

portable cautery device, which may not be available in a primary care office. Instead, the time-honored approach is to heat the end of an unfolded paper clip in a flame and to use that metal to burn through the nail into the hematoma.

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THE AUTHORS REPLY: Although we recognize, as Trautinger points out, that the pathobiology of sunburns may not be typical of most thermal burns, one of the most common causes of first-degree burns is sunburn. Thus, topical diclofenac sodium may be of use for many patients with first-degree burns. There is also evidence that other topical nonsteroidal antiinflammatory agents may be of benefit for thermal injuries. For example, a study of second-degree burns in adult sheep showed that topically applied ibuprofen decreased both local edema and prostanoid production in the burn tissue.¹

We agree that all efforts should be made to reduce the risk of bacterial cross-contamination between patients and wounds. Previous studies have shown that white-coat sleeves often contain patho-

genic bacteria such as *Staphylococcus aureus*.^{2,3} In these studies, a significant proportion of subjects laundered their coats only at monthly intervals. No study has shown contamination of white coats that were properly washed and changed on a daily basis. However, we agree with Guyot et al. that the use of clean short sleeves, as well as proper hand washing and gloves, should be encouraged.

Finally, as noted by Kaufman, in the absence of a portable cautery device, the end of an unfolded paper clip, heated in a flame, may be used to drain a subungual hematoma. Although we too have used this method in the past, in our experience it is now often difficult to find an alcohol lamp, let alone a match to light it.

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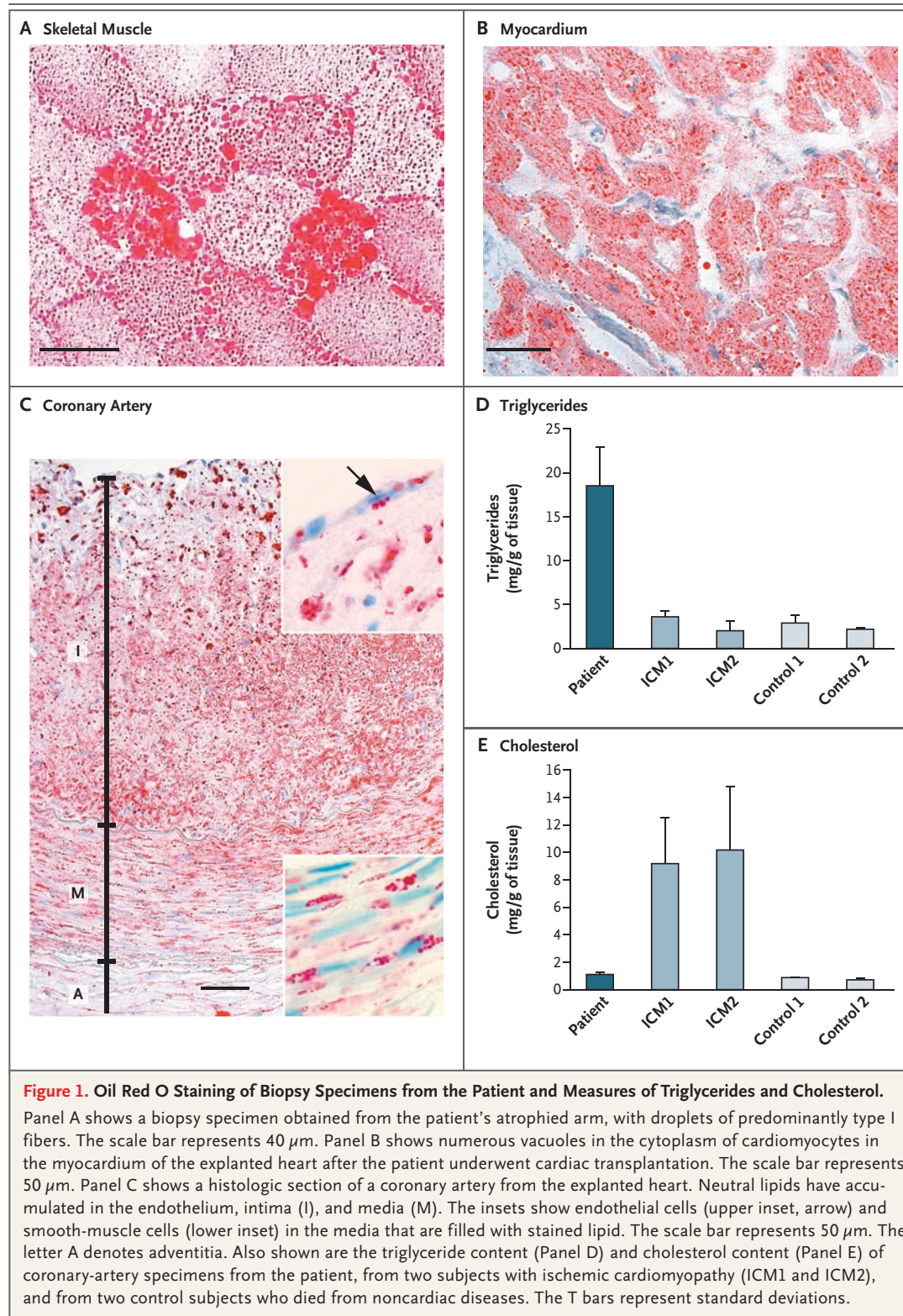
Triglyceride Deposit Cardiomyovasculopathy

