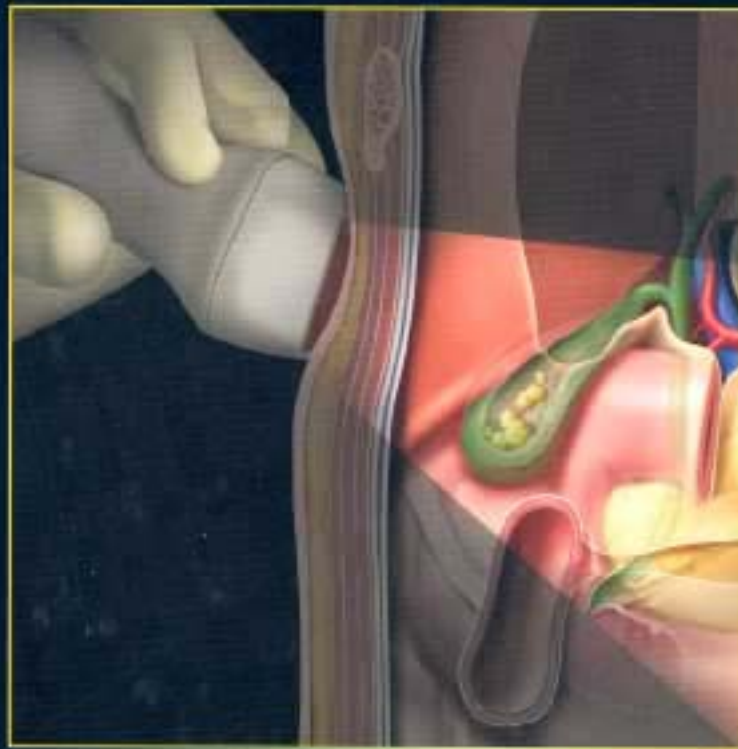


DIAGNOSTIC IMAGING



ULTRASOUND

Ahuja

Griffith • Wong • Antonio • Chu • Ho
Lolge • Paunipagar • Kennedy • Zwiebel
Sohaey • Ho • Woodward



مرکز خدمات فرهنگی سالکان

همگام با توسعه علمی و فرهنگی جهان معاصرو استفاده روزافزون کامپیوتر در بین جوامع بشری خصوصا رشته های مختلف علوم و استفاده بهینه از آخرین یافته های پزشکی دنیا و ارائه این یافته ها در قالب نرم افزارهای پزشکی (e-Book, VCD, DVD, Multimedia) ما را بر آن داشت که با گردآوری و ارائه این یافته ها گامی کوچک در راه ارتقاء سطح علمی متخصصین کلیه رشته های پزشکی کشور به صورت سمعی و بصری برداریم. امید است مشوق ما در این راه باشید.

لذا علاقه مندان می توانند برای دریافت هر یک از محصولات ارائه شده به ازاء هر CD مبلغ ۵۰۰۰ تومان به حساب سپهر به شماره: ۰۳۰۶۵۰۰۱۰۰۰۰۳ بانک صادرات ایران (قابل واریز از سراسر کشور) به نام جواد شیرنگ واریز و پس از فاکس فیش فوق به همراه نشانی دقیق خریدار نسبت به خرید اقلام و دریافت کالای مورد نظر خود اقدام نمایند.

نشانی مرکز: تهران - میدان انقلاب - ابتدای کارگر شمالی - ترسیده به بلوار

کشاوری - ابتدای خیابان میرخانی (نصرت) - پلاک ۶۴ - طبقه همکف

تلفن: ۴-۶۶۵۶۵۰۲۳ تلفکس: ۶۶۹۳۱۴۷۹ همراه: ۰۹۱۲۱۳۷۲۳۶۱

DIAGNOSTIC IMAGING ULTRASOUND



DIAGNOSTIC IMAGING ULTRASOUND

Anil T. Ahuja, MD, FRCR

Professor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China

James F. Griffith, MBCh, FRCR

Professor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China

K.T. Wong, MBChB, FRCR

Honorary Clinical Assistant Professor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China

Gregory E. Antonio, MD, FRANZCR

Associate Professor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China

Winnie C.W. Chu, MBChB, FRCR

Associate Professor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China

Stella S.Y. Ho, PhD, RDMS

Adjunct Assistant Professor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China

Shlok J. Lolge, MD

Clinical Tutor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China

Bhawan K. Paunipagar, MD, DNB

Clinical Tutor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China

Anne Kennedy, MD

Director of Women's Imaging
Associate Professor of Radiology
Adjunct Associate Professor of Obstetrics and Gynecology
University of Utah School of Medicine
Salt Lake City, Utah

Roya Sohaey, MD

Director of Ultrasound
Associate Professor of Radiology
Associate Professor of Obstetrics and Gynecology
Oregon Health and Science University
Portland, Oregon

Simon S.M. Ho, MBBS, FRCR

Assistant Professor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China

Paula J. Woodward, MD

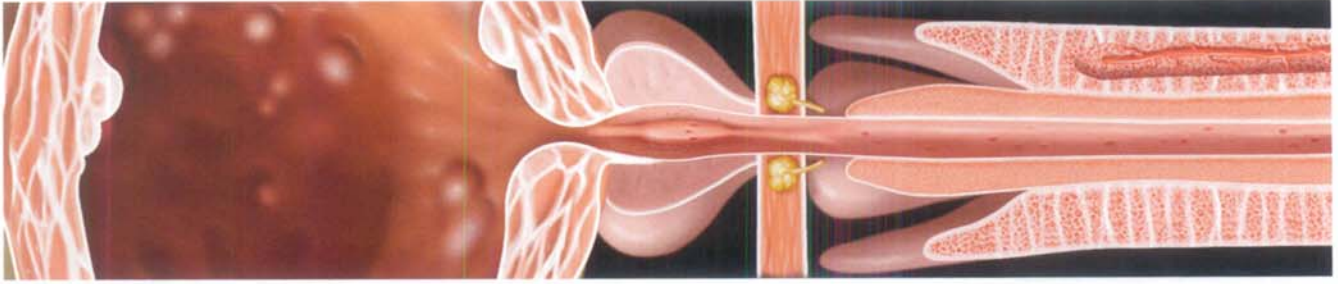
Professor of Radiology
Adjunct Professor of Obstetrics and Gynecology
University of Utah School of Medicine
Salt Lake City, Utah

William J. Zwiebel, MD

Former Professor of Radiology
University of Utah School of Medicine
Salt Lake City, Utah



Names you know, content you trust!



FOREWORD

It is a particular pleasure to be asked to provide a foreword for Anil Ahuja's timely contribution to the literature. I have long maintained that ultrasound is one of the most difficult of all imaging techniques, largely because the only person who can really assess the clinical problem is the operator who performed the study! Because of this it is critical that all those carrying out ultrasound are trained to an appropriate level. This book will help such training and provide a constant source of reference for workers faced with an unexpected lesion.

It is also pleasing to see a comprehensive text on ultrasound being developed at a time when many people wish to be trained in just one particular clinical subspecialty. While a musculoskeletal radiologist may become extremely competent in musculoskeletal ultrasound, there is still a pressing need for experts to be able to cover the whole range of ultrasound procedures. They will be the only people to advise on such developments as probe technology, ultrasound contrast agents, etc. There is no certainty that a patient presenting with a problem seemingly related to one body system may not have a lesion in another! Hence the importance of being able to switch from ultrasound of the hip to ultrasound of the iliac fossa. This book will assist such a comprehensive ultrasound approach.

With the rapidly increasing technical specifications of ultrasound machines and relative reduction in costs, it is not at all improbable that every ward of a hospital might soon "own" their own ultrasound machine. Indeed, in time, a personal ultrasound machine may become even more important than a stethoscope! These developments mean that ultrasound will have to be learned by a larger range of personnel and supervised to appropriate standards. This book will help all those participating in the wider scheme of ultrasound training. It will also be of enormous use to radiologists learning the technique and studying for postgraduate examinations.

The authors and the publisher have all done a superb job in making this book so attractive. I strongly believe that it will become *the* essential ultrasound text book and that Anil Ahuja's name will, as a result, become even more widely recognized within enlightened ultrasound departments. Congratulations to all.

Adrian K. Dixon, MD, FRCR, FRCP, FRCS, FMedSci
Professor of Radiology
University of Cambridge
Honorary Consultant Radiologist
Addenbrooke's Hospital
Cambridge, United Kingdom



PREFACE

I have been fortunate to know Drs. Ric Harnsberger & Anne Osborn. What started as an academic relationship has over the years developed into a close friendship. I am privileged to have been asked to undertake this project and it is their vision, enthusiasm, and support that has helped me accomplish this task.

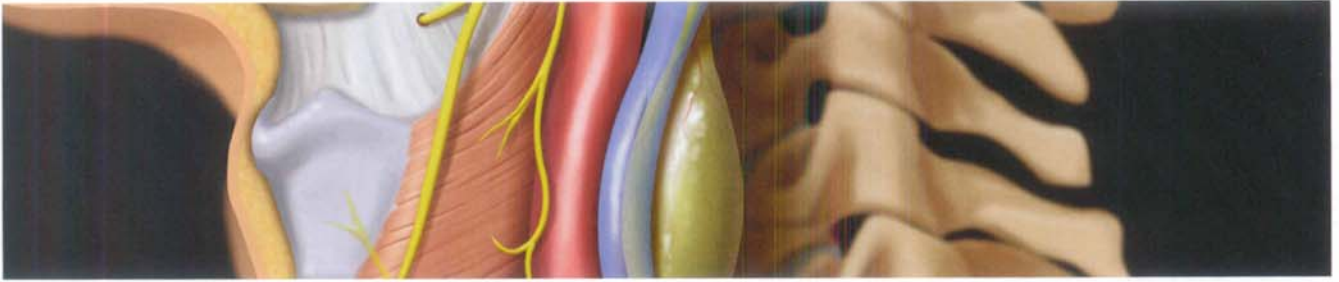
This book is unique in the Diagnostic Imaging series as it deals with a modality rather than a clinical specialty such as Head & Neck or Neuroradiology. Its scope is therefore wide, but this book is limited to clinical conditions that general sonologists, radiologists, clinicians, & residents commonly encounter in routine practice. The discussion of the role of ultrasound in Obstetric & Pediatric imaging has been restricted as these have been dealt with separately in other books in the Diagnostic Imaging series.

Although it is a book on ultrasound, you will find information & images from other modalities. In this era of multimodality imaging, techniques complement each other in diagnosis & management of patients. It is therefore essential to be familiar with the role of ultrasound in relation to other modalities. Each diagnosis contains common imaging appearances, basic pathology, treatment options and prognosis. The section introductions contain relevant information on anatomy, practical tips, technical parameters for optimal scanning. The protocol section includes indications where other imaging modalities may be necessary. The image annotation & key facts box crystallize relevant information and are ideal for those with short attention spans.

This book would not have been possible without the help of friends (authors and contributors) from various parts of the world. They have been generous with their images, expertise, time and patience, and I remain forever indebted. In particular I would like to acknowledge Dr. Chander Lulla & Prof. Ravi Ramakantan for their generosity with images and Prof. William Zwiebel & Prof. Paula Woodward for their help in preparing the table of contents. The team from Amirsys has been superb. Despite being in different continents & time zones they have patiently guided me along the entire process and none of this would have been possible without their help. Lastly, on behalf of all the authors I would like to thank sonographers in our respective departments for their dedication to this unique imaging modality.

