Contemporary Treatment of Acute Pancreatitis

Series Editor: Kamal M.F. Itani, MD, FACS
Chief of Surgery, Boston VA Health Care System; Professor of Surgery, Boston University; Associate Chief of Surgery, Boston Medical Center and The Brigham and Women’s Hospital, Boston, MA

Contributors:
Scott F. Gallagher, MD
Assistant Professor, Department of Surgery, University of South Florida, Tampa, FL.

Michel M. Murr, MD, FACS
Associate Professor, Department of Surgery, University of South Florida, Tampa, FL.

Table of Contents

Introduction . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 2
Case 1 . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 2
Case 2 . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 5
Case 3 . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 8
Case 4 . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 10
Summary Points . . . . . . . . . . . . . . . . . . . . . . . . . . . 11
References . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 11

Cover Illustration by Joe Wilder, MD

Copyright 2004, Turner White Communications, Inc., 125 Strafford Avenue, Suite 220, Wayne, PA 19087-3391, www.turner-white.com. All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, mechanical, electronic, photocopying, recording, or otherwise, without the prior written permission of Turner White Communications, Inc. The editors are solely responsible for selecting content. Although the editors take great care to ensure accuracy, Turner White Communications, Inc., will not be liable for any errors of omission or inaccuracies in this publication. Opinions expressed are those of the authors and do not necessarily reflect those of Turner White Communications, Inc.
INTRODUCTION

Acute pancreatitis is an inflammatory disease of the pancreas that can progress into a systemic inflammatory state with resultant multi-organ dysfunction. It is estimated that most of the 250,000 individuals diagnosed with acute pancreatitis each year in the United States develop pancreatitis secondary to gallstones or alcohol intake. Most episodes of acute pancreatitis are mild and resolve with minimal specific interventions; however, approximately 2% to 5% of patients develop severe pancreatitis associated with pancreatic necrosis and may require operative intervention.1 The precipitating factor induces acinar cell injury resulting from colocalization of zymogen granules and lysozymes. This localized inflammation in the pancreas can propagate systematically as pancreatic enzymes induce cytokine production in the pancreas and affected organs, such as the lungs, liver, and kidneys. Despite advancements in our understanding of the pathophysiology of acute pancreatitis, the mainstay of treatment remains nonoperative, except in patients with necrotizing pancreatitis. Multiple clinical trials utilizing anticytokine agents have failed to reduce mortality.2,3 This monograph uses hypothetical cases to review the evaluation and management of acute pancreatitis.

NOMENCLATURE

In response to confusion in nomenclature, a clinically based classification system has been adopted by many experts worldwide and will be used throughout this text4:

Mild acute pancreatitis. An acute inflammation of the pancreas with minimal distant organ dysfunction and an uneventful recovery.

Severe acute pancreatitis. An acute inflammation of the pancreas associated with organ failure and/or local complications such as necrosis, abscess, or pseudocyst.

Acute fluid collection. Acute fluid collections occur early in the course of acute pancreatitis, are located in or near the pancreas, and always lack a wall of granulation or fibrous tissue. This is the most misunderstood term and is commonly confused with a pseudocyst.

Pancreatic necrosis. Diffuse or focal areas of nonviable pancreatic parenchyma, which are typically associated with peripancreatic fat necrosis.

Acute pseudocyst. A collection of pancreatic fluid enclosed by a wall of fibrous or granulation tissue that arises as a consequence of acute pancreatitis, chronic pancreatitis, or trauma to the pancreas.

Pancreatic abscess is a collection of pus, usually in proximity to the pancreas, containing little or no pancreatic necrosis, which arises as a consequence of acute pancreatitis or trauma to the pancreas.

CASE 1

PATIENT PRESENTATION

Patient 1 is a 42-year-old man who is admitted to the hospital with acute onset of abdominal pain, nausea, and vomiting. He has epigastric tenderness but no peritoneal signs. Admission laboratory test results are: hemoglobin, 16.2 mg/dL; leukocyte count, 18 × 10^3/mm^3; serum creatinine, 1.3 mg/dL; total bilirubin, 1.8 mg/dL; amylase, 432 U/L; lipase, 242 U/L; and alkaline phosphatase level, 260 U/L.

• What is an appropriate diagnostic plan for this patient?

DISCUSSION

This patient’s clinical presentation is consistent with acute pancreatitis. Other abdominal conditions that mimic acute pancreatitis can be ruled out by history, physical examination, and laboratory data; however, imaging modalities should be used to confirm the diagnosis of acute pancreatitis.

Clinical Presentation

The cardinal clinical symptom of acute pancreatitis is constant epigastric pain of insidious onset, often radiating to the back. Other clinical findings include anorexia,