

# Supraventricular Tachycardia Triggered by Spicy Food Consumption: A Novel Case Report and Review of Potential Mechanisms

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## Abstract

Supraventricular tachycardia (SVT) is a common cardiac arrhythmia characterized by rapid heart rates originating above the ventricles. Numerous triggers, such as caffeine, alcohol, and stimulants, have been extensively reported; however, the role of dietary spices as potential triggers remains largely unexplored. Capsaicin, the active ingredient in chili peppers, acts primarily through transient receptor potential vanilloid type 1 (TRPV1) channels, exerting diverse physiological effects, including modulation of cardiovascular function. While capsaicin has documented long-term cardiovascular protective properties, its acute arrhythmogenic potential through other mechanisms remains understudied.

We report an unprecedented case of a 48-year-old male who presented with recurrent episodes of SVT triggered consistently by spicy food intake. The patient arrived at the emergency department approximately 30 minutes after spicy food consumption with a heart rate of 215 beats per minute, resistant to vagal maneuvers. Administration of intravenous adenosine successfully restored sinus rhythm. Comprehensive cardiac evaluations, including echocardiography, coronary angiography, and electrophysiological studies, revealed no underlying cardiac pathology.

Based on this case, we hypothesize a potential role for capsaicin-induced activation of TRPV4 channels, influencing cardiac contractility and arrhythmogenesis. Prior literature has primarily addressed capsaicin's interaction via TRPV1 channels, neglecting acute electrophysiological disturbances. This novel clinical scenario underscores the need for clinicians to consider spicy food consumption as a possible trigger for SVT in differential diagnosis. Further studies are warranted to clarify acute cardiac responses and electrophysiological mechanisms triggered by capsaicin and related dietary spices.

**Keywords:** Supraventricular tachycardia, capsaicin, TRPV channels, spicy food, arrhythmogenesis

## Introduction

Spices have been utilized for various purposes throughout human history (1). Spices play a significant role in culinary cultures worldwide, influencing numerous physiological systems, from the stimulation of chemosensory receptors through taste, aroma, and pungency, to their effects on surface membranes such as skin and mucosa, and, further impacting cardiovascular, respiratory, autonomic, and metabolic functions (2). Recent experimental and clinical studies on capsaicin, the active component of chili peppers, have demonstrated its significant role in cardiac

physiology and pathologies, predominantly mediated via activation of the transient receptor potential vanilloid type 1 (TRPV1) channels (3). Piperine (from black pepper) and capsaicin have exhibited positive inotropic and chronotropic effects and demonstrated cross-tachyphylaxis in isolated rat atria (4). The known cardiac mechanism of capsaicin and the physiological role of TRPV1 channels in cardiac tissues are illustrated below (Figure 1) (5).

Supraventricular tachycardia (SVT) is defined as a tachycardia originating from above the ventricles, characterized by a heart rate exceeding 100 beats per minute. SVTs are classified based



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on their origin, including sinus tachycardia, atrial tachycardia, atrioventricular nodal reentrant tachycardia, and another form of reentrant tachycardia (6). SVT is usually self-limiting and generally not life-threatening; however, prolonged episodes accompanied by syncope, chest pain, or dyspnea necessitate intervention. The presence of accessory conduction pathways contributing to SVT increases morbidity and mortality risks, warranting more aggressive treatment (7). Acute interventions include vagal nerve stimulation (8), adenosine (9), calcium channel blockers (10), beta-adrenergic receptor antagonists (8), and procainamide, whereas definitive management often involves catheter ablation (11).

Although there are publications regarding cardiac rhythm disturbances triggered by spice consumption, clinicians typically do not prioritize spices when evaluating patients presenting with such disturbances. Here, we present a previously unreported case of SVT triggered by spice consumption. The informed consent form was obtained from the patient for this case report.

## Case Report

A 48-year-old male presented to the emergency department (ED) with palpitations that started approximately 30 minutes after consuming spicy food and lasted for nearly one hour without relief. Upon admission, vital signs were as follows: blood pressure 110/80 mmHg, heart rate 215 bpm, and oxygen saturation 97% on room air. The patient denied any other active complaints. Medical records revealed multiple prior ED visits with similar palpitations following spicy food intake. He reported no chronic illnesses, medication use, cardiac history, or family history of sudden cardiac death. Detailed questioning revealed no alcohol, tobacco, energy drink, or stimulant use.

