addressed previously in detail. The above case illustrates, as a previous letter has pointed out,[1] that another aspect of SMBG, the preparation of the finger prior to blood sampling, may at times cause significant difficulties if not performed correctly.

MICHAEL R. MACADAMS, M.D.

From the Department of Internal Medicine, The University of California, Davis, Sacramento, California.

Address reprint requests to Dr. Michael R. MacAdams at P.C.C. Division of General Med., 2221 Stockton Blvd., Sacramento, CA 95817.

REFERENCE

[1] Hana, A. K., and Keon, H. M.: Infection and SMBG. Diabetes Care 1984; 7:612.

Nonstaphylococcal Abscesses in Diabetic Individuals

The incidence of colonization of the skin and nasopharynx by *Staphylococcus aureus* is increased in diabetic patients taking insulin compared with the normal population and diabetic patients using oral agents.[1-3] It is generally assumed that the usual causative agent of furuncles, carbuncles, and similar abscess processes in diabetic subjects is *Staphylococcus aureus*.[4,5] Although many of these infections are indeed caused by *Staphylococcus aureus*, some of them may be caused by other bacteria and also may be polymicrobial.

Over a period of 6 mo, nine diabetic patients were admitted to Rancho Los Amigos Medical Center with closed abscesses that were found to be caused by organisms other than *Staphylococcus aureus*. The patients presented with localized pain and swelling but with few systemic manifestations. The sites involved were scalp, breast, groin, flank, thigh, and calf.

Culture material was obtained under aseptic conditions either by needle aspiration or during surgical incision and drainage. Culturette swabs (Marion Scientific, Kansas City, MO) were used for aerobic cultures and specimens that were sent for anaerobic cultures were placed in an anaerobic transport medium (Anatrans, Carr Scarborough Microbiologicals, Decatur, GA). Aerobic cultures were plated on blood agar, Columbia CNA, and MacConkey media (BBL, Cockeysville, MD) and incubated at 35°C in 6–8% CO_2 for ≥48 h. Anaerobic cultures were plated on CDC anaerobic blood agar, phenylethylalcohol blood agar, and laked blood agar with kanamycin and vancomycin (BBL). The specimen was also inoculated into thiogycollate broth. The plates were incubated anaerobically at 35°C and held for ≥7 days. Anaerobic cultures were performed in five of the nine patients.

The organisms isolated were *Staphylococcus epidermidis* (N = 4), *E. coli* (N = 3), Enterococcus (N = 3), *Proteus mirabilis* (N = 2), *Serratia marcescens* (N = 1), *Streptococcus viridans* (N = 1), and *Citrobacter freundii* (N = 1). Five patients had monomicrobial infections, two with *Staphylococcus epidermidis* and one each with *Citrobacter freundii*, *Proteus mirabilis*, and *Serratia marcescens*. Four patients had polymicrobial infections, three with three organisms and one with two organisms. *Staphylococcus epidermidis* was the most frequently isolated organism (four of nine patients). Enterococcus and *E. coli* were found in three patients each and *Proteus mirabilis* in two. Aerobic gram-negative organisms were found in six of nine abscesses. Despite intravenous antibiotic therapy and an initial incision and drainage, only one patient healed without further surgical intervention.

Anaerobic cultures were obtained in five of the nine patients. In these patients no anaerobes were isolated.

Abscesses in diabetic individuals may be mono- or polymicrobial and may be caused by organisms other than *Staphylococcus aureus*, including S. *epidermidis*, diphtheroids, enterobacteriaceae, and Enterococcus. The findings of this study are similar to those of Meislin et al.,[6] with the exception that Enterococcus was among the isolates and no anaerobes were found. However, specific collection and culture techniques for anaerobes were not carried out in four of nine of the patient cases in the current report. The findings in our study were similar to those of Meislin et al. in that the clinical appearance of an abscess caused by *Staphylococcus aureus* is not distinguishable from that caused by other organsims. The *Staphylococcal epidermidis* identified in our patients were cultured from closed abscesses using aseptic techniques and represent, in our opinion, infecting bacteria, not skin contaminants.

In summary, this study demonstrates that soft tissue abscesses in the diabetic patient may be polymicrobial and may be caused by bacteria other than *Staphylococcus aureus*. Appropriate antibiotic therapy, directed at the causative organism(s), and surgical intervention in addition to the initial incision and drainage may be required for healing.

ALICE N. BESSMAN, M.D.

From the Section of Infectious Disease and Diabetes, Department of Medicine, Ranchos Los Amigos Medical Center, 7601 East Imperial Highway, Downey, California 90242.

Address reprint requests to Alice N. Bessman, M.D., at the above address.

REFERENCES

[1] Tuazon, C. U.: Skin and skin structure infections in the patient at risk: carrier state of *Staphylococcus aureus*. Am. J. Med. 1984; 76:166–71.

[2] Wheat, J. L.: Infection and diabetes mellitus. Diabetes Care 1980; 3:187–97.

[3] Casey, J.: Immunity and infections in diabetic patients. Cardiovasc. Rev. Rep. 1982; 3:1159–65.

[4] Rabinowitz, S. G.: Infection in the diabetic patient. *In* Diabetes Mellitus, Vol. V. Rifkin, H., and Raskin, P., Eds. Bowie, MD, Brady, 1981:214.

[5] Olson, O. C.: Diagnosis and Management of Diabetes Mellitus. Philadelphia, PA, Lea & Febiger, 1981:132.

