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CASE REPORT

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Prolonged sinus arrest on electrocardiogram recording during apheresis donation in young female donor with convulsive syncope

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Key Clinical Message

An 18-year-old young female with a convulsive syncope during apheresis donation demonstrated a prolonged sudden sinus arrest continued for approximately 30-second on monitor ECG. This prolonged sinus arrest may relate accidental serious complications in ordinal apheresis donation. Continuous electrocardiogram monitor and correspondence are indispensable.

KEYWORDS

apheresis donation, convulsive syncope, ECG monitor, prolonged sinus arrest

1 | INTRODUCTION

The vasovagal reflex (VVR) and citrate reactions are major side effects of blood donation.[1,2] In annual review, the Blood Division of the Japanese Red Cross reported that, for five million donors, there were 3863 severe VVRs (0.079%) and 421 citrate reactions (0.009%).[3] Popovsky et al[4] pointed out that very severe outcome such as syncope with convulsion or an event requiring hospitalization may occur allogeneic and autologous blood donation. To avoid serious complications such as heart events associated with blood, reservation is important. However, the associated pathophysiology is not fully elucidated.

Previously, we developed a signal-averaged electrocardiograph (SA-ECG) system (Fukuda Denshi, Co., Ltd., Tokyo, Japan) that uses a Mason-Likar lead system. The following analysis of sympathetic nervous activity (ratio of low-to-high frequency, LF/HF), QTc interval, and ST-T changes followed by repolarization map is possible immediately after the end of monitor recording.[5]

In this paper, we present a prolonged sinus arrest on XYZ-leads ECG in young female donor with convulsive syncope during apheresis donation.

2 | PRESENTATION

The apheresis donor was an 18-year-old female of 155 cm in height and 56 kg in weight. She suffered a convulsive syncope during platelet and plasma apheresis donation at her first time. The subject had no history of syncope or epileptic seizure in daily life. Prior to donation, her vital signs were 125/70 mm Hg blood pressure and 73 beats per minute heart rate. SA-ECG recording was performed, as approved by the Ethics Committee of the Blood Division of the Japan Red Cross Society (No. 2016-027). Five electrodes (Fukuda Denshi Co., Ltd., Japan) were placed on the surface of the anterior chest with a Mason-Likar lead system.[5] SA-ECG recording covered all stages of the component donation, from before needle puncture to its withdrawal. The platelet concentrate (PC) and platelet poor plasma (PPP) apheresis donation was performed using a bowl type equipment (Hemonetics Japan Co., Ltd., Japan) with an anticoagulant citrate dextrose solution A (ACD-A) (Kawasumi Co., Ltd., Japan). The total depletion volume was 104 mL (51 mL of PC and 53 mL of plasma) after processed blood volume of 438 mL (including 10:1 ratio of ACD-A solution). At the first blood return for PC and PPP apheresis donation, she had a convulsive

