J Korean Med Sci 1998; 13: 291-4
ISSN 1011-8934

Digital Infrared Thermographic Imaging in Patients with Gastroesophageal Reflux Disease

We performed a thermographic study to observe any possible interaction between the esophageal acid perfusion and the temperature changes of skin surface in patients with gastroesophageal reflux disease (GERD). Twenty-seven patients with GERD were categorized as group I (globus symptoms with posterior laryngitis) and group II (heartburn and/or regurgitation symptoms). Patients and 6 healthy volunteers underwent Bernstein test (BT) and digital infrared thermographic imaging (DITI) simultaneously. The positive rate for BT in group I and group II was 22.2% and 55.6%, respectively, and the DITI positive rate was 55.6% for group I and 50.0% for group II. None of healthy control were positive in BT or DITI. All subjects with DITI positive were hypothermic. The overall accordance rate between DITI and BT was 69.7%. All group I patients showed a diffuse type, while in group II, 4 patients showed diffuse type and 5 patients showed localized type ($p<0.05$). In patients with DITI (+)/BT (−), 83.3% showed diffuse type, whereas equal numbers of diffuse and localized type were noted in patients with DITI (+)/BT (+). In conclusion, acid contact with a sensitive mucosa leads to an activation of the sympathetic nervous system in some patients with GERD, inducing skin surface hypothermia.

Key Words : *Gastroesophageal reflux, thermography*

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Received : November 11, 1997
Accepted : February 2, 1998

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INTRODUCTION

Esophageal acid stimulation leads to a viscerocardiac reflex mechanism which may constrict the coronary arteries (1). Chauhan et al. (2) reported that esophageal acid perfusion could produce angina and significantly reduce coronary blood flow documented by intracoronary Doppler catheter. However, they found that the lack of any significant effect in the heart transplant patients, in whom the heart was denervated, suggested a neural reflex. They suggested that the presence of such a reflex might be a mechanism for "Linked Angina" in patients with coronary artery disease.

It is questioned that acid contact with a sensitive esophageal mucosa lead to an outflow from the sympathetic nervous system. However, skin temperature, a function of superficial perfusion, is largely controlled by the sympathetic vasoconstrictor nerve (3, 4). Thus, increased sympathetic excitation that is a reflection of nerve root irritation may cause active vasoconstriction, resulting in decreased skin temperature (3, 4).

Therefore, we performed this study to observe any possible interaction between the esophageal acid perfusion and skin surface temperature changes using DITI in patients with gastroesophageal reflux disease (GERD).

MATERIALS AND METHODS

Subjects

Twenty seven patients with symptoms of GER for at least 6 months participated in the study. These patients were categorized as group I (globus symptoms with posterior laryngitis, 2 males, 7 females; mean age, 43.1 yr) and group II (heartburn and/or regurgitation symptoms, 4 males, 14 females; mean age, 40.9 yr). As a control group, 6 healthy volunteers (4 males, 2 females; mean age, 49.5 yr) were also evaluated.

Study protocol

Patients and healthy volunteers underwent Bernstein test and digital infrared thermographic imaging (Dorex

Inc, West Collins, CA, USA) simultaneously after an overnight fast. These studies were performed after any medications known to affect GI motility or acid secretion had been stopped for at least 48 hours. The study was approved by the Ethics Committee of Yonsei University College of Medicine. Written informed consents were obtained from all subjects.

Bernstein test (BT)

The modified acid perfusion test was performed following baseline manometry while the patient remained supine (5). Normal saline was infused for 15 minutes followed by 0.1 N HCl infusion for 15 minutes or until symptoms were produced. The test was considered positive when the patient's symptoms or substernal burning pain were reproduced during acid perfusion and relieved by saline.

Digital infrared thermographic imaging (DITI)

This computerized infrared thermography has a resolution 256×240 pixels and a thermal resolution of 0.1℃. For measurement of temperature in the DITIs of the control and patient groups, we divided the anterior thorax into 6 sectors. DITI of anterior thorax area was taken at 1 m distance during an acid perfusion test. This examination were performed in a controlled temperature (19 to 21℃) and humidity (50±5%), and draft free laboratory. All subjects were undressed and allowed to adjust to the room temperature for 15 minutes.