Physical examination showed a Glasgow Coma scale of 15, good general appearance, normal lung auscultation, tachycardic heart rhythm, without additional sounds or murmurs, and normal findings in other systems. Initial electrocardiogram (Figure 2) demonstrated SVT [hazard ratio (HR) 215 bpm]. Following unsuccessful modified Valsalva maneuvers, intravenous adenosine administration (6 mg, then 12 mg) restored normal sinus rhythm (HR 92 bpm, Figure 3). Cardiac enzymes and blood tests were within normal limits. Cardiology consultation and bedside echocardiography revealed no abnormalities. The patient remained stable without recurrence of SVT during ED observation. He was discharged after six asymptomatic hours with advice to avoid spicy foods. Subsequent elective coronary angiography and electrophysiological studies were unremarkable.

## Discussion

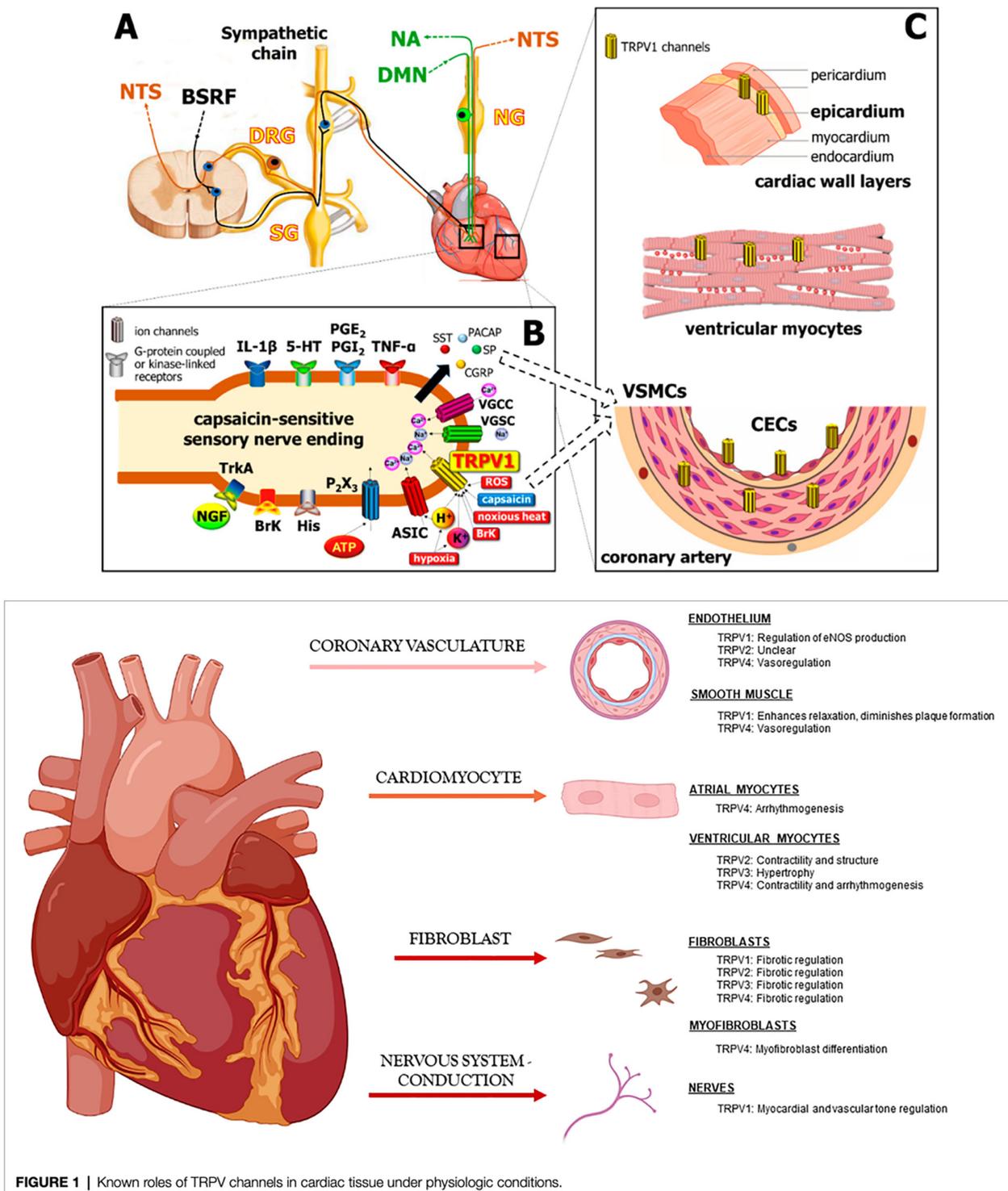
Various underlying factors associated with SVT have been reported, with alcohol and caffeine consumption being the most common dietary triggers. However, spicy food has not been previously documented in the literature as a potential acute precipitant of SVT episodes (12). While a limited number of studies have explored the cardiovascular implications of capsaicin the active compound in chili peppers none have directly associated it with SVT (13,14).