The preparation of this book has brought members of my department closer, helped make new friendships, & consolidate old ones. I have enjoyed the process & hope you find this book useful.

Anil T. Ahuja, MD, FRCR
Professor
Department of Diagnostic Radiology and Organ Imaging
The Chinese University of Hong Kong
Hong Kong, China



ACKNOWLEDGMENTS

Illustrations

Lane R. Bennion, MS
Richard Coombs, MS
Wes Price, MS

Image/Text Editing

Douglas Grant Jackson
Amanda Hurtado
Roth LaFleur

Medical Text Editing

Paula J. Woodward, MD
Anne Kennedy, MD
Daniel N. Sommers, MD
Marta E. Heilbrun, MD
Akram M. Shaaban, MBBCh

Case Management

Christopher Odekirk

Contributors

Jitendra Astekar
Nitin Chaubal
Avinash Gutte
Mukund Joshi
Sudheer Joshi
Arun Kinare
Ann King
William K.M. Kong
Aniruddha Kulkarni
Paul S.F. Lee
Tom W.K. Lee
Yolanda Y.P. Lee
Darshana Rasalkar
Rhian Rhys
Iain Stewart
Ki Wang
Simon C.H. Yu

Associate Editor

Kaerli Main

Production Lead

Melissa A. Hoopes



SECTIONS

- Liver** 1
- Biliary System** 2
- Pancreas** 3
- Spleen** 4
- Urinary Tract** 5
- Renal Transplants** 6
- Adrenal Gland** 7
- Abdominal Wall/Peritoneal Cavity** 8
- Female Pelvis** 9
- Scrotum** 10
- Head and Neck** 11
- Breast** 12
- Musculoskeletal** 13
- Vascular** 14

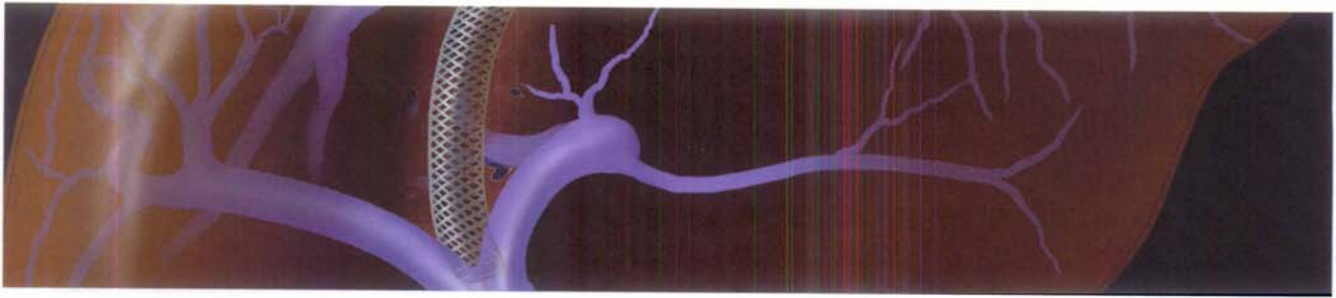


TABLE OF CONTENTS

SECTION 1 Liver

Introduction and Overview

Hepatic Sonography <i>Gregory E. Antonio, MD, FRANZCR</i>	1-2
--	-----

Diffuse Parenchymal Disease

Acute Hepatitis <i>Gregory E. Antonio, MD, FRANZCR</i>	1-6
Cirrhosis, Hepatic <i>Gregory E. Antonio, MD, FRANZCR</i>	1-10
Schistosomiasis, Hepatic <i>Gregory E. Antonio, MD, FRANZCR</i>	1-16
Steatosis, Hepatic <i>Gregory E. Antonio, MD, FRANZCR</i>	1-20
Parenchymal Calcification, Hepatic <i>Gregory E. Antonio, MD, FRANZCR</i>	1-24
Diffuse Microabscesses, Hepatic <i>Gregory E. Antonio, MD, FRANZCR</i>	1-26
Lymphoma, Hepatic <i>Gregory E. Antonio, MD, FRANZCR</i>	1-28

Cyst and Cyst-Like Lesions

Hepatic Cyst <i>Gregory E. Antonio, MD, FRANZCR</i>	1-32
Caroli Disease <i>Gregory E. Antonio, MD, FRANZCR</i>	1-36
Biloma <i>Gregory E. Antonio, MD, FRANZCR</i>	1-40
Pyogenic Hepatic Abscess <i>Gregory E. Antonio, MD, FRANZCR</i>	1-42
Pyogenic Peri-Hepatic Abscess <i>Gregory E. Antonio, MD, FRANZCR</i>	1-48
Amebic Hepatic Abscess <i>Gregory E. Antonio, MD, FRANZCR</i>	1-50
Hepatic Echinococcus Cyst <i>Gregory E. Antonio, MD, FRANZCR</i>	1-54
Hepatic Trauma <i>Gregory E. Antonio, MD, FRANZCR</i>	1-60

Focal Solid Masses

Hepatic Adenoma <i>Gregory E. Antonio, MD, FRANZCR</i>	1-64
Focal Nodular Hyperplasia <i>Gregory E. Antonio, MD, FRANZCR</i>	1-68
Hepatocellular Carcinoma <i>Gregory E. Antonio, MD, FRANZCR</i>	1-72
Hepatic Cavernous Hemangioma <i>Gregory E. Antonio, MD, FRANZCR</i>	1-78
Hepatic Metastases <i>Gregory E. Antonio, MD, FRANZCR</i>	1-82

Vascular Conditions

Portal Hypertension <i>William J. Zwiebel, MD</i>	1-88
Porto-Systemic Collaterals <i>William J. Zwiebel, MD</i>	1-92
TIPS Shunts <i>William J. Zwiebel, MD</i>	1-96
Portal Vein Occlusion <i>William J. Zwiebel, MD</i>	1-100
Budd-Chiari Syndrome <i>William J. Zwiebel, MD</i>	1-104
Portal Vein Gas <i>William J. Zwiebel, MD</i>	1-108
Post-Transplant Liver <i>William J. Zwiebel, MD</i>	1-110

SECTION 2 Biliary System

Introduction and Overview

Biliary Sonography <i>K.T. Wong, MBChB, FRCR</i>	2-2
---	-----

Gallstones and Mimics

Cholelithiasis <i>K.T. Wong, MBChB, FRCR</i>	2-6
Echogenic Bile, Blood Clots, Parasites <i>K.T. Wong, MBChB, FRCR</i>	2-12

