

Localized Heat Urticaria from 95-GHz Millimeter Waves

John A. Gibbons

INTRODUCTION: Local heat urticaria is a physical urticaria caused by the environmental stimulus of heat. Typically, the resultant lesion, a wheal, is pruritic (itches) and is sometimes accompanied by a burning sensation. It is a self-limited phenomenon that resolves after 1.5–2 h. The prevalence of local heat urticaria in the general population has been estimated as 1 in 200,000 persons.

CASE REPORT: The subject, a 39-yr-old active duty man, participated in a test of a 95-GHz energy beam designed to heat the skin. He had delayed presentation of raised, erythematous, nonpruritic, nonpainful areas at five of the exposure sites where the skin temperature exceeded 54°C. All wheals resolved within 2 h of the exposures.

KEYWORDS: hives, physical urticaria, 95-GHz, millimeter wave.

Gibbons JA. Localized heat urticaria from 95-GHz millimeter waves. *Aerosp Med Hum Perform*. 2017; 88(6):586–588.

Local heat urticaria is a physical urticaria caused by the environmental stimulus of heat. Duke first described local heat urticaria in the medical literature.⁹ Mast cells within the outer layers of the skin acutely release histamine and other proinflammatory mediators. Histamine in turn causes dilation of the vascular bed between the epidermis and dermis, causing local erythema and edema (redness and swelling). Typically, the resultant lesion, a wheal, is pruritic (itches) and is sometimes accompanied by a burning sensation. It is a self-limited phenomenon that resolves after 1.5–2 h. Clinical medical evaluation for recurrent urticaria associated with local heating includes placing a test tube containing water or a metal body heated to 44°C (C) against the skin for 4 to 5 min. Up to 55°C has been recommended by other authors. Wheal formation typically begins within a few minutes of the heat exposure.⁸ This is differentiated from angioedema, which results from dilation of the vascular bed between the dermis and hypodermis, though urticaria is present with approximately 80% of angioedema occurrences.⁵

Physical urticaria is present in 20 to 30% of adults with chronic urticaria, or about 0.2–0.3% of the general population. Local heat urticaria is rare.⁸ A review of 2310 people with urticaria classified 370 with physical urticarias and only 4 with local heat urticaria.⁴ There are from 100 to 200 case reports of local heat urticaria in the medical literature, depending upon the source. Alternately, the prevalence of local heat urticaria in the general population has been estimated as 1 in 200,000 people or an incidence of 0.0005%. Angioedema occurs with urticaria in about 40% of cases. Serious consequences of angioedema can

occur if the edema involves the respiratory tract.¹⁴ Local heat urticaria of the skin does not carry this risk.

Other forms of physical urticaria include cold, pressure, water, vibration, cholinergic (or generalized heat), and solar urticarias. Cholinergic urticaria accounts for about 30% of physical urticaria cases. It produces pruritus and wheals in association with a rise in core body temperature, such as seen with exercise or hot baths. Solar urticaria has been reported to comprise 0.4–0.5% of chronic urticaria cases. Solar urticaria can produce pruritus and wheals after only minutes of sunlight exposure, either direct or through glass.⁷ Often, a person may have more than one type of physical urticaria, such as cholinergic urticaria and pressure urticaria. A detailed history regarding the trigger events plus focused diagnostic testing may be required to distinguish localized heat urticaria from cholinergic or solar urticaria.¹⁹

CASE REPORT

The subject, a 39-yr-old active duty U.S. Air Force man, gave voluntary informed consent to participate in a research study of

From the Bioeffects Division, Human Effectiveness Directorate, 711th Human Performance Wing, Fort Sam Houston, TX.

This manuscript was received for review in June 2016. It was accepted for publication in February 2017.

Address correspondence to: John A. Gibbons, D.O., M.P.H., Chief, Aeromedical Services, Bioeffects Division, 711th Human Performance Wing, 4141 Petroleum Drive, Fort Sam Houston, TX 78234; jogibbon@jhsph.edu.

Reprint & Copyright © by the Aerospace Medical Association, Alexandria, VA.

DOI: <https://doi.org/10.3357/AMHP.4707.2017>

a 95-GHz energy beam designed to heat the skin. Pre-exposure evaluation revealed the subject developed erythema, without edema, after stroking the skin on his posterior thorax with a blunt tip. He was not taking any medication, prescription or over-the-counter.