Previous case reports and observational studies have linked spicy food consumption to a spectrum of cardiovascular and systemic effects, including syncope, urticaria, gastrointestinal symptoms, coronary vasospasm, acute myocardial infarction, and even fatal myocardial ischemia in predisposed individuals (12,15). These findings highlight that capsaicin's bioactive role extends beyond gastrointestinal discomfort and may involve acute cardiovascular modulation.

From a mechanistic perspective, capsaicin is a potent agonist of the TRPV channels, primarily TRPV1 and, to a lesser extent, TRPV4. While TRPV1 activation has been associated with long-term cardioprotective outcomes such as nitric oxide-mediated vasodilation, antihypertensive effects, and attenuation of atherosclerosis (16), its acute effects-especially in the context of electrical conduction abnormalities-remain under-investigated. Recent preclinical studies suggest that TRPV4 channels, which are also expressed in ventricular cardiomyocytes, can influence myocardial contractility and are implicated in arrhythmogenesis through calcium influx and mechanosensitive signaling pathways.

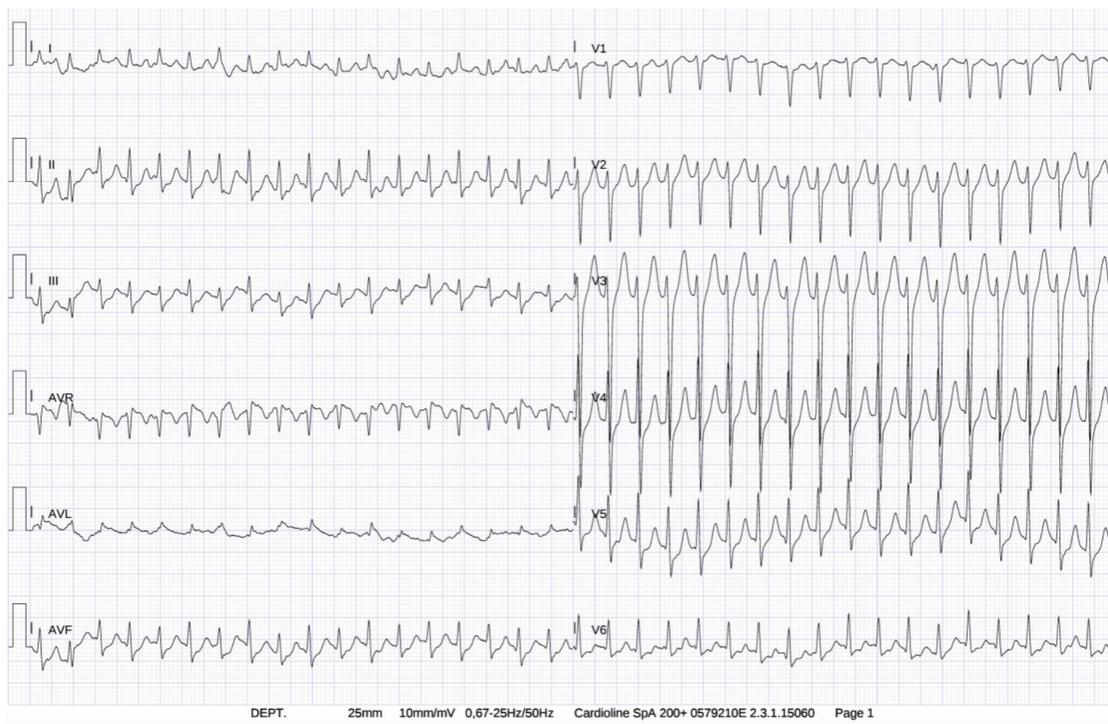
Our hypothesis builds on these findings: capsaicin may have triggered SVT in this patient via acute TRPV4 activation, leading to intracellular calcium dysregulation and enhanced automaticity or reentry mechanisms within the atrioventricular node. This is supported by limited evidence showing acute elevations in heart rate following capsaicin ingestion, although electrophysiological studies assessing arrhythmic potential are notably lacking.

Furthermore, individual susceptibility may play a significant role. Genetic variability in TRPV channel expression, underlying autonomic tone, and pre-existing subclinical conduction abnormalities might predispose certain individuals to arrhythmic events following spicy food intake. Unfortunately, the absence of baseline Holter or electrophysiological data in this patient limits causal inference.



