Changes in P-R Interval Immediately Before, During, and After Acute Myocardial Infarction

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Summary

A 48 years old male developed acute cardiac pain manifested by prolonged P-R interval as the only electrocardiographic sign. Subsequently the electrocardiogram evolved an acute myocardial infarction with a normal P-R interval. Immediately thereafter prolonged P-R interval reappeared to become established even after digitalis was long discontinued.

Additional Indexing Words:
First degree A-V block  Acute myocardial infarction  Coronary artery disease  Angina pectoris

The importance of prolonged P-R interval in the diagnosis of coronary artery disease was emphasized recently by Calleja and Guerrero. They pointed out that coronary artery disease with angina alone is accountable for 32% of all causes of prolonged P-R interval.

The case herein reported illustrates prolonged P-R interval as the only abnormality in the first electrocardiogram taken after the onset of cardiac pain. Subsequent tracings revealed the development of abnormalities universally accepted as indicative of acute myocardial infarction. However, during the electrocardiographic evolution the P-R interval became normal then followed by permanent prolongation.

Case Report

D.S., 48-year-old, machine operator was brought to St. Luke’s hospital on July 6, 1973 because of substernal chest pain starting a few minutes before admission. The pain was continuous with radiation over the right anterior chest accompanied by cold clammy perspiration and dypsnea persisting up to the time of examination at the emergency room. He was given stat. Demerol 30 mg i.m. and 1 tablet of sublingual peritrate with nitroglycerine. Stat. electrocardiogram showed prolonged P-R interval of 0.24 sec. The ST-segments and T waves were normal. There were no abnormal Q waves.

He denied any previous cardiac pain and had no knowledge of hypertension,

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diabetes, hypercholesterolemia, and hyperuricemia. He smoked 1 pack/day for several years.

Physical examination showed a well developed male in acute pain and dypsnea. The blood pressure was 120/80; temperature, 36.5°C; respiration, 22/min; pulse, 70/min. The heart was regular with no gross cardiomegaly. There were no murmurs, thrill, gallop, or rub. The lungs were clear. There were no abnormalities in the abdomen and no leg edema. The neurologic examination was normal.

Serial serum glutamic oxaloacetic transaminase (SGOT) determinations showed on July 7, 1973, 820 units; July 8, 164 units; July 11, 38 units, and August 2, 25 units. Serial electrocardiograms revealed evolutionary changes of acute antero-lateral myocardial infarction.

During the first 3 days in the coronary care unit he had recurrent cardiac pain relieved by Demerol. Pericardial friction rub was heard on the second day but disappeared on the third day. Thereafter his course remained uneventful until he was discharged on July 19, 1973. Therapy consisted of low salt diet, lanoxin 0.25 mg O.D. from July 9 to July 18, glucose-insulin-potassium solution (20 units regular insulin, 20 mEq potassium chloride in 1 L 10% dextrose in water) every 12 hours for 5 days; inderal (propranolol hydrochloride) 20 mg bid from July 12 and peritrate 10 mg from July 10 to the day he was discharged.

DISCUSSION

The only electrocardiographic abnormality in an otherwise normal electrocardiogram was the prolonged P-R interval (Fig. 1). The onset of pain a few minutes before admission and the persistence of the same while the electrocardiogram was taken in the emergency room are fortuitous in establishing a relationship between cardiac pain and prolongation of the P-R interval.

It is of interest that subsequent tracings revealed the development of acute antero-lateral myocardial infarction with no prolongation of the P-R interval in the next 48 hours (July 7, 8) immediately following the acute onset

![Fig. 1. The P-R interval is 0.24 sec. The rest of the tracing is normal. (July 6, '73).](image)
of cardiac pain (Fig. 2). This normalization of the P-R interval has no obvious explanation.

The tracing on July 16 while on digitalis showed first degree A-V block (Fig. 3). Digitalis was discontinued on July 19 when he was discharged from the hospital.

Fig. 2a. Development of acute antero-lateral myocardial infarction. (July 7, '73).

Fig. 2b. Further evolution of acute antero-lateral myocardial infarction. (July 8, '73). Both tracings have normal P-R interval.

Fig. 3. P-R interval again prolonged. Lanoxin started on July 9, 1973 (see text). (July 16, '73).
The report of Calleja and Guerrero\textsuperscript{1)} showed that coronary heart disease with infarction was responsible for 20\% while coronary heart disease with angina accounted for 32\% of all causes of prolonged P-R interval studied. It appears that the present case clearly illustrates the involvement of the P-R interval in both stages of coronary artery disease.

Digitalis may have played a part in the reappearance of the first degree A-V block but the persistence of this abnormality in 2 other records, August

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig4a.png}
\caption{P-R interval remains prolonged. Lanoxin stopped (August 13: top, September 24, '73: bottom) on July 19, 1973.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig5.png}
\caption{Residual changes of antero-lateral myocardial infarction with prolonged P-R interval. (November 20, '73)}
\end{figure}
13, 1973 and September 24 (Fig. 4), when the patient was no longer under digitalis negates the participation of the latter as an important causal factor. In addition, with or without digitalis the length of the prolongation of the P-R interval remained the same. Five months after the acute cardiac pain the electrocardiogram on November 20 (Fig. 5) showed an old antero-lateral myocardial infarction and a prolonged P-R interval.

References