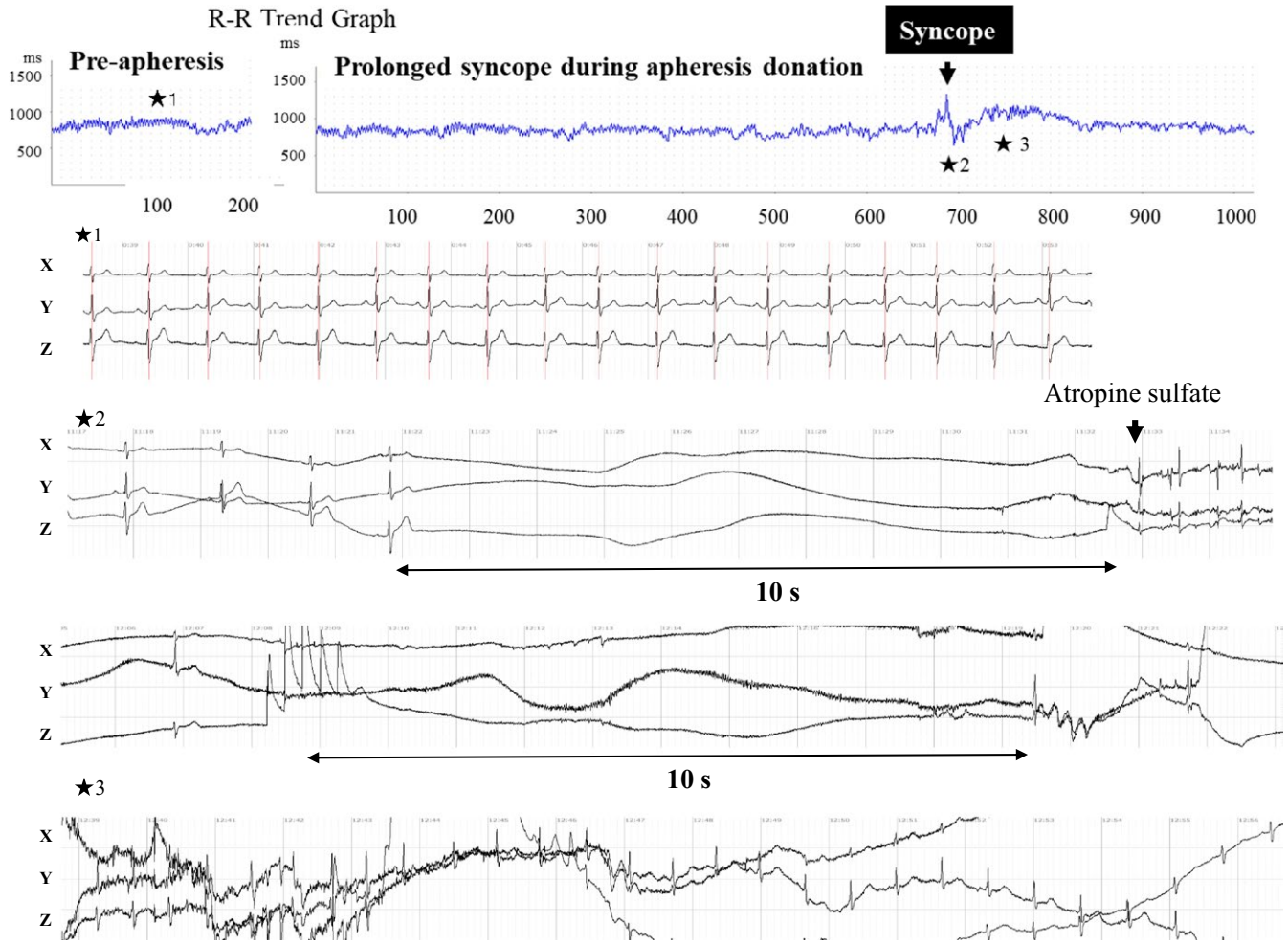

FIGURE 1 Representative R-R trend graph and continuous XYZ-leads ECG during prolonged syncope. Upper: R-R interval trend graph at pre-apheresis and during apheresis donation. ★1: Continuous XYZ-leads ECG at pre-apheresis. ★2: Continued representative continuous XYZ-leads ECG during a convulsive syncope. Note sinus bradycardia followed by approximately 10-sec sudden sinus arrest that was intermittently continued for approximately 30-sec. Arrow indicated the time point when atropine sulfate was injected intramuscularly. ★3: Restored sinus rhythm at the recovery from a convulsive syncope

syncope. Blood pressure at the radial artery was undetectable during the syncope with convulsion. Immediately after the airway was secured, 0.5 mg atropine sulfate was injected intramuscularly. At approximately 5 minutes after recovery from the convulsive syncope, the subject received 500 mL of replacement fluid. Vital signs were 100/55 mm Hg blood pressure, 73 beats per minute heart rate, and 98% peripheral capillary oxygen saturation. At 20 minutes after recovery from prolonged syncope, there was no significant neurological abnormality.

3 | SA-ECG FINDINGS

There was no abnormal ECG characterization of QT prolongation on the pre-apheresis state. During convulsive syncope, XYZ-leads ECG demonstrated a sinus bradycardia followed by a 10-second sudden sinus arrest that was intermittently continued for approximately 30-seccond shown in Figure 1. In a baseline pre-apheresis recording, the continuous XYZ-leads ECG recording indicated normal sinus rhythm (73 beats per minute), and SA-ECG indicated no significant abnormality in the conduction system (Figure 2B). The value of LF/HF was slightly lower at pre-apheresis (0.79) compared than that at presyncope (1.04) shown in Figure 2A. At 20 minutes after recovery from a convulsive syncope, the value of repolarization dispersion map was not significantly changed compared that at pre-apheresis, although QTc interval indicated a slightly extended (415 ms) shown in Figure 2B. One hour after the syncope with convulsion, the donor confirmed no neurological perturbation.

4 | DISCUSSION

Convulsive syncope accompanied by VVR is an infrequent but high-risk complication in apheresis donation. Several