The test plan included 16 exposures to a 95-GHz energy beam of differing intensity and duty cycle. The beam was configured to expose a 4-cm diameter spot on the skin. Real time skin temperature monitoring was performed. The subject was instructed to continue the exposure until the discomfort was intolerable. All exposures were terminated either by the subject releasing a handheld switch that stopped the 95-GHz transmission, the subject moving out of the path of the 95-GHz energy beam, or automatically by sensing that the exposed skin temperature reached 60°C (140°F) for 3 s.

The subject received a total of 16 exposures during the testing session over approximately 30 min, 8 to the abdomen and 8 to the lumbar region. Erythema was noted at all 16 sites after completing each exposure. Power density ranged from 3 to 12 W/cm². Pulse length ranged from 0.1 to 1.3 s. Exposure duration ranged from 1.3 to 16.5 s. Final skin temperatures ranged from 49.5 to 58.6°C. Thermal induced skin damage is not a linear function of temperature. The integral of Cumulative Equivalent Minutes – 43 (CEM-43) for the peak skin temperature is used to estimate the thermal dose by calculating the temperature over time as an equivalence to time at 43°C. CEM-43 values ranged from 0.7 to 128.8 for the subject.

He had delayed presentation of raised, erythematous, non-pruritic, nonpainful areas at six of the exposure sites where the skin temperature exceeded 54°C. CEM-43 values equal to or greater than 11.1 were associated with development of a wheal. No wheal was present where the skin was below this temperature or CEM-43 value. There were no symptoms suggestive of systemic effect, such as lightheadedness, shortness of breath, or change in vision. All wheals resolved within 2 h of the exposures. **Fig. 1** illustrates some of these wheals in the lumbar region.

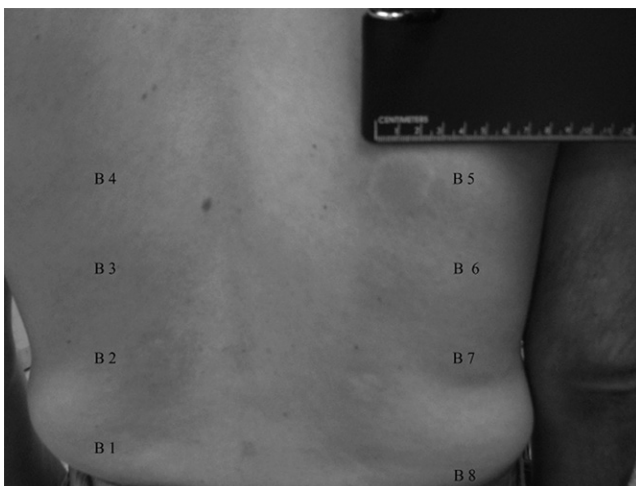


Fig. 1. 95-GHz exposure sites.

The subject underwent a thorough medical history and physical examination to identify any predisposition or other etiology for the development of urticaria. Medical history was significant for childhood allergies, including grass, for which he received immunotherapy shots. He has been asymptomatic since age 13. His medical history was negative for a previous episode of urticaria from any cause. Physical examination revealed: 1) nonexudative degeneration of the OD macula with a small hypo-pigmented area; 2) a few hypertrophied lymph patches on the posterior pharyngeal wall without postnasal drainage; and 3) decreased light touch sensation over the lateral aspect of the superior right thigh. The remainder of the physical examination was unremarkable. In summary, the subject had a history of atopy not receiving current medical treatment.

DISCUSSION

Local heat urticaria was reported by Bryce et al. after exposure to a 95-GHz energy beam. This previous subject developed extensive erythema across her back and dermatographism that followed the seams of an overlying sweater.³ Immediately post-exposure, extensive erythema that was mildly tender and moist appeared on her posterior thorax. Within 20 min, wheals and most of the erythema resolved. All of the erythema resolved within 2 h. These are the only two cases of local heat urticaria after over 13,000 human exposures to 95-GHz energy beams. Neither developed systemic symptoms or angioedema.

