Transient appearance of antegrade conduction via an AV accessory pathway caused by atrial fibrillation in a patient with intermittent Wolff-Parkinson-White syndrome

S Niwano, Y Kitano, M Moriguchi, T Izumi

Abstract
A 55 year old man with intermittent Wolff-Parkinson-White (WPW) syndrome had an episode of atrial fibrillation (AF) that lasted for 117 days. After interruption of the AF a Δwave appeared that lasted for two days and then disappeared. Exercise stress and isoprenaline infusion could not reproduce the Δwave, but after another episode of AF which lasted for seven days a persistent Δwave appeared that lasted for six hours. In an electrophysiological study performed on a day without a Δwave, neither antegrade nor retrograde conduction via an accessory pathway was seen, but after atrial burst pacing (at 250 ms cycle length) for 10 minutes, a Δwave appeared lasting for 16 seconds. Atrial electrical remodelling—that is, the shortening of the atrial effective refractory period caused by AF, is a possible mechanism of the appearance of the Δwave.

Keywords: Wolff-Parkinson-White syndrome; atrial fibrillation; electrical remodelling

Several mechanisms have been suggested to underlie the intermittent appearance of antegrade conduction via an atrioventricular accessory pathway in patients with intermittent Wolff-Parkinson-White (WPW) syndrome. In some of these mechanisms, the refractoriness of the accessory pathway itself or the atrial muscle close to the atrial connection of the accessory pathway is thought to play an important role in the intermittency. Refractoriness is an intrinsic electrophysiological property of the cardiac muscle, but it can be modified by heart rate, the tone of the autonomic nervous system, serum electrolytes, hypoxia, cardioactive drugs, and other factors.

It has been reported that the continuation of tachyarrhythmia can also modify the refractoriness of the cardiac muscle, a phenomenon that is now understood as "electrical remodelling".

We present a patient with intermittent WPW syndrome, in whom persistent antegrade conduction via an accessory pathway—that is, a Δwave, appeared transiently only after the interruption of atrial fibrillation (AF).

Case report
A 55 year old man was diagnosed with intermittent WPW syndrome in 1987 but the Δwave was rarely observed. When he visited a local hospital, the surface ECG showed sinus rhythm and incomplete right bundle branch block, but no Δwave was observed (fig 1A).

First episode of AF
When the patient visited his local hospital again because of palpitation on 29 March 1997, the surface ECG showed AF and no Δwave was observed at this time. He was prescribed oral procainamide (1000 mg/day) but the AF continued (fig 2). The Δwave was documented first in this episode on 19 April, but its appearance was intermittent and the shortest RR interval with the Δwave was 460 ms. Because of the continuation of AF, the patient developed congestive heart failure. Because of the negative inotropic action on cardiac muscle, procainamide was discontinued on 16 July. His heart failure improved during hospitalisation, but AF continued and the shortest RR interval with the Δwave was gradually shortened (fig 2). Catheter ablation of the accessory pathway was considered and the patient was moved to our hospital on 16 July 1997. At the time of admission the ECG showed AF and an intermittent appearance of the Δwave; the shortest RR interval with the Δwave was 320 ms (fig 1B).

On 23 July, before a scheduled electrophysiological study, his cardiac rhythm went from AF to sinus rhythm. The AF had lasted for 117 days. Immediately after interruption of the AF the Δwave was persistently observed (fig 1C); however, it disappeared about two days after the interruption of AF (fig 1D). During this episode of the appearance and disappearance of the Δwave, no antiarrhythmic drugs were used (fig 2).