Gallbladder Cholesterol Polyp
K.T. Wong, MBChB, FRCR

2-16

Serous Cystadenoma
K.T. Wong, MBChB, FRCR

3-20

Gallbladder Wall Pathology

Thickened Gallbladder Wall
K.T. Wong, MBChB, FRCR

2-20

Acute Calculous Cholecystitis
K.T. Wong, MBChB, FRCR

2-22

Acute Acalculous Cholecystitis
K.T. Wong, MBChB, FRCR

2-26

Chronic Cholecystitis
K.T. Wong, MBChB, FRCR

2-28

Porcelain Gallbladder
K.T. Wong, MBChB, FRCR

2-30

Hyperplastic Cholecystosis
K.T. Wong, MBChB, FRCR

2-32

Gallbladder Carcinoma
K.T. Wong, MBChB, FRCR

2-36

Ductal Pathology

Biliary Ductal Dilatation
K.T. Wong, MBChB, FRCR

2-40

Choledochal Cyst
K.T. Wong, MBChB, FRCR

2-42

Choledocholithiasis
K.T. Wong, MBChB, FRCR

2-46

Biliary Ductal Gas
K.T. Wong, MBChB, FRCR

2-50

Cholangiocarcinoma
K.T. Wong, MBChB, FRCR

2-52

Ascending Cholangitis
K.T. Wong, MBChB, FRCR

2-56

Recurrent Pyogenic Cholangitis
K.T. Wong, MBChB, FRCR

2-60

AIDS-Related Cholangiopathy
K.T. Wong, MBChB, FRCR

2-64

SECTION 3 Pancreas

Introduction and Overview

Pancreatic Sonography
K.T. Wong, MBChB, FRCR

3-2

Pancreatitis

Acute Pancreatitis
K.T. Wong, MBChB, FRCR

3-6

Pancreatic Pseudocysts
K.T. Wong, MBChB, FRCR

3-10

Chronic Pancreatitis
K.T. Wong, MBChB, FRCR

3-14

Simple Cysts and Cystic Neoplasms

Mucinous Cystic Pancreatic Tumor
K.T. Wong, MBChB, FRCR

3-18

Solid-Appearing Pancreatic Neoplasms

Ductal Pancreatic Carcinoma
K.T. Wong, MBChB, FRCR

3-24

Islet Cell Tumors
K.T. Wong, MBChB, FRCR

3-28

Solid and Papillary Neoplasm
K.T. Wong, MBChB, FRCR

3-32

SECTION 4 Spleen

Introduction and Overview

Splenic Sonography
Shlok J. Lolge, MD

4-2

Spleen

Accessory Spleen
Shlok J. Lolge, MD

4-6

Splenomegaly
Shlok J. Lolge, MD

4-8

Cysts & Cyst-Like Splenic Lesions
Shlok J. Lolge, MD

4-12

Splenic Tumors
Shlok J. Lolge, MD

4-16

Splenic Trauma
Shlok J. Lolge, MD

4-22

Splenic Calcifications
Shlok J. Lolge, MD

4-26

Splenic Vascular Disorders
William J. Zwiebel, MD

4-28

SECTION 5 Urinary Tract

Introduction and Overview

Urinary Tract Sonography
Winnie C.W. Chu, MBChB, FRCR

5-2

Normal Variants and Pseudolesions

Column of Bertin, Kidney
Winnie C.W. Chu, MBChB, FRCR

5-6

Renal Junction Line
Winnie C.W. Chu, MBChB, FRCR

5-8

Renal Ectopia
Winnie C.W. Chu, MBChB, FRCR

5-10

Horseshoe Kidney
Winnie C.W. Chu, MBChB, FRCR

5-14

Ureteral Duplication
Winnie C.W. Chu, MBChB, FRCR

5-18

Ureteral Ectopia
Winnie C.W. Chu, MBChB, FRCR

5-22

Ureteropelvic Junction Obstruction 5-26
Winnie C.W. Chu, MBChB, FRCR

Calculi and Calcinosis

Urolithiasis 5-30
Winnie C.W. Chu, MBChB, FRCR

Nephrocalcinosis 5-36
Winnie C.W. Chu, MBChB, FRCR

Hydronephrosis 5-40
Winnie C.W. Chu, MBChB, FRCR

Cysts and Cystic Disorders

Simple Renal Cyst 5-44
Stella S.Y. Ho, PhD, RDMS

Complex Renal Cyst 5-48
Stella S.Y. Ho, PhD, RDMS

Cystic Disease of Dialysis 5-54
Stella S.Y. Ho, PhD, RDMS

Multilocular Cystic Nephroma 5-58
Winnie C.W. Chu, MBChB, FRCR

Renal Papillary Necrosis 5-60
Stella S.Y. Ho, PhD, RDMS

Renal Trauma 5-62
Winnie C.W. Chu, MBChB, FRCR

Perinephric Fluid Collections 5-66
Stella S.Y. Ho, PhD, RDMS

Urinary Tract Infection

Acute Pyelonephritis 5-70
Stella S.Y. Ho, PhD, RDMS

Focal Bacterial Nephritis 5-72
Stella S.Y. Ho, PhD, RDMS

Emphysematous Pyelonephritis 5-74
Winnie C.W. Chu, MBChB, FRCR

Pyonephrosis 5-76
Winnie C.W. Chu, MBChB, FRCR

Renal Abscess 5-78
Stella S.Y. Ho, PhD, RDMS

Xanthogranulomatous Pyelonephritis 5-80
Stella S.Y. Ho, PhD, RDMS

Urinary Tract Tuberculosis 5-82
Stella S.Y. Ho, PhD, RDMS

Solid Renal Neoplasms

Renal Cell Carcinoma 5-86
Stella S.Y. Ho, PhD, RDMS

Renal Metastases 5-92
Stella S.Y. Ho, PhD, RDMS

Renal Angiomyolipoma 5-94
Stella S.Y. Ho, PhD, RDMS

Transitional Cell Carcinoma 5-98
Stella S.Y. Ho, PhD, RDMS

Renal Lymphoma 5-104
Stella S.Y. Ho, PhD, RDMS

Vascular Conditions

Renal Artery Stenosis 5-108
William J. Zwiebel, MD

Renal Vein Thrombosis 5-112
William J. Zwiebel, MD

Prostate

Prostatic Hypertrophy 5-116
Stella S.Y. Ho, PhD, RDMS

Prostatic Carcinoma 5-120
Stella S.Y. Ho, PhD, RDMS

Bladder

Diffuse Bladder Wall Thickening 5-124
Winnie C.W. Chu, MBChB, FRCR

Bladder Carcinoma 5-128
Winnie C.W. Chu, MBChB, FRCR

Ureterocele 5-132
Winnie C.W. Chu, MBChB, FRCR

Bladder Diverticulum 5-138
Winnie C.W. Chu, MBChB, FRCR

Bladder Calculi 5-142
Winnie C.W. Chu, MBChB, FRCR

Schistosomiasis, Bladder 5-144
Winnie C.W. Chu, MBChB, FRCR

SECTION 6 Renal Transplants

Introduction and Overview

Sonographic Features of Renal Allografts 6-2
Stella S.Y. Ho, PhD, RDMS

Renal Transplants

Allograft Hydronephrosis 6-6
Stella S.Y. Ho, PhD, RDMS

Perigraft Fluid Collections 6-10
Stella S.Y. Ho, PhD, RDMS

Allograft Rejection 6-14
Stella S.Y. Ho, PhD, RDMS

Renal Transplant Vascular Disorders 6-18
William J. Zwiebel, MD

Renal Transplant Fistula/Pseudoaneurysm 6-22
William J. Zwiebel, MD

SECTION 7 Adrenal Gland

Adrenal Hemorrhage 7-2
Shlok J. Lolge, MD

Myelolipoma 7-6
Shlok J. Lolge, MD

Adrenal Cysts 7-10
Shlok J. Lolge, MD

Pheochromocytoma <i>Shlok J. Lolge, MD</i>	7-12
Adrenal Carcinoma <i>Shlok J. Lolge, MD</i>	7-16

SECTION 8 Abdominal Wall/Peritoneal Cavity

Abdominal Wall Hernia <i>Shlok J. Lolge, MD</i>	8-2
Groin Hernias <i>Shlok J. Lolge, MD</i>	8-6
Ascites <i>Shlok J. Lolge, MD</i>	8-10
Peritoneal Carcinomatosis <i>Shlok J. Lolge, MD</i>	8-14
Peritoneal Space Abscess <i>Shlok J. Lolge, MD</i>	8-18
Appendicitis <i>Shlok J. Lolge, MD</i>	8-22
Intussusception <i>Shlok J. Lolge, MD</i>	8-26

SECTION 9 Female Pelvis

Introduction and Overview

Pelvic Anatomy & Imaging Issues <i>Paula J. Woodward, MD</i>	9-2
---	-----

Cervical and Myometrial Pathology

Nabothian Cyst <i>Roya Sohaey, MD</i>	9-6
Cervical Carcinoma <i>Anne Kennedy, MD</i>	9-10
Uterine Adenomyosis <i>Roya Sohaey, MD & Steven A. Larsen, MD</i>	9-14
Uterine Leiomyoma <i>Roya Sohaey, MD & Steven A. Larsen, MD</i>	9-18

Endometrial Disorders

Hematometocolpos <i>Anne Kennedy, MD</i>	9-22
Endometrial Polyp <i>Anne Kennedy, MD</i>	9-26
Endometrial Hyperplasia <i>Anne Kennedy, MD</i>	9-32
Endometrial Carcinoma <i>Anne Kennedy, MD</i>	9-34
Endometritis <i>Paula J. Woodward, MD</i>	9-38
Myometrial & Endometrial Calcification <i>Roya Sohaey, MD</i>	9-42
Synechiae <i>Paula J. Woodward, MD</i>	9-44

Pregnancy-Related Disorders

Ectopic Pregnancy <i>Roya Sohaey, MD</i>	9-48
Interstitial Ectopic Pregnancy <i>Paula J. Woodward, MD</i>	9-52
Failed First Trimester Pregnancy <i>Anne Kennedy, MD</i>	9-56
Retained Products of Conception <i>Paula J. Woodward, MD</i>	9-60
Gestational Trophoblastic Neoplasm <i>Roya Sohaey, MD</i>	9-62

Ovarian Cysts and Cystic Neoplasms

Functional Ovarian Cyst <i>Anne Kennedy, MD</i>	9-66
Hemorrhagic Cyst <i>Anne Kennedy, MD</i>	9-70
Ovarian Hyperstimulation <i>Karen Y. Oh, MD</i>	9-76
Serous Ovarian Cystadenoma/Carcinoma <i>Paula J. Woodward, MD</i>	9-80
Mucinous Ovarian Cystadenoma/Carcinoma <i>Paula J. Woodward, MD</i>	9-84
Ovarian Teratoma <i>Anne Kennedy, MD</i>	9-88
Polycystic Ovarian Syndrome <i>Anne Kennedy, MD</i>	9-94

Non-Ovarian Cystic Masses

Hydrosalpinx <i>Roya Sohaey, MD</i>	9-98
Tubovarian Abscess <i>Roya Sohaey, MD</i>	9-100
Parovarian Cysts <i>Roya Sohaey, MD</i>	9-104

Vaginal and Vulvar Cysts

Bartholin Cyst <i>Anne Kennedy, MD</i>	9-106
Gartner Duct Cyst <i>Anne Kennedy, MD</i>	9-108

Miscellaneous Ovarian Masses

Sex Cord-Stromal Tumor <i>Paula J. Woodward, MD</i>	9-112
Ovarian Fibrothecoma <i>Paula J. Woodward, MD & Kaerli Main</i>	9-116
Peritoneal Inclusion Cyst <i>Roya Sohaey, MD & Steven A. Larsen, MD</i>	9-120
Endometriomas <i>Roya Sohaey, MD</i>	9-124

SECTION 10 Scrotum

Introduction and Overview

Scrotal Sonography	10-2
<i>Bhawan K. Paunipagar, MD, DNB</i>	

Scrotum

Testicular Atrophy	10-6
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Undescended Testis	10-8
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Hydrocele	10-10
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Testicular & Epididymal Cysts	10-12
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Epidermoid Cyst	10-16
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Testicular Carcinoma	10-18
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Gonadal Stromal Tumor	10-22
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Testicular Microlithiasis	10-24
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Tubular Ectasia	10-26
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Testicular Torsion/Infarction	10-28
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Scrotal Trauma	10-32
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Epididymal Masses	10-34
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Epididymitis/Orchitis	10-38
<i>Bhawan K. Paunipagar, MD, DNB</i>	
Varicocele	10-44
<i>Bhawan K. Paunipagar, MD, DNB</i>	

SECTION 11 Head and Neck

Introduction and Overview

Head & Neck Sonography	11-2
<i>Anil T. Ahuja, MD, FRCR</i>	

Thyroid and Parathyroid

Differentiated Thyroid Carcinoma	11-6
<i>Anil T. Ahuja, MD, FRCR</i>	
Medullary Thyroid Carcinoma	11-12
<i>Anil T. Ahuja, MD, FRCR</i>	
Anaplastic Thyroid Carcinoma	11-16
<i>Anil T. Ahuja, MD, FRCR</i>	
Thyroid Non-Hodgkin Lymphoma	11-20
<i>Anil T. Ahuja, MD, FRCR</i>	
Hashimoto Thyroiditis	11-24
<i>Anil T. Ahuja, MD, FRCR</i>	

Multinodular Goiter	11-28
<i>Anil T. Ahuja, MD, FRCR</i>	
Graves Disease	11-32
<i>Anil T. Ahuja, MD, FRCR</i>	
Parathyroid Adenoma, Visceral Space	11-36
<i>Anil T. Ahuja, MD, FRCR</i>	

Adenopathy

Reactive Adenopathy	11-40
<i>Anil T. Ahuja, MD, FRCR</i>	
Squamous Cell Carcinoma Nodes	11-42
<i>Anil T. Ahuja, MD, FRCR</i>	
Tuberculous Adenopathy	11-46
<i>Anil T. Ahuja, MD, FRCR</i>	
Non-Hodgkin Lymphoma Nodes	11-48
<i>Anil T. Ahuja, MD, FRCR</i>	
Systemic Metastases, Neck Nodes	11-52
<i>Anil T. Ahuja, MD, FRCR</i>	

Salivary Glands

Sialadenitis	11-54
<i>Anil T. Ahuja, MD, FRCR</i>	
Benign Mixed Tumor, Submandibular Gland	11-58
<i>Anil T. Ahuja, MD, FRCR</i>	
Submandibular Gland Carcinoma	11-62
<i>Anil T. Ahuja, MD, FRCR</i>	
Kimura Disease	11-64
<i>Anil T. Ahuja, MD, FRCR</i>	
Sjogren Syndrome, Parotid	11-68
<i>Anil T. Ahuja, MD, FRCR</i>	

Parotid Space

Benign Mixed Tumor, Parotid	11-72
<i>Anil T. Ahuja, MD, FRCR</i>	
Warthin Tumor	11-76
<i>Anil T. Ahuja, MD, FRCR</i>	
Mucoepidermoid Carcinoma, Parotid	11-80
<i>Anil T. Ahuja, MD, FRCR</i>	
Adenoid Cystic Carcinoma, Parotid	11-84
<i>Anil T. Ahuja, MD, FRCR</i>	

Miscellaneous Lumps

Benign Masseter Muscle Hypertrophy	11-86
<i>Anil T. Ahuja, MD, FRCR</i>	
Ranula	11-88
<i>Anil T. Ahuja, MD, FRCR</i>	
Lymphangioma	11-92
<i>Anil T. Ahuja, MD, FRCR</i>	
2nd Branchial Cleft Cyst	11-98
<i>Anil T. Ahuja, MD, FRCR</i>	
Carotid Body Paraganglioma	11-102
<i>Anil T. Ahuja, MD, FRCR</i>	
Thyroglossal Duct Cyst	11-106
<i>Anil T. Ahuja, MD, FRCR</i>	
Lipoma	11-110
<i>Anil T. Ahuja, MD, FRCR</i>	

Vagus Schwannoma, Infrahyoid Carotid Space <i>Anil T. Ahuja, MD, FRCR</i>	11-114
Brachial Plexus Schwannoma <i>Anil T. Ahuja, MD, FRCR</i>	11-118