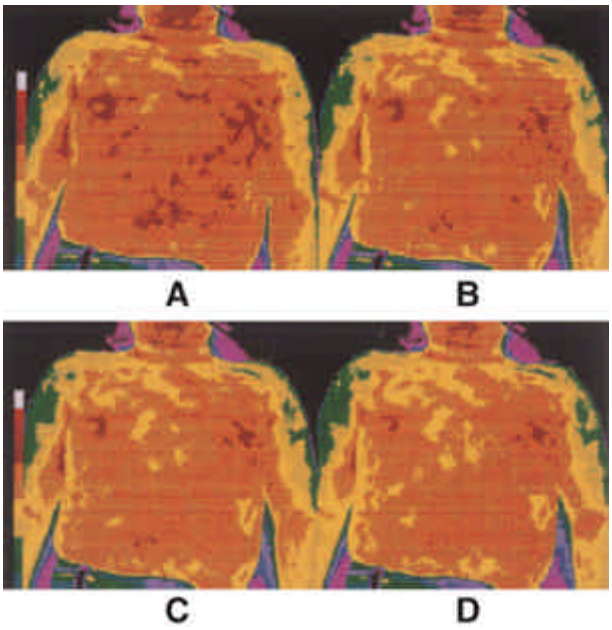

Fig. 1. Thermograms on anterior chest in healthy control. Resting state thermogram before normal saline (N/S) or acid infusion (A). No significant temperature changes were noted during N/S infusion (B), acid infusion (C), or N/S reinfusion (D).

Thermographic findings on acid infusion were considered as positive if a temperature difference of 0.3℃ or more in at least 3 sectors of anterior thorax compared with baseline temperature was detected (6, 7). All thermograms were categorized as hypothermic or hyperthermic type according to thermal changes, and their thermographic findings were classified as diffuse or localized type according to the distribution of surface temperature changes. In other words, localized type was indicated if a temperature difference of 0.3℃ or more in three to six sectors of anterior thorax compared with baseline temperature was detected, whereas diffuse type was defined as diffuse surface changes of temperature in the entire anterior thorax. All tests were interpreted by the same experienced thermologist without any knowledge of the subject's clinical information.

Statistical analysis

Stastical analysis of the relationship between patient's group, Bernstein test, and DITI types was carried out using the chi-square test. A p value less than 0.05 was considered to be statistically significant.

RESULTS

In healthy control, none was positive in BT or DITI (Fig. 1). In contrast, the positive rate of BT in group I and group II was 22.2% and 55.6%, respectively, and DITI positive rate was 55.6% for group I and 50.0% for group II (Table 1). There were no demographic differences between DITI positive and negative patients in group II. All subjects with DITI positive results were hypothermic. The overall accordance rate between DITI and BT was 69.7%. All group I patients showed a diffuse type, while in 4 patients showed diffuse type and 5 pa-

Table 1. Results of BT and DITI in patients and control

	Control (N=6)	Group I (N=9) (%)	Group II (N=18) (%)
BT(+)	0	2/9 (22.2)	10/18 (55.6)
DITI(+)	0	*5/9 (55.6)	*9/18 (50.0)

* All were hypothermic type.

Table 2. Types of DITI in patients with GERD

	Group I	Group II
Diffuse	5	4
Localized	0	5

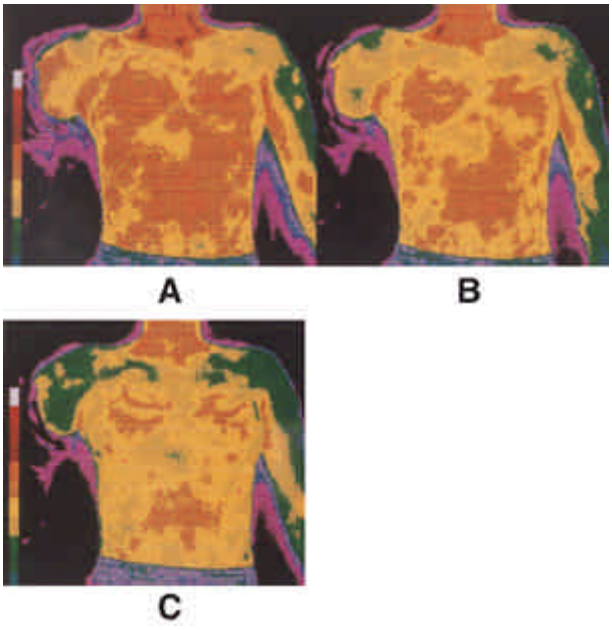

Fig. 2. Localized type of DITI positive in patient with GERD. Note localized skin hypothermia on anterior chest after acid infusion (C) comparing with baseline (A) and N/S infusion (B).

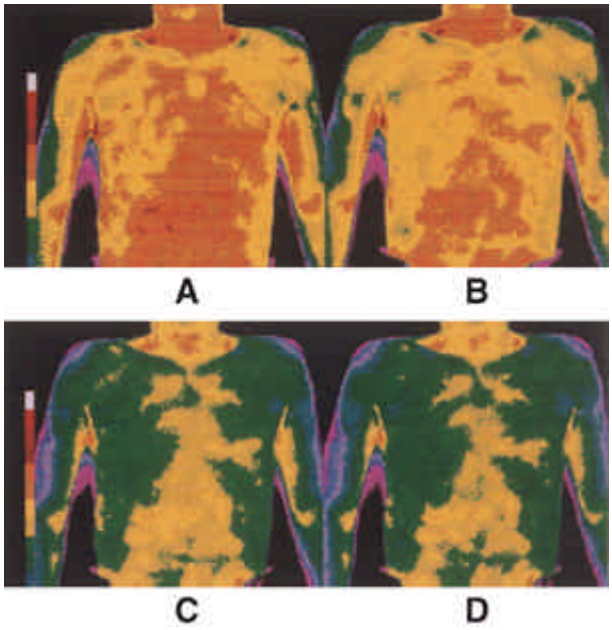

Fig. 3. Diffuse type of DITI positive in patients with GERD. Note diffuse hypothermia on anterior chest after acid infusion (C), compared with baseline (A) and N/S infusion (B). Hypothermia induced by acid infusion could not be recovered during N/S reinfusion (D).