Holter monitoring
Holter monitoring was performed on a day with Δwave appearance and a day without Δwave appearance. There was no significant difference in autonomic tone between these two days.
Figure 1  12 lead ECG recordings of the patient during his clinical course. (A) Before the first episode of AF. (B) During AF, which was approximately 100 days after the onset of the first episode of AF. (C) One day after the interruption of the first episode of AF. (D) Four days after the interruption of the first episode of AF.
Electrophysiologic study

Pilsicainide

Electrical remodelling of accessory pathway conduction

and oral disopyramide (300 mg/day) for three
days. Intravenous disopyramide (50 mg)
in the surface 12 lead ECG (fig 1C). Isoprenaline
was infused intravenously at a dose up to
1 mg/kg/hour and the whole pacing protocol
was repeated, but no accessory pathway
conduction was documented. The shortening
of the refractoriness of the atrial muscle or the
accessory pathway itself by atrial electrical
remodelling was considered as a possible
cause of the accessory pathway conduction;
therefore, atrial burst pacing for 10 minutes at
a cycle length of 250 ms was performed at the
low right atrial site. Immediately after the
interruption of the atrial pacing, the Δwave
appeared lasting for only 16 seconds. The
effective refractory period measured at the
right low lateral site was shortened from 202 to
188 ms.

SECOND EPISODE OF AF

On 30 July, AF recurred and lasted for seven
days (fig 2). Intravenous disopyramide (50 mg)
and oral disopyramide (300 mg/day) for three
days did not interrupt the AF, therefore, oral
pilsicainide (150 mg/day) was administered,
and the AF was interrupted on the third day.
During this episode of AF, no Δwave was
observed initially, but it started to appear about
two hours after the onset of AF, and its
appearance was intermittent. As in the first
episode, the Δwave became persistent immediately after
the interruption of the AF, but it lasted only
about six hours this time. Oral pilsicainide was
used for the interruption and prevention of AF
during the second episode of appearance and
disappearance of the Δwave. With the continu-
ation of oral pilsicainide, AF did not recur for
up to eight months.

Discussion

A simple phase 3 or 4 block is not the likely
mechanism of the transient appearance of
Δwaves in our patient because heart rate
dependency was not observed. Because the
electrophysiological properties of the accessory
pathway could not be evaluated in the electrophysiological study, the participation of the
decremental property and the impedance mismatch is not known. Influence of the auto-
nomic tone is unlikely as there was no
significant difference in autonomic tone in the
heart rate variability analysis between the days
with and without Δwave appearance. Retro-
grade concealed conduction is also an unlikely
mechanism as no pattern of electrical stimula-
tion could document the conduction through
the accessory pathway. A change in atrial
activation sequence is also an unlikely mecha-
nism because pacing at various atrial sites
could not document conduction through the
accessory pathway during electrophysiological
study. Because (1) the Δwave appeared tran-
siently only after interruption of AF; (2) the
minimal RR interval with the Δwave shortened
gradually during the episodes of AF; and (3)
atrial burst pacing reproduced the Δwave.
appearance, and the atrial effective refractory period was shortened in the electrophysiological study, the shortening of the refractoriness of the atrial muscle or the accessory pathway itself—that is, electrical remodelling, is thought to be at least part of the mechanism of this phenomenon in our patient. The role of antiarrhythmic drugs in transient appearance of the Δwave is unclear; however, the action of class Ia drugs does not seem to be a determining factor because the Δwave appeared after discontinuation of procainamide in the first episode but during continuation of pilsicainide in the second episode.

It has been documented that continuous rapid atrial excitement causes a shortening of the atrial refractoriness as well as a decrease in its rate dependency—that is, atrial electrical remodelling. It is unclear whether similar electrical remodelling can occur in conduction through atrioventricular accessory pathways. In patients with manifest WPW syndrome, rapid atrial excitation such as AF causes a rapid ventricular response, especially in patients with short refractoriness of the accessory pathway—that is, pseudo-ventricular tachycardia, so that it is practically impossible to follow up such patients for a longer period in AF rhythm.

Accessory pathways are thought to originate anatomically from atrial tissue, and therefore it seems possible for an accessory pathway itself to show electrical remodelling, similar to atrial tissue. In a patient with intermittent WPW syndrome, the anatomical level of the block of the conduction through the accessory pathway is unclear. It could be in the accessory pathway itself or in the atrial or ventricular muscles close to the accessory pathway connection. Although we hypothesised that electrical remodelling was a mechanism for the transient appearance of the Δwave in our patient, the site that showed shortening of the refractoriness by atrial remodelling could not be determined.

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