Congenital

Venous Vascular Malformation <i>Anil T. Ahuja, MD, FRCR</i>	11-120
Dermoid and Epidermoid <i>Anil T. Ahuja, MD, FRCR</i>	11-124

SECTION 12 Breast

Introduction and Overview

Breast Sonography <i>Anne Kennedy, MD</i>	12-2
--	------

Breast

Cysts & Cyst-Like Lesions <i>Kathleen H. Puglia, MD</i>	12-6
Breast Cancer, DCIS <i>Karen Y. Oh, MD</i>	12-10
Solid Non-Malignant Breast Masses <i>Kathleen H. Puglia, MD & Anne Kennedy, MD</i>	12-14
Fat Necrosis <i>Kathleen H. Puglia, MD</i>	12-18
Breast Abscess <i>Kathleen H. Puglia, MD</i>	12-22
Intraductal Papilloma <i>Karen Y. Oh, MD</i>	12-26
Ductal Ectasia <i>Kathleen H. Puglia, MD</i>	12-30
Gynecomastia <i>Karen Y. Oh, MD</i>	12-34

SECTION 13 Musculoskeletal

Introduction and Overview

Musculoskeletal Sonography <i>James F. Griffith, MBCh, FRCR</i>	13-2
--	------

Tendon Disorders

Rotator Cuff Tendinosis <i>James F. Griffith, MBCh, FRCR</i>	13-6
Rotator Cuff Tear <i>James F. Griffith, MBCh, FRCR</i>	13-10
Non-Rotator Cuff Tendinosis <i>James F. Griffith, MBCh, FRCR</i>	13-16
Non-Rotator Cuff Tendon Tears <i>James F. Griffith, MBCh, FRCR</i>	13-22
Tenosynovitis <i>James F. Griffith, MBCh, FRCR</i>	13-28

Elbow Epicondylitis <i>James F. Griffith, MBCh, FRCR</i>	13-32
---	-------

Subcutaneous and Muscle Injury

Fat Injury <i>James F. Griffith, MBCh, FRCR</i>	13-38
Muscle Infarction <i>James F. Griffith, MBCh, FRCR</i>	13-42
Muscle Injury <i>James F. Griffith, MBCh, FRCR</i>	13-46

Arthropathies

Osteoarthritis <i>James F. Griffith, MBCh, FRCR</i>	13-50
Inflammatory Arthritis <i>James F. Griffith, MBCh, FRCR</i>	13-54
Developmental Hip Dysplasia <i>James F. Griffith, MBCh, FRCR</i>	13-58

Neurovascular Abnormalities

Nerve Injury <i>James F. Griffith, MBCh, FRCR</i>	13-62
Peripheral Nerve Sheath Tumor <i>James F. Griffith, MBCh, FRCR</i>	13-66
Carpal Tunnel Syndrome <i>James F. Griffith, MBCh, FRCR</i>	13-72

Infection

Soft Tissue Infection <i>James F. Griffith, MBCh, FRCR</i>	13-76
Bone Infection <i>James F. Griffith, MBCh, FRCR</i>	13-82
Joint Infection <i>James F. Griffith, MBCh, FRCR</i>	13-88
Post-Operative Infection <i>James F. Griffith, MBCh, FRCR</i>	13-92

Articular and Para-Articular Masses

Hemarthrosis & Lipohemarthrosis <i>James F. Griffith, MBCh, FRCR</i>	13-96
Gout and Pseudogout <i>James F. Griffith, MBCh, FRCR</i>	13-100
Baker Cyst <i>James F. Griffith, MBCh, FRCR</i>	13-104
Bursitis <i>James F. Griffith, MBCh, FRCR</i>	13-110
Ganglion Cyst <i>James F. Griffith, MBCh, FRCR</i>	13-114
Parameniscal Cyst <i>James F. Griffith, MBCh, FRCR</i>	13-118
Synovial Tumor <i>James F. Griffith, MBCh, FRCR</i>	13-122

Soft Tissue Tumors

Plantar Fasciitis & Fibromatosis <i>James F. Griffith, MBCh, FRCR</i>	13-126
--	--------

Peripheral Lipoma <i>James F. Griffith, MBCh, FRCR</i>	13-130
Soft Tissue Sarcoma <i>James F. Griffith, MBCh, FRCR</i>	13-134
Peripheral Vascular Anomaly <i>James F. Griffith, MBCh, FRCR</i>	13-138
Foreign Body and Injection Granulomas <i>James F. Griffith, MBCh, FRCR</i>	13-144

SECTION 14 Vascular

Introduction and Overview

Vascular Imaging & Doppler <i>Simon S.M. Ho, MBBS, FRCR</i>	14-2
--	------

Cerebrovascular

Carotid Stenosis/Occlusion <i>Simon S.M. Ho, MBBS, FRCR</i>	14-6
Vertebral Stenosis/Occlusion <i>Simon S.M. Ho, MBBS, FRCR</i>	14-12

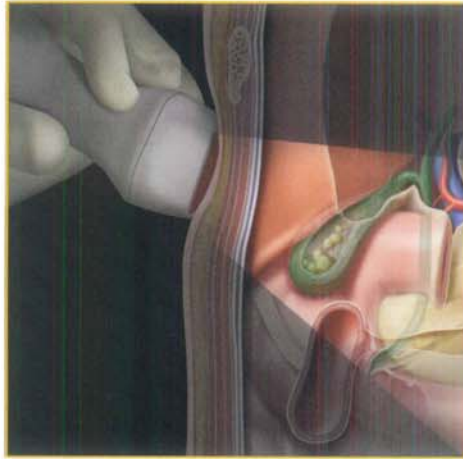
Abdominal Vessels

Aortic/Iliac Aneurysm <i>Simon S.M. Ho, MBBS, FRCR</i>	14-18
Aorto-Iliac Occlusive Disease <i>Simon S.M. Ho, MBBS, FRCR</i>	14-24
IVC Obstruction <i>Simon S.M. Ho, MBBS, FRCR</i>	14-30

Extremities

Deep Vein Thrombosis <i>Simon S.M. Ho, MBBS, FRCR</i>	14-36
Extremity Arterial Occlusive Disease <i>Simon S.M. Ho, MBBS, FRCR</i>	14-42
Peripheral Arterial Pseudoaneurysm <i>Simon S.M. Ho, MBBS, FRCR</i>	14-48
Peripheral Arteriovenous Fistula <i>Simon S.M. Ho, MBBS, FRCR</i>	14-54
Varicose Veins/Incompetent Perforator <i>Simon S.M. Ho, MBBS, FRCR</i>	14-58

DIAGNOSTIC IMAGING ULTRASOUND





SECTION 1: Liver

Introduction and Overview

Hepatic Sonography	1-2
--------------------	-----

Diffuse Parenchymal Disease

Acute Hepatitis	1-6
Cirrhosis, Hepatic	1-10
Schistosomiasis, Hepatic	1-16
Steatosis, Hepatic	1-20
Parenchymal Calcification, Hepatic	1-24
Diffuse Microabscesses, Hepatic	1-26
Lymphoma, Hepatic	1-28

Cyst and Cyst-Like Lesions

Hepatic Cyst	1-32
Caroli Disease	1-36
Biloma	1-40
Pyogenic Hepatic Abscess	1-42
Pyogenic Peri-Hepatic Abscess	1-48
Amebic Hepatic Abscess	1-50
Hepatic Echinococcus Cyst	1-54
Hepatic Trauma	1-60

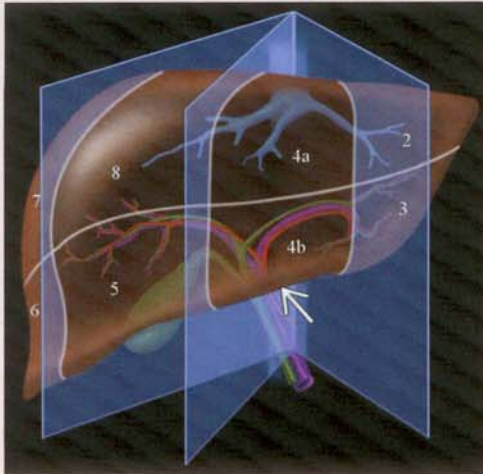
Focal Solid Masses

Hepatic Adenoma	1-64
Focal Nodular Hyperplasia	1-68
Hepatocellular Carcinoma	1-72
Hepatic Cavernous Hemangioma	1-78
Hepatic Metastases	1-82

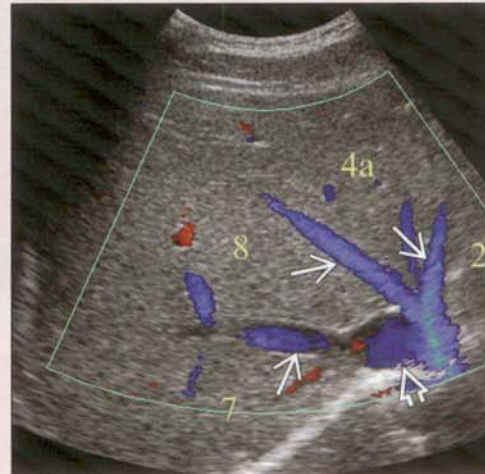
Vascular Conditions

Portal Hypertension	1-88
Porto-Systemic Collaterals	1-92
TIPS Shunts	1-96
Portal Vein Occlusion	1-100
Budd-Chiari Syndrome	1-104
Portal Vein Gas	1-108
Post-Transplant Liver	1-110

HEPATIC SONOGRAPHY



Graphic shows hepatic segments defined by vascular anatomy: 3 vertical planes along the hepatic veins & an oblique plane along the main portal branches. Segment 1 is between portal vein & IVC.



Transverse color Doppler ultrasound shows three hepatic veins draining into the IVC. Vertical planes defined by 3 hepatic veins divide the liver into 4 segments.