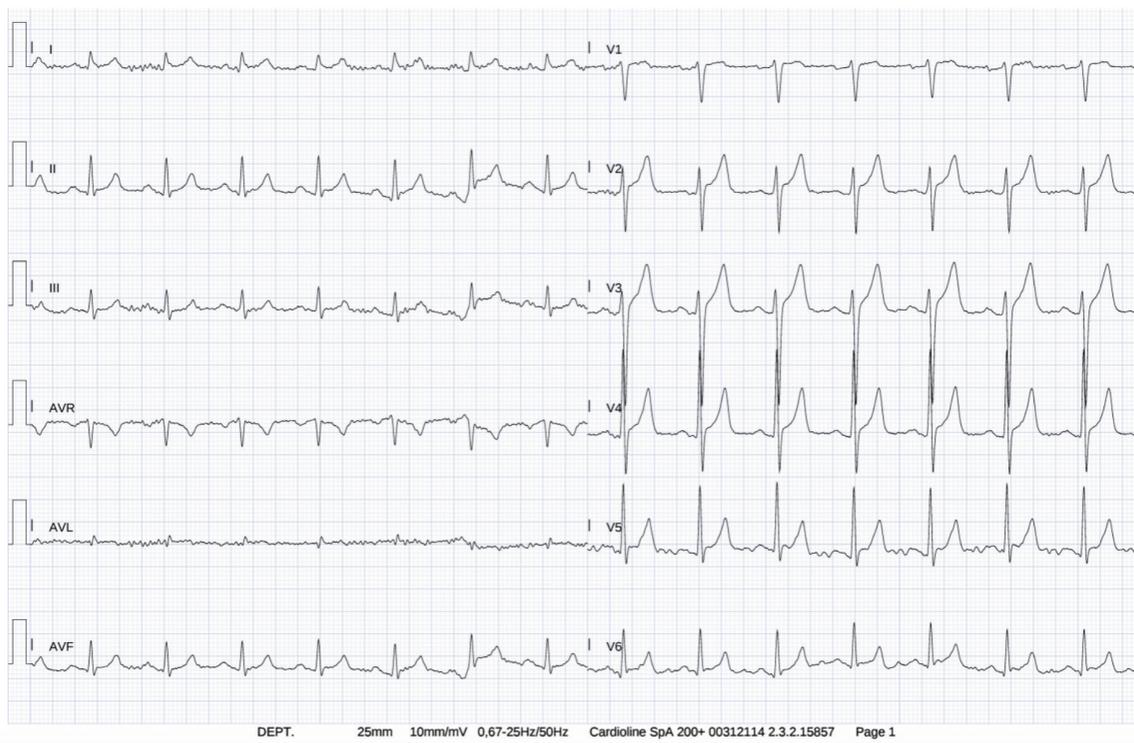
**Figure 1.** Known cardiac mechanism of action of capsaicin (3) and known roles of TRPV channels on cardiac tissue under physiological conditions (5)

TRPV: Transient receptor potential vanilloid, NTS: Nucleus tractus solitarius, BSRF: Brainstem respiratory field, DRG: Dorsal root ganglion, SG: Sympathetic ganglion, NA: Nucleus ambiguus, DMN: Dorsal motor nucleus, NG: Nodose ganglion, IL-1 $\beta$ : Interleukin-1 beta, 5-HT: 5-hydroxytryptamine, PGE: Prostaglandin E, TNF- $\alpha$ : Tumor necrosis factor-alpha, SST: Somatostatin, PACAP: Pituitary adenylate cyclase-activating polypeptide, SP: Substance P, CGRP: Calcitonin gene-related peptide, VGCC: Voltage-gated calcium channels, VGSC: Voltage-gated sodium channels, ROS: Reactive oxygen species, BrK: Bradykinin, ATP: Adenosine triphosphate, NGF: Nerve growth factor, VSMC: Vascular smooth muscle cell, CEC: Cerebral endothelial cell



**Figure 2.** ECG taken at the patient’s first application

ECG: Electrocardiogram, AVR: Augmented vector right, AVL: Augmented vector left, AVF: Augmented vector foot



**Figure 3.** Control ECG taken after 6-12 mg intravenous adenosine treatment

ECG: Electrocardiogram, AVR: Augmented vector right, AVL: Augmented vector left, AVF: Augmented vector foot

## Conclusion

In conclusion, this case introduces a novel and plausible trigger for SVT that has been previously overlooked in emergency clinical settings. It underscores the need for a more comprehensive dietary history in patients presenting with idiopathic arrhythmias. Future prospective studies are warranted to investigate the acute electrophysiological effects of capsaicin and delineate the specific roles of TRPV1 and TRPV4 channels in cardiac rhythm regulation.

## Ethics

**Informed Consent:** The informed consent form was obtained from the patient for this case report.

## Footnotes

### Authorship Contributions

Concept: İ.K., Data Collection or Processing: K.S.K., Writing: İ.K., K.S.K.

**Conflict of Interest:** No conflict of interest was declared by the authors.

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