TO THE EDITOR: A 41-year-old man was admitted to our hospital with ventricular tachycardia in 2003. Biopsy specimens obtained from the right ventricle showed neutral lipid deposition in cardiomyocytes. In 2004, the patient had catecholamine-dependent congestive heart failure, and a left ventricular assist system was implanted. Skeletal-muscle atrophy in the arms became evident, and staining of biopsy specimens with oil red O showed droplets of predominantly type I fibers (Fig. 1A). Levels of plasma lipids and carnitine were normal.

In June 2007, the patient underwent cardiac transplantation. Microscopical examination of the explanted heart revealed numerous vacuoles that stained positive for oil red O in the cytoplasm of cardiomyocytes (Fig. 1B). The triglyceride content in the left ventricles was markedly increased, as compared with that in three control subjects without heart disease (data not shown). The patient's

coronary arteries showed diffuse intimal thickening and fibroatheromatous lesions. Vacuoles were observed in the cytoplasm of endothelial cells, in the smooth-muscle cells in the media of the coronary arteries (Fig. 1C), and in the foam cells in the intima. Cells that were positive for oil red O staining were seen in the endothelium, intima (Fig. 1C, upper inset), and media (Fig. 1C, lower inset). Surprisingly, the triglyceride content (Fig. 1D), but not the cholesterol content (Fig. 1E), in the patient's atherosclerotic coronary arteries was much higher than that in two control subjects and in two patients with ischemic cardiomyopathy.

To determine the molecular mechanism for this triglyceride deposition, we sequenced the adipose triglyceride lipase gene (*ATGL*, also known as *PNPLA2*), which encodes an essential intracellular triglyceride lipase.¹ The patient was homozygous for a point mutation in exon 7 of *ATGL* (c.865C→T; p.Gln289X), which is identical to a mutation re-



ported by Fischer et al.² in a patient with mild myopathy.

The atherosclerotic lesion that we observed in this patient was unusual³ because the accumulated lipid was triglyceride rather than cholesterol, lipid-laden cells were distributed through all layers of the arterial wall, and the patient had normal plasma triglyceride levels. These phenotypes may result from the mutation in *ATGL*.⁴

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Emergence of Extensive Drug Resistance during Treatment for Multidrug-Resistant Tuberculosis

TO THE EDITOR: We report the development of fluoroquinolone-resistant tuberculosis and extensively drug-resistant tuberculosis during second-line treatment for multidrug-resistant tuberculosis in Karakalpakstan, Uzbekistan. Eighty-seven patients were treated with a regimen containing at least five drugs to which the infecting strain was presumed to be susceptible, according to recommendations from the World Health Organization.^{1,2} We performed drug-susceptibility testing and DNA fingerprinting on *Mycobacterium tuberculosis* isolates collected at baseline and during treatment.

None of the 87 patients had ofloxacin resistance at baseline, yet ofloxacin resistance developed during treatment in 18 patients (21%), and 10 patients (11%) were classified as having extensively drug-resistant tuberculosis.³ Only 5 (28%) of the 18 patients with ofloxacin resistance were successfully treated. Isolates from 13 patients had identical DNA fingerprints throughout treatment, probably reflecting the induction and amplification of ofloxacin resistance. A mixed infection, with two strains at baseline, was found in one patient, whereas the isolates obtained from four patients during treatment had DNA fingerprints that differed from those of the baseline isolates, indicating potential reinfection (Fig. 1).

Among the 13 patients with identical strains at baseline and during treatment, second-line resistance and a severe clinical condition at baseline were significantly associated with the development of ofloxacin resistance on univariate analysis ($P=0.002$ and $P=0.03$, respectively) (see the Supplementary Appendix, available with the full text of this letter at www.nejm.org). Both factors remained significantly associated with fluoroquinolone resistance in a multivariate model ($P=0.007$ and $P=0.03$, respectively). Interestingly, 9 of the 13 patients were infected with a multidrug-resistant tuberculosis clone that is highly prevalent in this region, suggesting a higher propensity of particular strains to acquire resistance. A reduction in population diversity caused by clonal expansion of particular multidrug-resistant strains also renders strain differentiation based on IS6110 fingerprints more difficult. Thus, some of the presumed amplification might represent reinfection with a fluoroquinolone-resistant variant of the same strain.

This study shows that exogenous reinfection with extensively drug-resistant *M. tuberculosis* strains may occur during second-line treatment of multidrug-resistant tuberculosis. The reinfecting strains from three patients showed DNA fingerprint patterns and resistance profiles that were identical to