IMAGING ANATOMY

Anatomic Relationships

- Liver lies in right hypochondrium (mostly protected by rib cage), epigastrium and left hypochondrium
- Superior: Both hemidiaphragm and the undersurface of heart
- Inferiorly: Gallbladder, porta hepatis, hepatic flexure, second part of duodenum
- Left: Esophagus and stomach

Histology

- Hepatic lobules (around 1 cm) form the liver parenchyma
- In each lobule there is a central hepatic vein from which branching plates of hepatocytes extend towards the periphery
- Plates of hepatocytes are separated by hepatic sinusoids through which portal venous blood flows towards central hepatic vein
- Hepatocytes extract metabolites from the portal venous blood, acting as a filter for nutrients, toxins
- Hepatocytes secrete bile into canaliculi which run within the plates of hepatocytes and drain in an opposite direction to portal venous blood and form hepatic ductules and eventually bile ducts

Vasculature

- Liver receives a dual blood supply from the portal vein and hepatic artery (which explains rarity of infarction)
- Intra-hepatic branches of the portal vein, hepatic artery and bile duct run together throughout the liver (portal triad)
- Portal vein
 - Receives venous blood from subdiaphragmatic part of esophagus, stomach, small and large bowel, gallbladder, pancreas and spleen
 - Forms by convergence of splenic and superior mesenteric veins behind the neck of the pancreas
 - Runs within the hepatoduodenal ligament posterior to the hepatic artery and common bile duct
 - Approximately 8 cm long

- Divides at the porta hepatis into the left and right main portal veins
- Right main portal vein gives cystic vein to gallbladder before entering right lobe of liver and dividing
- Left main portal vein is joined by the ligamentum teres (obliterated left umbilical vein) and ligamentum venosum (obliterated ductus venosus) as it enters the left lobe
- Hepatic artery
 - Originates from celiac trunk (from aorta) as the common hepatic artery
 - Runs anterior to the portal vein and to the left of common bile duct in hepatoduodenal ligament
 - Divides at porta hepatis into left and right hepatic arteries, ramifies and accompanies portal veins and bile ducts
- Hepatic veins
 - Within liver, these run separate from portal triad
 - Sinusoids of hepatic lobules drain into intra- and sub-lobular veins then into hepatic veins
 - Typically three upper hepatic veins drain into the IVC: Right, middle (from caudate lobe) & left
 - Smaller, less consistent veins from the caudate lobe drain directly into a lower portion of IVC

Parenchymal Segmentation

- Couinaud's classification is the most commonly used
- Segment 1 (caudate lobe) lies between portal vein & inferior vena cava (IVC)
 - Unique in that it is supplied by the right and/or left portal vein(s), and drains directly into IVC
- Other segments are produced by four dividing planes
 - Vertically divided by the three planes along the three hepatic veins
 - Horizontally divided by the plane through the left and right main portal veins
 - 2: Left lateral superior segment
 - 3: Left lateral inferior segment
 - 4a: Left medial superior segment
 - 4b: Left medial inferior segment
 - 5: Right anterior inferior segment
 - 6: Right posterior inferior segment

Key Facts

- Unparalleled spatial resolution: Sonographic resolution of near- & mid-field hepatic lesions is unmatched by other imaging modalities
- Real-time imaging: Allows accurate guided biopsy/treatment of hepatic lesion(s)
- Limitations: Poor resolution of deep structures (penetration limited by acoustic attenuation) & inability to produce extended field-of-view image (due to overlying ribs & shape of liver)
 - Thus multiple views required for complete evaluation
- Key structures to identify
 - Hepatic parenchyma: Echotexture, distribution of vessels, surface contour
 - Portal and hepatic vessels (use Doppler study demonstrate patency and flow)
 - Porta hepatis: Vessels, biliary ducts & lymph nodes
 - Gallbladder fossa: Gallbladder
 - Perihepatic: Fluid or mass
- Lesion localization: Record using hepatic segment classification (& record adjacent vessels) for follow-up examinations
 - Caudate lobe (segment 1)
 - Left lateral (2 superior & 3 inferior) segments
 - Left medial (4a superior & 4b inferior) segments
 - Right inferior (5 anterior & 6 posterior) segments
 - Right superior (7 posterior & 8 anterior) segments
- Vascularity: Use color &/or power Doppler to demonstrate lesion vascularity (may help shorten list of differential diagnosis)
 - Use spectral Doppler to interrogate for flow direction and velocity of blood within vessels

- 7: Right posterior superior segment
- 8: Right anterior superior segment

Ultrasound Appearance

- Normal liver parenchyma appears homogeneous and composed of fine echoes
 - As internal references for echogenicity
 - Liver is slightly more hyperechoic than normal renal cortex
 - Liver is more hypoechoic than spleen
- Wall of hepatic vein is not resolved with ultrasound, compared with wall of portal vein which is echogenic

ANATOMY-BASED IMAGING ISSUES

Key Concepts or Questions

- Liver is a large organ and there are many potential "blind spots" obscured by overlying anatomical structures, most of these can be overcome with different patient positions and interrogation planes
- Lower edge of the normal liver lies just below the subcostal margin, providing an acoustic window for interrogation of the liver
 - This acoustic window may be lost when obscured by bowel (with gas) and/or ribs; usually occurs if lower edge of liver is displaced superiorly (due to cirrhosis or a mass pushing up liver)

Imaging Approaches

- Supine, subcostal/subxiphoid
 - Good for left lobe and anterior segments of right lobe
- Right anterior oblique, subcostal
 - Good for posterior segments of right lobe and for looking behind calcified lesions
 - Good for subdiaphragmatic areas and porta hepatis (which may be obscured by anterior ribs or bowel gas in the supine position)
- Right lateral oblique, lower intercostal
 - Good for high-riding or small cirrhotic liver
 - Additional view of porta hepatis if anteriorly obscured by gas

Imaging Protocols

- Reduce bowel (gas) distention and increase gallbladder filling with a 4-6 hour fast prior to ultrasound
- All segments of the liver should be interrogated for a complete examination
- Interrogation with suspension of respiration in inspiration helps lower the liver
- Color Doppler interrogation of the main hepatic vein and main branches of the left and right portal vein
- Direction of flow of the main portal vein should be stated for patients with cirrhosis/portal hypertension
 - Normal portal venous flow is hepatopetal (from other organs towards the liver)
- Color Doppler and power Doppler interrogation of lesions

Imaging Pitfalls

- Missing liver
 - Situs inversus or hepatic hernia through diaphragm

Normal Measurements

- In midclavicular sagittal plane: Liver length should be less than 15 cm
 - Riedel lobe is a thin inferior extension from lateral aspect of right hepatic lobe

PATHOLOGY-BASED IMAGING ISSUES

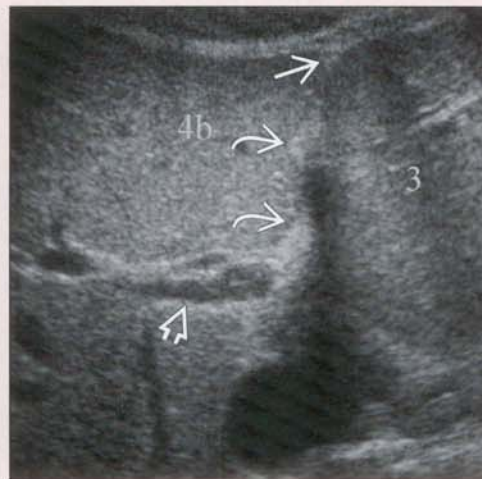
Imaging Approaches

- Ultrasound is an inexpensive, fast, radiation-free and mobile examination
 - It is sensitive for the detection of most hepatic lesions
 - Spatial resolution of near- and mid-field hepatic lesions is unmatched by other imaging modalities
- Hepatic ultrasound is limited by poor resolution of deep structures (penetration limited by acoustic attenuation) and inability to produce an extended field-of-view image

HEPATIC SONOGRAPHY



Oblique color Doppler ultrasound shows the portal vein bifurcation ➤. This plane divides superior & inferior hepatic segments. Note caudate lobe ➤ & fissure for ligamentum venosum ➤.



Transverse transabdominal ultrasound shows the ligamentum teres ➤ and related fissure ➤ which separates segment 4b from 3. Note fissure extends to left portal vein ➤.

- This makes relating a hepatic lesion to the surrounding anatomy difficult
- CT and MR may be more useful when such problems with ultrasound arise
- For imaging work-up of suspicious hepatic lesions
 - Ultrasound is good at locating the lesion and for monitoring progress
 - Real-time imaging capability of ultrasound allows accurate guided biopsy of lesion
 - Complimentary information of the lesion from CT and/or MR helps to reduce the need for biopsy
 - Intravenous ultrasound contrast agents are more sensitive in picking up subtle lesions and also demonstrate dynamic enhancing characteristics similar to CECT

Imaging Protocols

- Lesions detected by ultrasound should be further supplemented with color &/or power Doppler

Imaging Appearances of Focal Abnormalities

- Simple fluid: Through transmission (hypo-/anechoic); posterior acoustic enhancement
- Fluid with debris: Homogeneous low level echogenic content, fluid debris level when contents settle
 - Septae may be present
- Gas: Echogenic (in non-dependent position of cavity) and posterior ring down artifact
- Calcification: Echogenic and posterior acoustic shadowing

Differential Diagnosis for Focal Lesions

- Hyperechoic lesion
 - Fat-containing lesion
 - Hemangioma, adenoma, focal nodular hyperplasia
 - Hepatocellular carcinoma (HCC)
 - Hyperemic/hypervascular metastasis (gastrointestinal, ovarian, pancreatic, melanoma)
 - Calcification: Infection/infestation, neoplastic, vascular
 - Hematoma
- Hypoechoic lesion

- Cystic: Simple cyst, cystic neoplasm/metastases (ovarian, stomach, pancreas, colon)
- Fluid containing: Hydatid cyst, hematoma, abscess or necrotic neoplasm
- Lesion with internal septae
 - Cystic metastasis
 - Complicated simple cyst: Infection or hemorrhage into simple cyst
 - Infective collection: Pyogenic abscess, amebic abscess, hydatid cyst
 - Cavernous hemangioma, biliary cystadenoma, hepatic hamartoma
- Venous invasion
 - HCC
- Infiltrative lesion
 - HCC, lymphoma

EMBRYOLOGY

Embryologic Events

- Perinatal circulatory changes
 - In utero, blood returns from placenta via umbilical vein & ductus venosus to IVC
 - Umbilical vein is obliterated & forms ligamentum teres (free-edge of falciform ligament) after birth
 - Ductus venosus is obliterated & forms ligamentum venosum after birth

Practical Implications

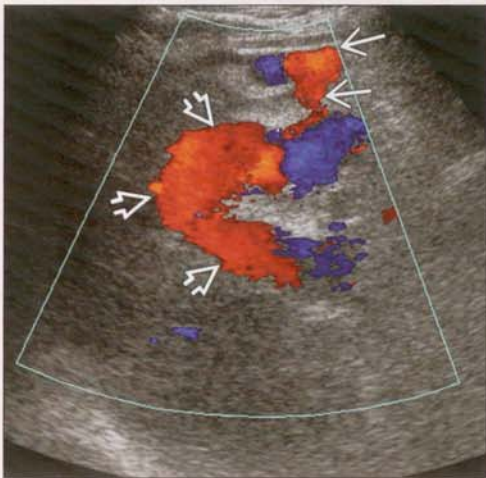
- Circulatory/ligamentous structures
 - Portal hypertension may result in re-canalization of previously obliterated vessels as collaterals
 - Abscess and biloma may insinuate along these tissue/ligamentous planes

RELATED REFERENCES

1. Gray's Anatomy: The Anatomical Basis of Clinical Practice. Editor-in-Chief Susan Standring. 39th Ed. Elsevier, 2005

HEPATIC SONOGRAPHY

IMAGE GALLERY



(Left) Transverse color Doppler ultrasound shows a recanalized umbilical vein \rightarrow (from ligamentum teres), channeling blood from the portal \rightarrow to the systemic circulation. **(Right)** Transverse transabdominal ultrasound shows the gallbladder \rightarrow & IVC \rightarrow . A line joining these two structures represents the division between the left (segment 4b) and right (segment 5) lobes of liver.

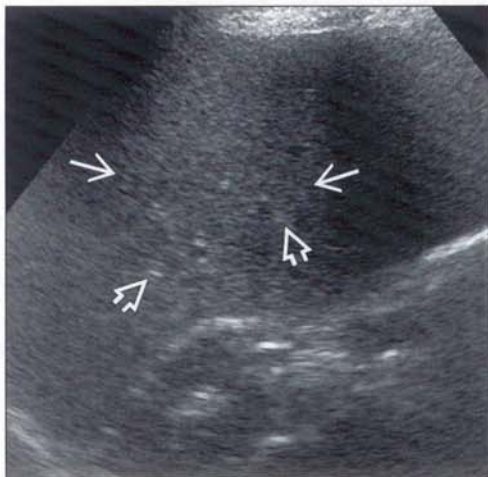


(Left) Transverse transabdominal ultrasound shows the portal vein \rightarrow , common bile duct \rightarrow , and hepatic artery \rightarrow in the hepatoduodenal ligament. **(Right)** Oblique transabdominal ultrasound in the right anterior oblique position is good for interrogating right upper segments \rightarrow . Subcostal regions may still be obscured \rightarrow .



