

The depression of P-Ta segment in case 2 and 3, which appeared later with symptoms and signs of pericarditis, was caused possibly by subepicardial atrial lesions following pericarditis. Especially in case 2 the downward displacement of P-R segment seemed to result not merely from sympathetic effects of tachycardia but mainly from atrial injury current, because, as lately mentioned by Tranchesi and associates, the
atrial J point was not maintained in the isoelectric line but displaced downward, particularly in leads I, aVF, V4 and V5. In pericarditis produced by the puncture of the right atrial free wall, a high incidence of the displacement of P-Ta segment is possibly predicted. This may be due to the selective involvement of the right atrium.

**SUMMARY**

Three cases which developed shock and traumatic pericarditis following the inadvertent puncture of the free wall of the right atrium by the transseptal needle are described. All cases showed atrial injury current which was produced presumably by the puncture of the right atrial free wall in one case and by subepicardial atrial lesions following pericarditis in the other 2 cases.

**REFERENCES**