With 95 GHz, tissue heating is dependent upon the amount of incident energy received. About 87% ($1 - 1/e^2$) of the energy from 95 GHz is limited to the outer 0.3 mm of skin, one wavelength in depth. Only 0.2% of the incident energy remains at a depth of three wavelengths, 0.9 mm. The total skin depth of the epidermis and dermis ranges from 2 mm to 4 mm. Thus, less than 1% of incident energy reaches the vascular bed between the dermis and hypodermis. The exposed skin area was 4 cm² for this individual. The human pain threshold is 43–44°C. Tissue damage is detectable at 55°C and a second or third degree burn develops with an exposure to 60°C for 6 s.^{18,21}

Chang and Zic⁶ presented a case of local heat urticaria. Chang and Zic's individual developed urticaria after a warm bath, or exposure to oven heat or sunlight, supporting a diagnosis of solar urticaria rather than local heat urticaria. Their accompanying literature review found only 47 case reports of this condition. Medical reports were unavailable for nine persons. They concluded from these references that about 50% of the remaining 38 people with heat urticaria experienced systemic symptoms.⁶ Systemic symptoms were reported to include syncope, fatigue, headaches, wheezing, dyspnea, nausea, vomiting, abdominal cramps, diarrhea, flushing, and fever. Of the 17 total people reported with systemic symptoms, 10 individuals also had solar urticaria and/or cold urticaria. Another three persons reported to have local heat urticaria actually had symptoms only as a result of sun exposure, a more proper diagnosis being solar urticaria. Of the 38 people, 4 (< 11%) had isolated local heat urticaria with systemic symptoms. Michaelsson and Ros reported

an individual with occasional nausea and fever from prolonged use of a hair dryer and development of local heat urticaria after exposure to a temperature of 43°C.¹⁷ This person's family had eight other members with sensitivity to heat, but symptomatology was only reported for the case subject. Higgins and Friedmann reported an individual with flushing, dizziness, and syncope after immersion in a hot bath, and development of local heat urticaria after exposure to a temperature of 42°C.¹² Johansson et al. reported an individual with weakness after a hot bath and development of local heat urticaria after exposure to a temperature of 42°C.¹³ Grant reported an individual with occasional headache, nausea, and lightheadedness with exposure of "larger areas" and local urticaria or angioedema from a hot water bath at 44°C in the laboratory.¹¹ For three of these four people, systemic symptoms were secondary to immersion in hot baths, not from focal exposures.

Since Chang and Zic published their case report, additional people with local heat urticaria have been reported. Three case reports describe persons with dizziness and syncope that occurred after a hot bath or hand washing with hot water.^{1,10,15} Another case report described an individual with dizziness and syncope after a hot bath, but not local reaction to hand washing with hot water.²⁰ Three case reports mention people with no systemic symptoms associated with local heat urticaria.^{2,16,20} In summary, systemic symptoms have been associated with local heat urticaria only after people had prolonged or extensive exposure, such as during hot baths or showers.

This is the second report of a previously asymptomatic individual developing local heat urticaria after a 95-GHz energy beam exposure, an incidence of <0.02%. The duration of exposure is too short to produce systemic symptoms based upon existing local heat urticaria clinical case reports. The depth of skin penetration by 95 GHz is sufficient to dilate the blood vessels at the epidermal-dermal junction and produce a wheal. Penetration depth is not sufficient to cause dilation of the dermal-hypodermal blood vessels and produce angioedema. The human repel response prevents sufficient exposure duration for thermal energy to be conducted to the dermal-hypodermal junction blood vessels and cause dilation or angioedema. It is possible that a large diameter urticarial reaction where a border is difficult to perceive may be mistaken for angioedema. To date, 95-GHz energy has a low incidence of local heat urticaria, consistent with the incidence of local heat urticaria within the general population. Research also shows 95-GHz energy has no systemic effects or long-term sequelae.¹⁸

ACKNOWLEDGMENTS

The author wishes to thank the principal investigator of the research study during which this reported event occurred, Jeffrey Whitmore, Ph.D. The experiment was conducted in compliance with DoDI 3216.02 and AFI 40-402. The author also wishes to thank Brent Voorhees, M.S., and Charles Beason, M.S., for providing technical information on the 95-GHz exposures described in this case report. The views expressed are those of the author and do not necessarily reflect those of the U.S. Government, U.S. Department of Defense, or the U.S. Air Force.

Author and affiliation: John A. Gibbons, D.O., M.P.H., Chief, Aeromedical Services, Bioeffects Division, 711th Human Performance Wing, Fort Sam Houston, TX.