(Left) Oblique transabdominal ultrasound using an intercostal approach shows the upper part \rightarrow of the liver better, especially in high-riding or cirrhotic livers. Superficial regions are also better seen \rightarrow . **(Right)** Longitudinal transabdominal ultrasound shows the right kidney \rightarrow is normally slightly hypoechoic compared to the liver \rightarrow . The kidney is used as internal standard for echogenicity.

ACUTE HEPATITIS



Oblique transabdominal ultrasound shows diffuse hypoechoic liver parenchyma in acute viral hepatitis. Against this, portal triad walls stand out as echogenic foci ("starry-sky").



Oblique transabdominal ultrasound shows decreased echogenicity of liver parenchyma in acute hepatitis, which becomes similar to that of kidney and spleen.

TERMINOLOGY

Definitions

- Nonspecific inflammatory response of liver to various agents

IMAGING FINDINGS

General Features

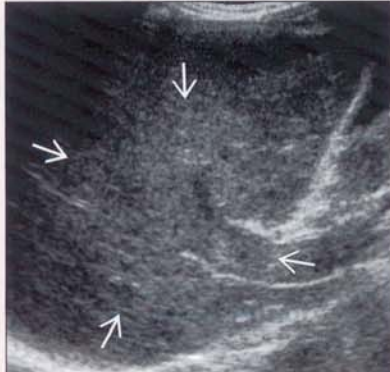
- Best diagnostic clue
 - Acute viral hepatitis on US
 - "Starry-sky" appearance: ↑ Echogenicity of portal triads against hypoechoic liver
 - Hepatomegaly and periportal lucency (edema)
- Location: Diffusely; involving both lobes
- Size
 - Acute: Enlarged liver
 - Chronic: Decrease in size of liver
- Other general features
 - Leading cause of hepatitis is viral infection
 - In medical practice, hepatitis refers to viral infection
 - Viral hepatitis

- Infection of liver by small group of hepatotropic viruses
- Stages: Acute, chronic active hepatitis (CAH) and chronic persistent hepatitis
- Responsible for 60% of cases of fulminant hepatic failure in US
- Alcoholic hepatitis: Acute and chronic
- Nonalcoholic steatohepatitis (NASH)
 - Significant cause of acute and progressive liver disease
 - May be an underlying cause of cryptogenic cirrhosis
- Imaging of viral/alcoholic hepatitis done to exclude
 - Obstructive biliary disease/neoplasm
 - To evaluate parenchymal damage noninvasively

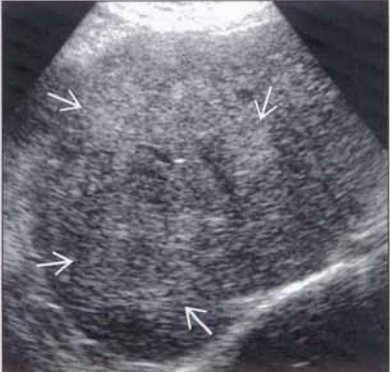
Ultrasonographic Findings

- Grayscale Ultrasound
 - Acute viral hepatitis
 - Hepatomegaly with diffuse decrease in echogenicity


DDx: Acute Hepatitis



Infiltrative HCC



Diffuse Metastases



Fatty Liver

Key Facts

Imaging Findings

- **Acute viral hepatitis**
- Hepatomegaly with diffuse decrease in echogenicity
- Splenomegaly and hepatic echogenicity diffusely becoming similar to spleen and renal cortex (normal liver is more echogenic than spleen and renal cortex)
- "Starry-sky" appearance: Increased echogenicity of portal triad walls against hypoechoic liver
- Periportal hypo-/anechoic area (hydropic swelling of hepatocytes)
- Thickening of GB wall; hypertonic GB, nontender
- Increase in echogenicity of fat in ligamentum venosum, falciform ligament, periportal tissues
- **Chronic active viral hepatitis**
- Increased echogenicity of liver

- "Silhouetting" of portal vein walls (loss of definition of portal veins)
- Heterogeneous parenchymal echotexture due to regenerating nodules
- Adenopathy in hepatoduodenal ligament
- Best imaging tool: Ultrasound to rule out biliary obstruction or other hepatic pathology

Top Differential Diagnoses

- Infiltrative Hepatocellular Carcinoma (HCC)
- Diffuse Metastases or Lymphoma
- Steatosis (Fatty Liver)

Diagnostic Checklist

- Ruling out other causes of "diffuse hepatomegaly"
- Two most consistent findings in acute hepatitis: Hepatomegaly and periportal edema

- Splenomegaly and hepatic echogenicity diffusely becoming similar to spleen and renal cortex (normal liver is more echogenic than spleen and renal cortex)
- "Starry-sky" appearance: Increased echogenicity of portal triad walls against hypoechoic liver
- Periportal hypo-/anechoic area (hydropic swelling of hepatocytes)
- Thickening of GB wall; hypertonic GB, nontender
- Increase in echogenicity of fat in ligamentum venosum, falciform ligament, periportal tissues
- **Chronic active viral hepatitis**
 - Increased echogenicity of liver
 - "Silhouetting" of portal vein walls (loss of definition of portal veins)
 - Heterogeneous parenchymal echotexture due to regenerating nodules
 - Adenopathy in hepatoduodenal ligament
- **Acute alcoholic hepatitis**
 - Hepatomegaly with diffuse increase in echogenicity
- **Late stage of alcoholic hepatitis**
 - Atrophic liver with micronodular cirrhosis

- Nonalcoholic steatohepatitis (NASH)
 - Indistinguishable from alcoholic hepatitis
- CECT
 - Acute and chronic viral hepatitis
 - ± Heterogeneous parenchymal enhancement
 - Chronic hepatitis: Regenerating nodules may be isodense with liver

MR Findings

- Viral hepatitis
 - Increase in T1 and T2 relaxation times of liver
 - T2WI: High signal intensity bands paralleling portal vessels (periportal edema)
- Alcoholic steatohepatitis (diffuse fatty infiltration)
 - T1WI in-phase GRE image: Increased signal intensity of liver than spleen or muscle
 - T1WI out-of-phase GRE image: Decreased signal intensity of liver (due to lipid in liver)

Imaging Recommendations

- Best imaging tool: Ultrasound to rule out biliary obstruction or other hepatic pathology

CT Findings

- NECT
 - Acute viral hepatitis
 - Hepatomegaly, gallbladder wall thickening
 - Periportal hypodensity (fluid/lymphedema)
 - Chronic active viral hepatitis
 - Lymphadenopathy in porta hepatis/gastrohepatic ligament and retroperitoneum (in 65% of cases)
 - Hyperdense regenerating nodules
 - Acute alcoholic hepatitis
 - Hepatomegaly
 - Diffuse hypodense liver (due to fatty infiltration)
 - Fatty infiltration may be focal/lobar/segmental
 - Chronic alcoholic hepatitis
 - Mixture of steatosis and early cirrhotic changes
 - Steatosis: Liver-spleen attenuation difference will be less than 10 HU
 - Normal liver has slightly ↑ attenuation than spleen

DIFFERENTIAL DIAGNOSIS

Infiltrative Hepatocellular Carcinoma (HCC)

- Background cirrhosis
- Invasion of portal vein

Diffuse Metastases or Lymphoma

- Hepatomegaly due to diffuse infiltration
- Background vascular architecture may/may not be distorted
- Lymphoma more common in immune-suppressed patients
 - Examples: AIDS and organ transplant recipients

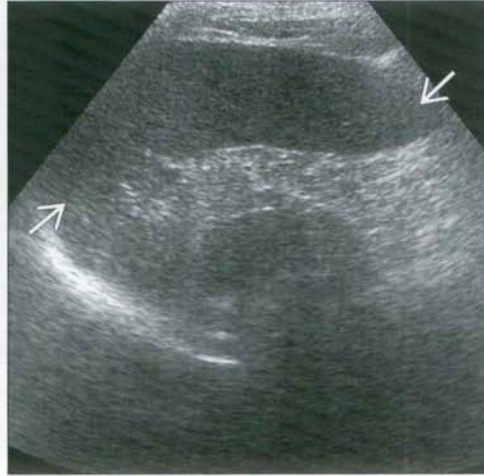
Steatosis (Fatty Liver)

- Hepatomegaly
- Diffuse, patchy or focal increase in echogenicity
- Normal vessels course through "lesion"

ACUTE HEPATITIS

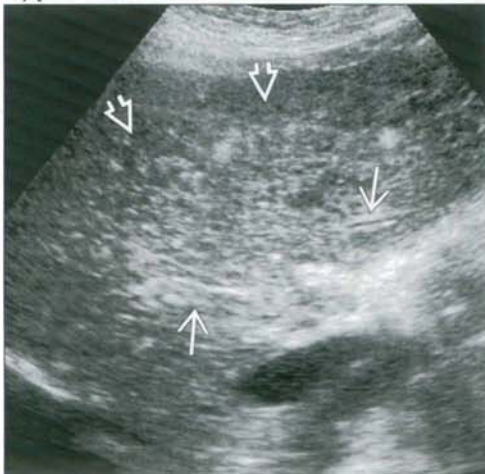
IMAGE GALLERY

Typical



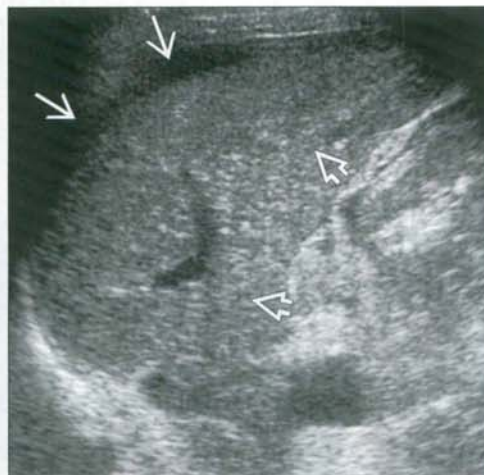
(Left) Oblique transabdominal ultrasound shows a markedly thickened gallbladder wall \Rightarrow in acute hepatitis. There is near obliteration of the lumen. Note small amount of ascitic fluid \Rightarrow . *(Right)* Oblique transabdominal ultrasound shows splenomegaly \Rightarrow in acute viral hepatitis. There is no splenic vein distension or evidence of collaterals.

Typical



(Left) Oblique transabdominal ultrasound shows heterogeneous echogenicity \Rightarrow of the liver in chronic active viral hepatitis. Portal vein walls \Rightarrow are difficult to define. *(Right)* Transverse transabdominal ultrasound shows lymphadenopathy \Rightarrow adjacent to the portal vein \Rightarrow in a patient with viral hepatitis.