tients showed localized type in group II ($p<0.05$) (Table 2). In 6 patients with DITI (+)/BT (−), diffuse type was noted in 5 (83.3 %), whereas diffuse or localized type was equally seen in patients with DITI (+)/BT (+) (Fig. 2, 3).

DISCUSSION

Studies using thermography have been reported in a wide variety of medical fields (8, 9). In most of the studies, however, this method was applied for detecting skin surface temperature variation only. The application of infrared imaging on viscerocutaneous response, as attempted in this study, has not been studied so far. As noted in a previous study, esophageal acid perfusion resulted in chest pain and caused the rate-pressure product elevation, an index of myocardial work load, in patients with coronary artery disease (10). However, the relevance between esophageal luminal pH and cardiac angina is still controversial. Garcia- Pulido J et al. (11) documented 2 cases of simultaneous peri-pain reflux and ischemic electrocardiographic (ECG) changes through simultaneous 24 hours Holter monitor and esophageal pH records. However, a number of previous studies could not find any correlation between ECG events and corresponding patterns of reflux (12-14). Davies HA et al. (1) found that plasma noradrenaline concentration was increased after esophageal acid perfusion, but the difference was not statistically significant. Therefore, they did not consider that acid contact with a sensitive mucosa led to an outflow from the sympathetic nervous system.

Neurovascular control of skin is mediated by the sympathetic nervous system (3, 4, 15). The components of this sympathetic response are both adrenergic and cholinergic. The adrenergic portion produces vasoconstriction and the cholinergic portion produces vasodilation. In other words, skin hyperthermia indicates the loss of sympathetic fiber function, whereas hypothermia reflects an increased sympathetic fiber function. We observed that none of the healthy control group showed positive results on DITI, wheareas 5 (55.6%) of 9 group I patients with posterior laryngitis who complained of globus symptom were DITI positive and 9 (50.0%) of 18 group II patients with GER who complained heartburn and/or regurgitation were DITI positive. All subjects who showed DITI positive were hypothermic. From this point of view, it seems likely that acid contact with a sensitive mucosa in some patients with GERD leads to an activation of sympathetic nervous system, inducing skin surface hypothermia (Fig. 4). In addition, our result showed that the overall accordance rate was 69.7% between DITI and BT. This result may suggest that acid contact with a sensitive mucosa leads to an activation of the sympathetic nervous system manifested by skin hypothermia as well as causing chest pain in some patients with GERD.

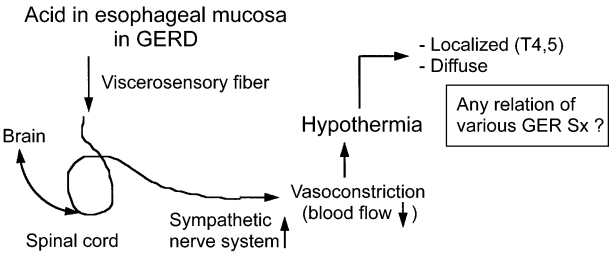

Fig. 4. A proposed mechanism of viscerosympathetic response to esophageal acid perfusion.

We classified thermographic findings as diffuse or localized type according to the distribution of the surface temperature changes. Interestingly, we found statistically significant differences between patient group and DITI types. All patients with DITI positive who complained of globus symptoms showed diffuse type. In 6 patients with DITI (+)/BT (−), 83.3% showed diffuse type, whereas diffuse or localized type was equally seen in patients with DITI (+)/BT (+). It may be suggested that in some patients pain originating from an esophageal acid stimulation projects into the corresponding dermatome, and also causes localized blood flow changes in the skin. But the observations for these 2 types (diffuse or localized) with hypothermia require further study on any relevance to different GER symptoms. Furthermore, if we had checked the plasma level of catecholamine simultaneously during the test along with esophageal mucosal electrical potential difference in this study, we might have explained the possible mechanism of DITI (−) patients with GERD.

In conclusion, acid in contact with a sensitive mucosa in some patients with GERD led to an activation of the sympathetic nervous system which was demonstrated as skin surface hypothermia.

Acknowledgements

We would like to thank JW Ha, M.D for his helpful information and Miss EA Yoon for assistance with the thermographic study.

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