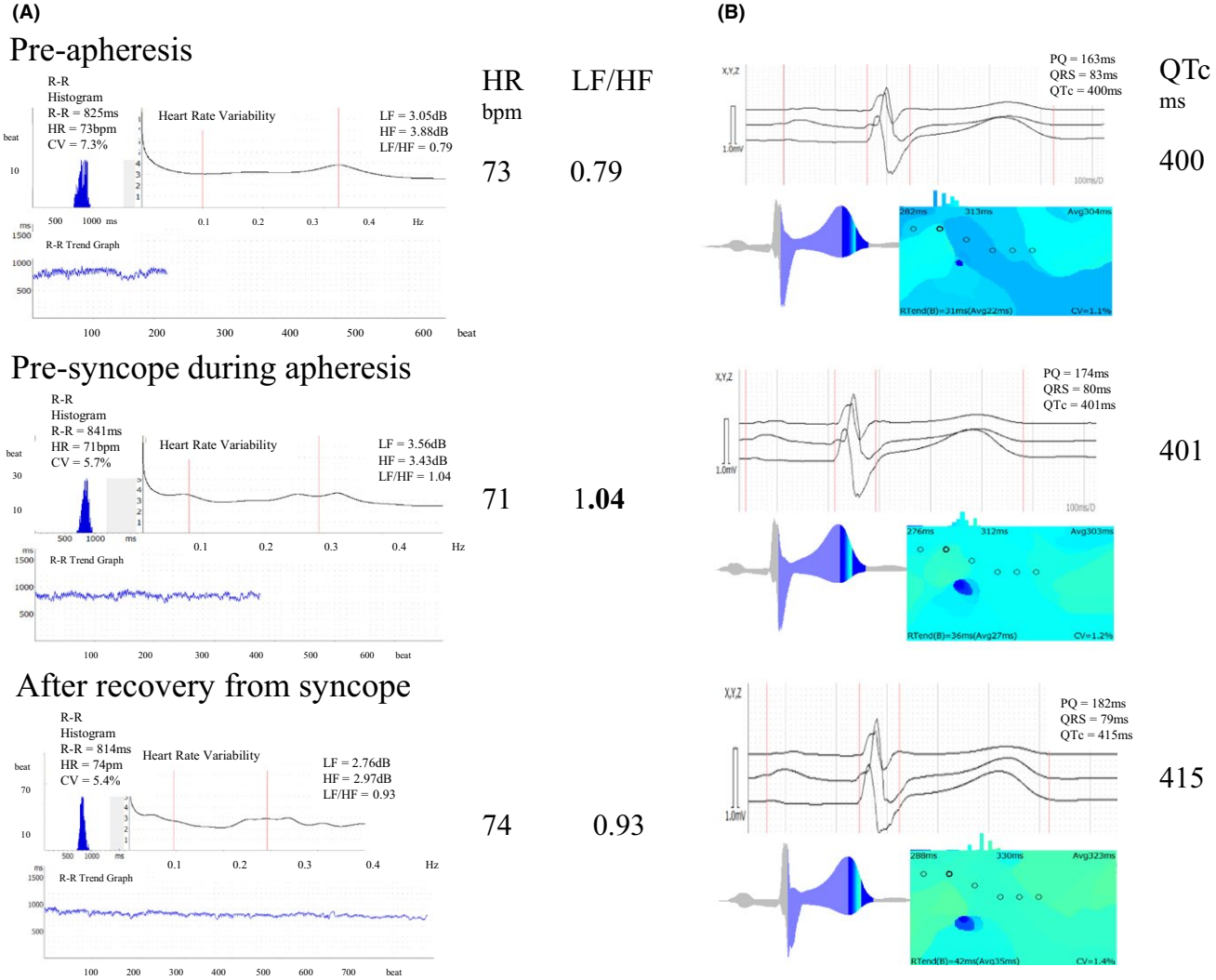

FIGURE 2 Serial R-R trend graph including the value LF/HF (A: left), and averaged QTc interval and repolarization dispersion maps (B: right) at pre-apheresis, presyncope, and after recovery from syncope. The value of LF/HF was slightly lower at pre-apheresis (0.79) than that at presyncope (1.04) shown in Figure 2A. The averaged QTc interval was not significantly changed (400 ms at pre-apheresis, 401 ms at presyncope during apheresis). The repolarization dispersion maps showed no significant difference among the pre-apheresis and presyncope during apheresis and after recovery from a convulsive syncope shown in Figure 2B

factors including neurogenic reflex, anxiety, hypo-blood volume, cardiac abnormalities, and among other factors may relate the onset of VVR. An accentuated anxiety at first apheresis donation may relate a syncope with convulsion in this young female. Graham suggested the hypothesis that an anxiety would be more prominent in those donors who subsequently fainted.[6] Previously, transient disruption of cerebral blood flow for 8-10 seconds may relate in loss of consciousness.[7] However, there is little report showing a direct evidence-related incontinence with convulsions. In the present case study, we proved that a convulsive syncope at apheresis donation related a prolonged sinus arrest based on neural mediated cardioinhibitory type. Generally, an intravenous administration of 0.5 mg atropine sulfate may be useful as an initial response.

Very severe complication or an event requiring hospitalization may occur allogeneic and autologous blood donation.[4] Cassens et al[8] reported that the diagnosed VVR probably induced the circulatory arrest during apheresis donation rather than the administration of G-CSF. A VVR with a convulsive syncope at allogeneic and autologous blood donation may induce serious cardiovascular events. Goodnough et al[9] reported the severe complication of two cases of myocardial infarction during autologous blood donation.

A decreasing blood-donor pool in the presence of increasing blood transfusion demands has resulted in the need to make maximal use of each blood donor and, particularly, younger donors. Clearly, accurate risk assessment in the prediction of VVR with syncope during blood donation is vital for the prevention of serious side effects in donors. A

continuous ECG monitoring including a repolarization nature may be valuable for a risk management of VVR or an event requiring hospitalization.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTION

KN: is a cardiologist and is one of the parties to the case studied; MI: is an engineer, developed the program, and analyzed electrocardiogram; TF: is a cardiologist and analyzed the pathophysiology; YM: is a cardiologist and contributed to overall interpretation.

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