Typical

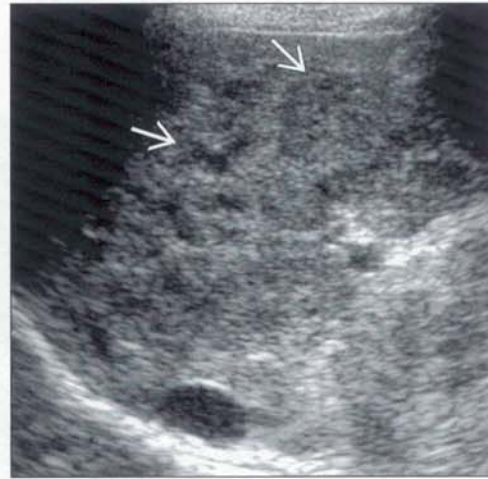


(Left) Transverse transabdominal ultrasound shows the rounded contour \Rightarrow of hepatomegaly and diffuse increase in echogenicity \Rightarrow in acute alcoholic hepatitis. *(Right)* Oblique transabdominal ultrasound shows cirrhosis in a patient with chronic viral hepatitis. Note atrophic liver bordered by ascites \Rightarrow . Note heterogeneous hepatic echo pattern \Rightarrow .

CIRRHOSIS, HEPATIC



Longitudinal transabdominal ultrasound shows a small right hepatic lobe with coarsened echotexture and increased echogenicity. The liver is surrounded by ascites.



Oblique transabdominal ultrasound shows macronodular cirrhosis with multiple solid heterogeneous nodules.

TERMINOLOGY

Definitions

- Chronic liver disease characterized by diffuse parenchymal necrosis with extensive fibrosis and regenerative nodule formation

IMAGING FINDINGS

General Features

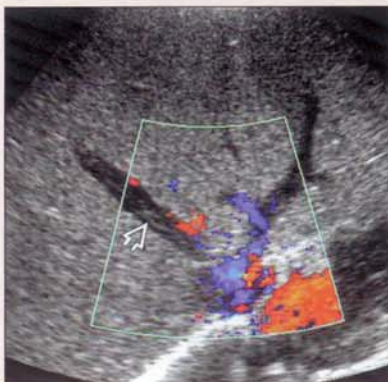
- Best diagnostic clue: Nodular contour, coarse echotexture +/- hypoechoic nodules
- Location: Diffuse liver involving both lobes
- Size: General atrophy with relative enlargement of the caudate/left lobes
- Key concepts
 - Common end response of liver to a variety of insults and injuries
 - Classification of cirrhosis based on morphology, histopathology and etiology
 - Classification
 - Micronodular (Laennec) cirrhosis (< 1 cm diameter): Alcoholism (60-70% cases in US)

- Macronodular (postnecrotic) cirrhosis: Viral hepatitis (10% in US; majority of cases worldwide)
- Mixed cirrhosis
 - Alcohol abuse is most common cause in West; hepatitis B in Asia
 - One of 10 leading causes of death in Western world (6th in US)

Ultrasonographic Findings

- Grayscale Ultrasound
 - Nodular liver surface contour
 - Hepatomegaly (early stage)/normal size/shrunken
 - Enlarged caudate lobe & lateral segment of left lobe
 - Atrophy of right lobe & medial segment of left lobe
 - Increased echogenicity of fissures & portal structures
 - Coarsened echotexture, increase parenchymal echogenicity
 - Associated signs of fatty infiltration
 - Regenerating nodules (siderotic)
 - Iso-/hypoechoic nodules (regenerating nodules)
 - Hyperechoic rim (surrounding fibrosis)
 - Dysplastic nodules (> 1 cm)
 - Considered to be pre-malignant

DDx: Cirrhosis



Budd Chiari



Infiltrative HCC



Diffuse Mets

CIRRHOSIS, HEPATIC

Key Facts

Imaging Findings

- Best diagnostic clue: Nodular contour, coarse echotexture +/- hypoechoic nodules
- Hepatomegaly (early stage)/normal size/shrunken
- Enlarged caudate lobe & lateral segment of left lobe
- Atrophy of right lobe & medial segment of left lobe
- Increased echogenicity of fissures & portal structures
- Regenerating nodules (siderotic)
- Signs of portal hypertension (PHT)
- Signs of hypo-albuminemia

Top Differential Diagnoses

- Budd-Chiari Syndrome
- Hepatocellular Carcinoma
- Treated Metastatic Disease

Pathology

- Micronodular (Laennec) cirrhosis: Alcohol
- Macronodular (postnecrotic) cirrhosis: Viral
- Steatosis → hepatitis → cirrhosis
- Alcohol (60-70%), chronic viral hepatitis B/C (10%)
- 3rd leading cause of death for men 34-54 years
- US: Hepatitis C (cirrhosis) causes 30-50% of HCC
- Japan: Hepatitis C (cirrhosis) 70% of HCC cases

Clinical Issues

- Fatigue, jaundice, ascites, encephalopathy
- Gynecomastia and testicular atrophy in males
- Virilization in females

Diagnostic Checklist

- Rule out other causes of "nodular dysmorphic liver"

- Difficult to differentiate from small hepatocellular carcinoma (HCC)
- Compression of hepatic veins
- Signs of portal hypertension (PHT)
 - Portal vein (> 13 mm), splenic (> 11 mm), superior mesenteric (> 12 mm), coronary (> 7 mm)
 - Dilated hepatic & splenic arteries with increased flow
 - Splenomegaly
 - Portal cavernoma (cavernous transformation of portal vein)
 - Portosystemic shunts: Lienorenal, gastrosplenic, paraumbilical
 - Ascites
- Signs of hypo-albuminemia
 - Ascites
 - Edematous gallbladder wall and bowel wall
- Color Doppler
 - Hepatic vein: Portalization of hepatic vein
 - Loss of normal triphasic/flattened hepatic vein
 - Turbulence if hepatic vein compressed
 - Portal vein: Increased pulsatility, decreased velocity
 - Hepatofugal (away from liver) flow: Not candidate for splenorenal shunt/needs portacaval or mesocaval shunt
 - Hepatic artery: Dilatation of hepatic arteries with increased arterial flow

CT Findings

- Nodular contour & widened fissures
- Atrophy of right lobe & medial segment of left lobe
- Enlarged caudate lobe & lateral segment of left lobe
- Regenerative nodules; fibrotic & fatty changes
- Portal hypertension: Varices, ascites, splenomegaly
- Siderotic regenerative nodules
 - NECT: Increased attenuation due to iron content
 - CECT: Nodules disappear after contrast
 - Nodules & parenchyma enhance to same level
- Dysplastic regenerative nodules
 - NECT: Large nodules: Hyperdense (↑ iron + ↑ glycogen)
 - Small nodules: Isodense with liver (undetected)
 - CECT: Iso-/hyperdense to normal liver

- Fibrotic and fatty changes
 - NECT
 - Fibrosis: Diffuse lacework, thick bands & mottled areas of decreased density
 - Fatty changes: Mottled areas of low attenuation
 - CECT
 - Fibrosis: Less evident due to enhancement to same degree of liver
 - Confluent fibrosis: May show delayed persistent enhancement
 - Fatty changes: Areas of low attenuation

MR Findings

- Siderotic regenerative nodules: Paramagnetic effect of iron within nodules
 - T1WI: Hypointense
 - T2WI: Increased conspicuity of low signal intensity
 - T2 gradient-echo or FLASH: Markedly hypointense
 - Gamna-Gandy bodies (siderotic nodules in spleen)
 - Caused by hemorrhage (portal hypertension) into splenic follicles
 - T1 and T2WI: Hypointense
 - T2 GRE and FLASH images: Markedly hypointense
- Dysplastic regenerative nodules
 - T1WI: Hyperintense compared to liver parenchyma
 - T2WI: Hypointense relative to liver parenchyma
- Fibrotic and fatty changes
 - T1WI: Fibrosis: Hypointense; fat: Hyperintense
 - T2WI: Fibrosis: Hyperintense; fat: Hypointense
- MR angiography
 - Varices: Tortuous structures of high signal intensity

Imaging Recommendations

- Best imaging tool: Grayscale and color ultrasound

DIFFERENTIAL DIAGNOSIS

Budd-Chiari Syndrome

- Liver damaged, but no bridging fibrosis
- Occluded or narrowed hepatic veins ± IVC
- Collateral vessels extending to capsule
- Ascites

CIRRHOSIS, HEPATIC

- Acute phase: Hepatomegaly, hemorrhagic infarct
- Chronic phase: Fibrosis (post-infarct), "large regenerative nodules", collaterals
- Caudate lobe sparing (enlargement)

Hepatocellular Carcinoma

- Hypoechoic lesion within cirrhotic liver
- Portal vein thrombosis/invasion

Treated Metastatic Disease

- Example: Breast cancer metastases to liver
 - May shrink and fibrose with treatment
 - Simulating nodular contour of cirrhotic liver

Hepatic Sarcoidosis

- Systemic noncaseating granulomatous disorder
- Hypoattenuating nodules (size: Up to 2 cm)
- Hypointense nodules on T1 and T2WI MR

PATHOLOGY

General Features

- General path comments
 - Micronodular (Laennec) cirrhosis: Alcohol
 - Macronodular (postnecrotic) cirrhosis: Viral
 - Catalase oxidation of ethanol → damage cellular membranes & proteins
 - Cellular antigens → inflammatory cells → immune mediated cell damage
 - Steatosis → hepatitis → cirrhosis
 - Regenerative (especially siderotic) nodules → dysplastic nodules → HCC
 - Dysplastic nodules considered premalignant
- Etiology
 - Alcohol (60-70%), chronic viral hepatitis B/C (10%)
 - Primary biliary cirrhosis (5%)
 - Hemochromatosis (5%)
 - Primary sclerosing cholangitis, drugs, cardiac causes
 - Malnutrition, hereditary (Wilson), cryptogenic
 - In children: Biliary atresia, hepatitis, α -1 antitrypsin deficiency
- Epidemiology
 - 3rd leading cause of death for men 34-54 years
 - Risk of HCC
 - US: Hepatitis C (cirrhosis) causes 30-50% of HCC
 - Japan: Hepatitis C (cirrhosis) 70% of HCC cases
 - 2.5x higher in cirrhotic hepatitis B positive
 - Alcohol & primary biliary cirrhosis: 2-5 fold ↑ risk
 - Mortality due to complication
 - Ascites (50%), variceal bleeding (25%), renal failure (10%), bacterial peritonitis (5%), complications of ascites therapy (10%)

Gross Pathologic & Surgical Features

- Alcoholic cirrhosis
 - Early stage: Large, yellow, fatty, micronodular liver
 - Late stage: Shrunken, brown-yellow, hard organ with macronodules
- Postnecrotic cirrhosis
 - Macronodular (> 3 mm - 1 cm); fibrous scars

Microscopic Features

- Portal-central, portal-portal fibrous bands

- Micro & macronodules; mononuclear cells
- Abnormal arteriovenous interconnections

CLINICAL ISSUES

Presentation

- Most common signs/symptoms
 - Alcoholic cirrhosis: May be clinically silent (10-40% found at autopsy)
 - Nodular liver, anorexia, malnutrition, weight loss
 - Portal hypertension: Splenomegaly, varices, caput medusae
 - Fatigue, jaundice, ascites, encephalopathy
 - Gynecomastia and testicular atrophy in males
 - Virilization in females
- Clinical Profile: Patient with history of alcoholism, nodular liver, jaundice, ascites & splenomegaly
- Lab data: Abnormal liver function tests; anemia
 - Alcoholic cirrhosis: Severe increase in AST (SGOT)
 - Viral: Severe increase in ALT (SGPT)

Demographics

- Age: Middle and elderly age group
- Gender: Males more than females

Natural History & Prognosis

- Complications
 - Ascites, variceal hemorrhage, renal failure, coma
 - HCC: Due to hepatitis B, C and alcoholism
- Prognosis
 - Alcoholic cirrhosis: 5 year survival in less than 50%
 - Advanced disease: Poor prognosis