REFERENCES

1. Baba T, Nomura K, Hanada K, Hashimoto I. Immediate-type heat urticaria: report of a case and study of plasma histamine release. *Br J Dermatol*. 1998; 138(2):326–328.
2. Barnes CO, Kulczycki A. Localized heat induced urticaria: a case report. [Abstract.] *J Allergy Clin Immunol*. 2007; 119(1):S198.
3. Bryce LM, Beason CW, Ross J, Meffert J. Probable case report of heat-induced urticaria from 95 GHz millimeter waves. Poster session presented at: 81st Annual Scientific Meeting of the Aerospace Medical Association; Los Angeles, CA; 2010 May 10–13. Alexandria (VA): Aerospace Medical Association; 2010.
4. Champion RH. Urticaria: then and now. *Br J Dermatol*. 1988; 119(4): 427–436.
5. Champion RH, Roberts SOB, Carpenter RG, Roger JH. Urticaria and angio-oedema. *Br J Dermatol*. 1969; 81(8):588–597.
6. Chang A, Zic JA. Localized heat urticaria. *J Am Acad Dermatol*. 1999; 41(2, Pt. 2):354–356.
7. Dice JP. Physical urticaria. *Immunol Allergy Clin North Am*. 2004; 24(2):225–246.
8. Dice JP, Gonzalez-Reyes E. Physical urticarias. In: Saini S, Elmetts CA, editors. *UpToDate*. Waltham, (MA): UpToDate. [Accessed on 24 April 2015]. Available from <http://www.uptodate.com/contents/physical-urticarias>.
9. Duke WW. Urticaria caused specifically by the action of physical agents. *JAMA*. 1924; 83(1):3–9.
10. Fukunaga A, Shimoura S, Fukunaga M, Ueda M, Nagai H, et al. Localized heat urticaria in a patient is associated with a wealing response to heated autologous serum. *Br J Dermatol*. 2002; 147(5):994–997.
11. Grant JA, Findlay SR, Thuesen DO, Fine DP, Krueger GG. Local heat urticaria/angioedema: Evidence for histamine release without complement activation. *J Allergy Clin Immunol*. 1981; 67(1):75–77.
12. Higgins EM, Friedmann PS. Clinical report and investigation of a patient with localized heat urticaria. *Acta Derm Venereol*. 1991; 71(5):434–436.
13. Johansson EA, Reunala T, Koskimies S, Lagerstedt A, Kauppinen K, Timonen K. Localized heat urticarial associated with a decrease in serum complement factor B (C₃ proactivator). *Br J Dermatol*. 1984; 110(2): 227–231.
14. Kaplan AP. Angioedema. *World Allergy Organ J*. 2008; 1(6):103–113.
15. Koh YI, Choi IS, Lee SH, Lee JB, Park CH, Hong SN. Localized heat urticaria associated with mast cell and eosinophil degranulation. *J Allergy Clin Immunol*. 2002; 109(4):714–715.
16. Martín-Muñoz MF, Muñoz-Robles ML, González P, Martín-Esteban M. Immediate heat urticaria in a child. *Br J Dermatol*. 2002; 147(4): 813–815.
17. Michaëlsson G, Ros AM. Familial localized heat urticaria of delayed type. *Acta Derm Venereol*. 1971; 51(4):279–283.
18. Miller SA, D'Andrea JA, Cook MC, Chalfin S, Ziriac J, et al. TSRL-PA-13-0023. Summary of results from the active denial biological effects research program. Wright-Patterson AFB (OH): U.S. Air Force Research Laboratory; 25 Jan. 2013.
19. Schaefer P. Urticaria: evaluation and treatment. *Am Fam Physician*. 2011; 83(9):1078–1084.
20. Skrebova N, Takiwaki H, Miyaoka Y, Arase S. Localized heat urticaria: a clinical study using laser Doppler flowmetry. *J Dermatol Sci*. 2001; 26(2):112–118.
21. United States Consumer Product Safety Commission [Internet]. Bethesda, MD: U.S. Consumer Product Safety Commission. Publication 5098: Avoiding Tap Water Scalds. [Accessed 26 June 2015]. Available from: http://search.cpsc.gov/search?site=en_us_public_site&output=xml_no_dtd&getfileds=*&tlen=120&client=CPSC_frontend&proxystylesheet=CPSC_frontend&q=Tap%20Water%20Scalds&filter=0.