[6] Meislin, H. W., Lerner, S. A., Graves, M. H., et al.: Cutaneous

abscesses. Anaerobic and aerobic bacteriology and outpatient management. Ann. Intern. Med. 1977; 87:145–49.

Use of Low-Dose Continuous Corticosteroid Infusion to Facilitate Insulin Pump Use in Local Insulin Hypersensitivity

Hypersensitivity reactions may occur at the site of insulin injection and can cause painful local reactions, which complicate insulin therapy.[1] Furthermore, hypersensitivity reactions to insulin continue to be reported despite the use of highly purified animal and human insulins.[2] We report the results of investigation and management of a patient who had persistent local reactions at the sites of insulin injection with either intermittent or continuous subcutaneous insulin infusion (CSII) therapy. The patient's local reactions to insulin disappeared when low doses of corticosteroid were mixed with insulin administered by CSII.

The patient is a 16-yr-old women who had type I diabetes mellitus diagnosed 6 yr before our evaluation. She was first placed on lente beef-pork insulin without complication but began to have frequent hospitalization for diabetic ketoacidosis at 14 yr of age. She was started on twice daily subcutaneous injections of Iletin II purified pork regular and NPH insulins, but continued to have unstable diabetes mellitus. Her control improved for ~6 mo with CSII of Iletin II purified pork regular insulin administered by an insulin pump (Autosyringe 6C, Travenol, Deerfield, Illinois). She objected to continued use of the insulin pump and was placed on multiple daily subcutaneous injections of Iletin II purified pork regular and NPH insulins, and later on beef-pork ultralente and regular insulins. After requiring five hospitalizations for diabetic ketoacidosis in a 7-mo period, she was again placed on CSII, using Lilly Humulin regular insulin. After ~1 mo on this routine, she began to develop reactions at the insulin infusion site. Observation of skin test sites injected with single doses of insulin revealed that persistent induration resulted from the fact that the dermal reactions were "biphasic" in type. That is, wheal-and-flare (hive) reactions developed at the site of CSII within 15 min, peaked during the ensuing 15 min and decreased in size without complete resolution over the remaining 1–2 h. During the 2- to 6-h period after insulin injection, the site again became erythematous and pruritic and induration gradually developed. Reactions appeared fully developed by 6 h, at which time they were circumscribed, intensely pruritic, edematous, and painful to touch. These reactions frequently persisted for up to 24 h. *Staphylococcus aureus* was not cultured from the needle site used for CSII. Actrapid human insulin (Squibb-Novo, Princeton, NJ) was substituted for the Lilly preparation, but reactions still occurred. Reactions became progressively larger and more painful with continued insulin therapy. Attempts to block these local reactions with oral antihistamine therapy were unsuccessful. Also, changing the needle from the usual stainless steel needle to a plastic catheter failed to affect the size of the reaction. Reactions were especially large at the site of needle placement when it was left in place for >24 h. Further evaluation was initiated at that time.

The patient was skin tested with a battery of 14 insulin preparations using 0.02 ml of a 50-U/ml insulin solution prepared from U 100 insulin by diluting insulin with equal volumes of buffered phenol saline.[1] Appropriate control skin tests with phenol saline and histamine (1 mg/ml) were performed. Skin tests were positive at 20 min for all insulins tested, including zinc-free insulins, protamine-free insulins, and human insulins. No reactivity to zinc acetate or to the insulin diluents was noted. Reaction sites were observed at 6 h with local induration present at all insulin skin test sites. Insulin-specific IgE was measured (courtesy of Dr. Phillip Fireman) using a radioallergoabsorbent test.[2] Results were expressed in RAST units calculated as percentage counts bound to total counts. Insulin-specific antibodies of the IgE class were detectable to beef (6.5 U/ml), pork (5.4 U/ml), and human (5.5 U/ml) insulins. Values for IgE-anti-insulin in nondiabetic individuals average <4 U/ml and those for insulin-requiring diabetic individuals without reactions average <5 U/ml in this system. Insulin-specific IgG was measured using ^{125}I-labeled insulin in an insulin-antibody binding assay where results are reported as percent binding,[3] and was detectable in the two regular insulins tested: beef (24.7%) and pork (16.5%). Control values for nondiabetic individuals are <3% binding in this system.

Since previous reports have suggested that incorporation of small amounts of corticosteroids into insulin preparations are useful in patients with local insulin reactions who are receiving intermittent insulin therapy,[4,5] 0.22 mg methylprednisolone was added to each 5 U of human insulin placed in the pump (0.04 mg methylprednisolone/1 U insulin). The addition of this preparation resulted in immediate and complete resolution of the dermal reactions previously noted. Subsequently, lower doses of methylprednisolone (0.02 mg methylprednisolone/1 U insulin) were found to be equally effective. After several months, corticosteroid was gradually discontinued and reactions did not recur.

Two types of local reactions have been reported in patients on continuous insulin infusion—local hypersensitivity reactions and cutaneous abscesses associated with *Staphylococcus aureus* infection at the needle site.[6] The latter reactions are characterized by a pustule and on occasion may require incision, drainage, and antibiotic therapy. Four types of local hypersensitivity reactions to insulin have been reported.[1] These include immediate wheal-and-flare reactions, biphasic reactions with a wheal and flare followed by a late reaction at 6–12 h, and delayed hypersensitivity reactions and Arthus reactions. The timing and appearance of the reactions that occurred in this patient were biphasic-type insulin reactions, the more common type of dermal hypersensitivity to insulin.[1] In general, biphasic reactions appear to be decreased in size by oral antihistamine therapy, although in our experience