Treatment

- Alcoholic cirrhosis
 - Abstinence; decrease protein diet; multivitamins
 - Prednisone; diuretics (for ascites)
- Management limited to treating complications & underlying cause
- Advanced stage: Liver transplantation

DIAGNOSTIC CHECKLIST

Consider

- Rule out other causes of "nodular dysmorphic liver"

Image Interpretation Pearls

- Nodular liver contour; lobar atrophy & hypertrophy
- Regenerative nodules, ascites, splenomegaly, varices

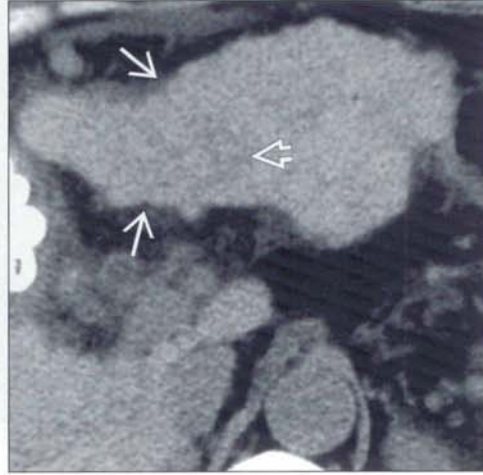
SELECTED REFERENCES

1. Nicolau C et al: Gray-scale ultrasound in hepatic cirrhosis and chronic hepatitis: diagnosis, screening, and intervention. *Semin Ultrasound CT MR.* 23(1):3-18, 2002
2. Tchelepi H et al: Sonography of diffuse liver disease. *J Ultrasound Med.* 21(9):1023-32; quiz 1033-4, 2002
3. Dodd GD et al: Spectrum of imaging findings of the liver in end-stage cirrhosis: Part I, gross morphology and diffuse abnormalities. *AJR.* 173:1031-6, 1999
4. Zwiebel WJ: Sonographic diagnosis of diffuse liver disease. *Semin Ultrasound CT MR.* 16(1):8-15, 1995

CIRRHOSIS, HEPATIC

IMAGE GALLERY

Typical



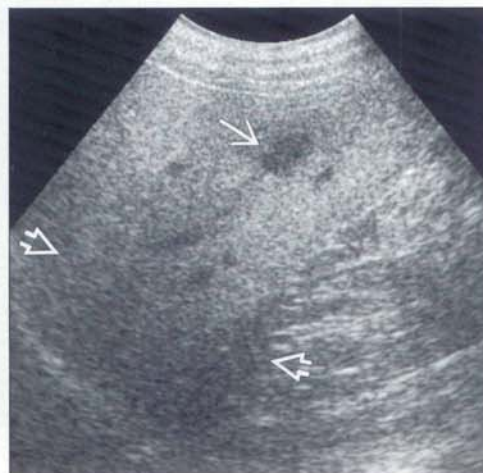
(Left) Longitudinal transabdominal ultrasound shows small right hepatic lobe with nodular surface ➡ highlighted by the surrounding ascites. Note coarsened echotexture of micronodular cirrhosis. **(Right)** Transverse NECT shows a magnified left hepatic lobe ➡ with irregular surface contour. The heterogeneity and nodularity of the cirrhotic liver parenchyma is subtle ➡

Typical



(Left) Transverse transabdominal ultrasound shows a well-defined hypoechoic nodule ➡ in a cirrhotic liver. This is the typical appearance of a regenerative nodule. **(Right)** Oblique transabdominal ultrasound shows a 2 cm hypoechoic nodule ➡ in a cirrhotic liver. Its large size was suspicious and subsequent biopsy showed it to be a dysplastic nodule.

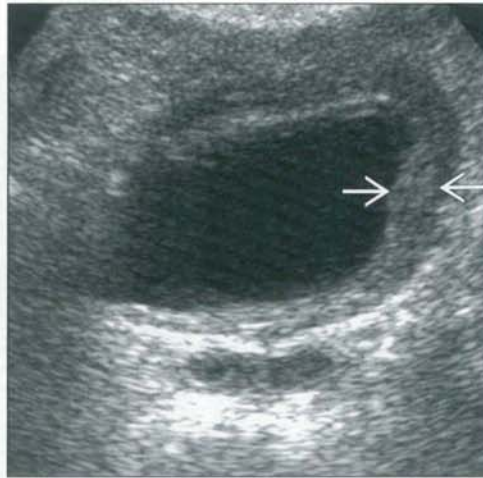
Typical



(Left) Longitudinal transabdominal ultrasound shows an enlarged caudate lobe ➡ compared to the atrophic medial segment of left lobe ➡. **(Right)** Oblique transabdominal ultrasound shows a hemangioma ➡ mimicking a regenerating nodule in a cirrhotic liver. Superimposed steatosis increased the echogenicity and caused acoustic attenuation ➡.

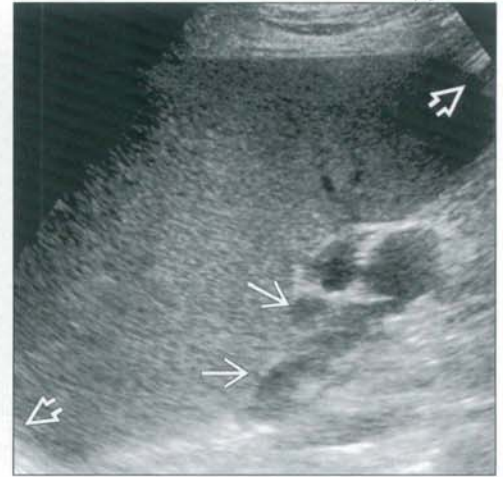
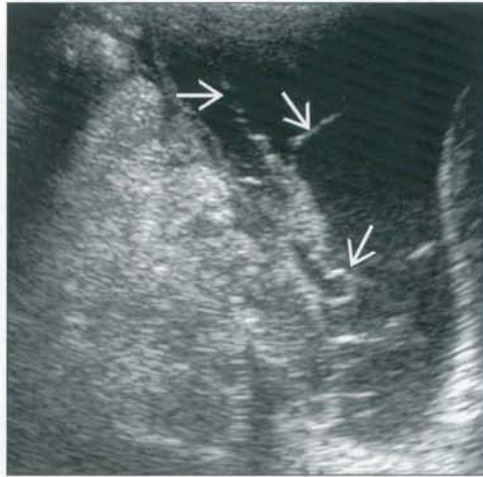
CIRRHOSIS, HEPATIC

(Left) Oblique transabdominal ultrasound shows diffuse gallbladder wall thickening \Rightarrow in a cirrhotic patient, related to hypo-albuminemia or poor venous drainage. **(Right)** Oblique transabdominal ultrasound shows loops of small bowel with thickened walls \Rightarrow , floating within ascitic fluid \Rightarrow . Mural edema may be due to portal hypertension or hypo-albuminemia.



Typical

(Left) Oblique transabdominal ultrasound shows chronic ascites in a cirrhotic patient. Note fibrin strands \Rightarrow running through the fluid. **(Right)** Oblique transabdominal ultrasound shows splenomegaly (16 cm between \Rightarrow) and splenic varices \Rightarrow due to portal hypertension.



Typical

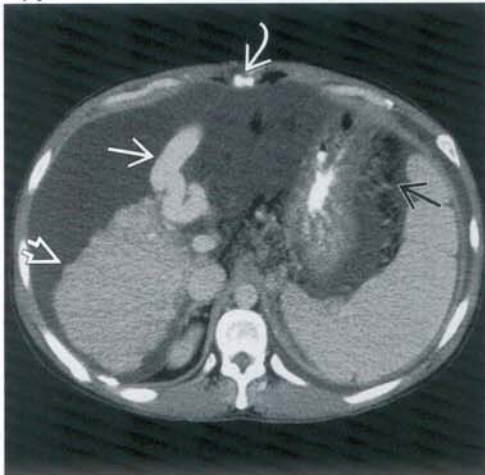
(Left) Oblique transabdominal ultrasound shows recanalization of the paraumbilical vein \Rightarrow , which acts as a portosystemic collateral to compensate for portal hypertension. **(Right)** Longitudinal color Doppler ultrasound shows flow in ectatic recanalized paraumbilical veins \Rightarrow as a result of portal hypertension.



Typical

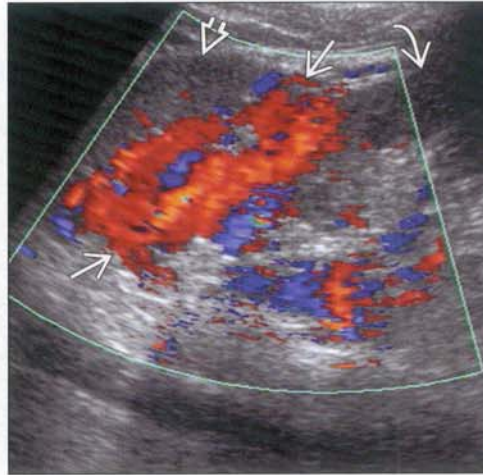
CIRRHOSIS, HEPATIC

Typical



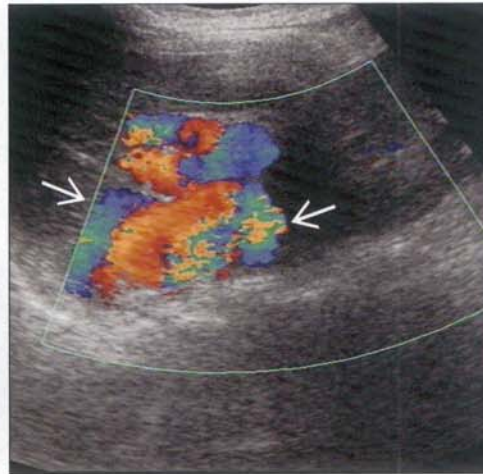
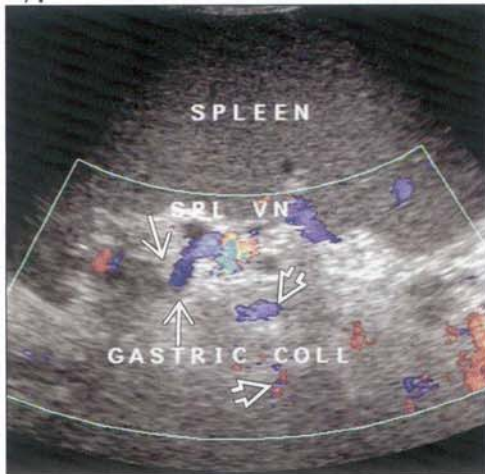
(Left) Transverse CECT shows ectatic recanalized paraumbilical vein & previous embolization . Cirrhotic liver has a nodular surface prominent short gastric veins & splenomegaly. **(Right)** Oblique color Doppler ultrasound shows portal cavernoma .

Typical



(Left) Oblique transabdominal ultrasound shows ectatic lienorenal collaterals between the spleen and superior pole of the left kidney . **(Right)** Oblique color Doppler ultrasound shows flow within the lienorenal collaterals (demonstrated in the previous image) between the spleen and left kidney .

Typical



(Left) Oblique color Doppler ultrasound shows splenomegaly with splenic varices and gastric collaterals . **(Right)** Oblique color Doppler ultrasound shows enlargement of short gastric veins and turbulent flow. These collaterals help decompress portal hypertension in a cirrhotic liver.