

Ultrasound model predicts liver disease progress

Amerigo Allegretto

Mar 20, 2024



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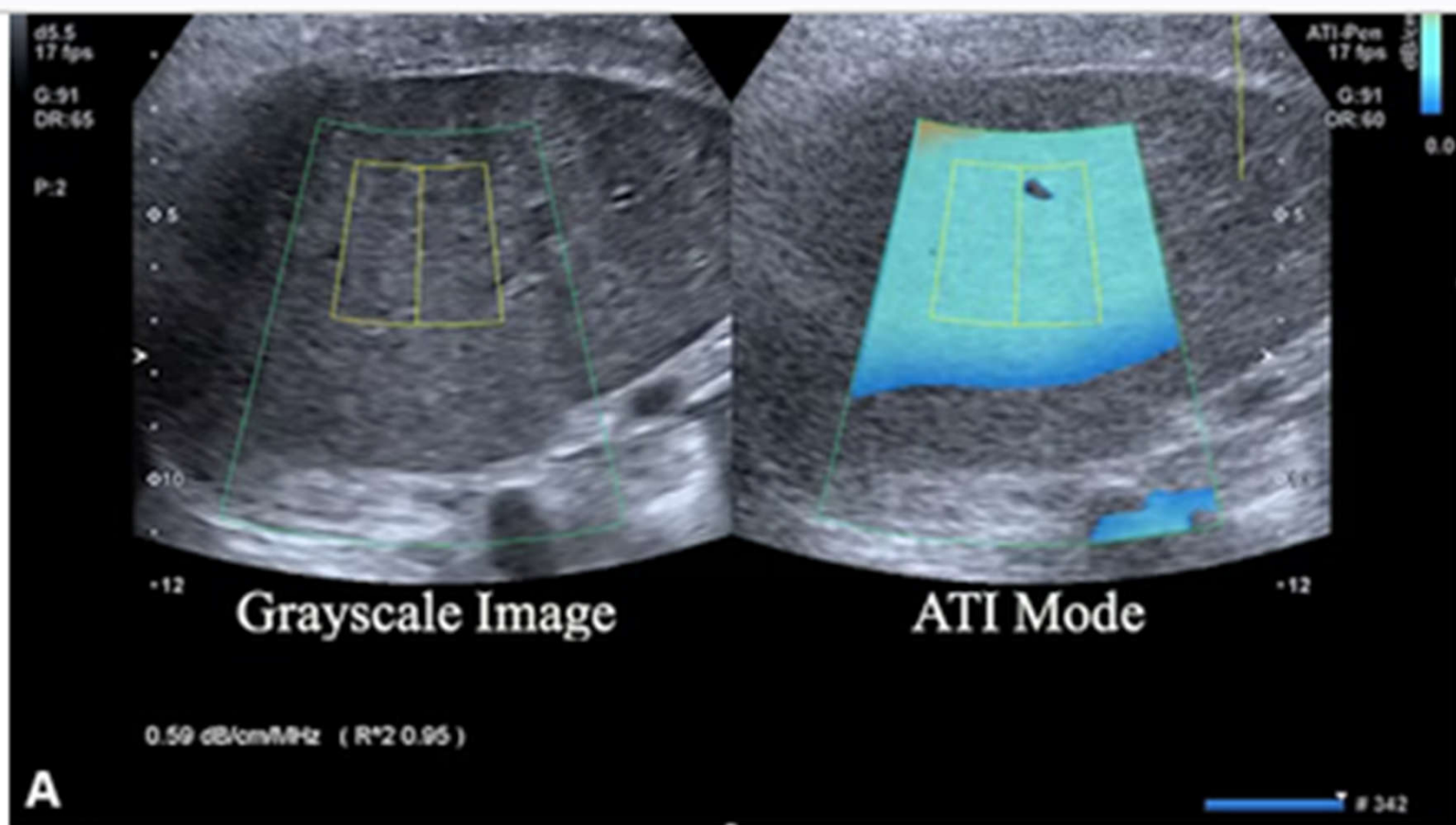
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Original Research

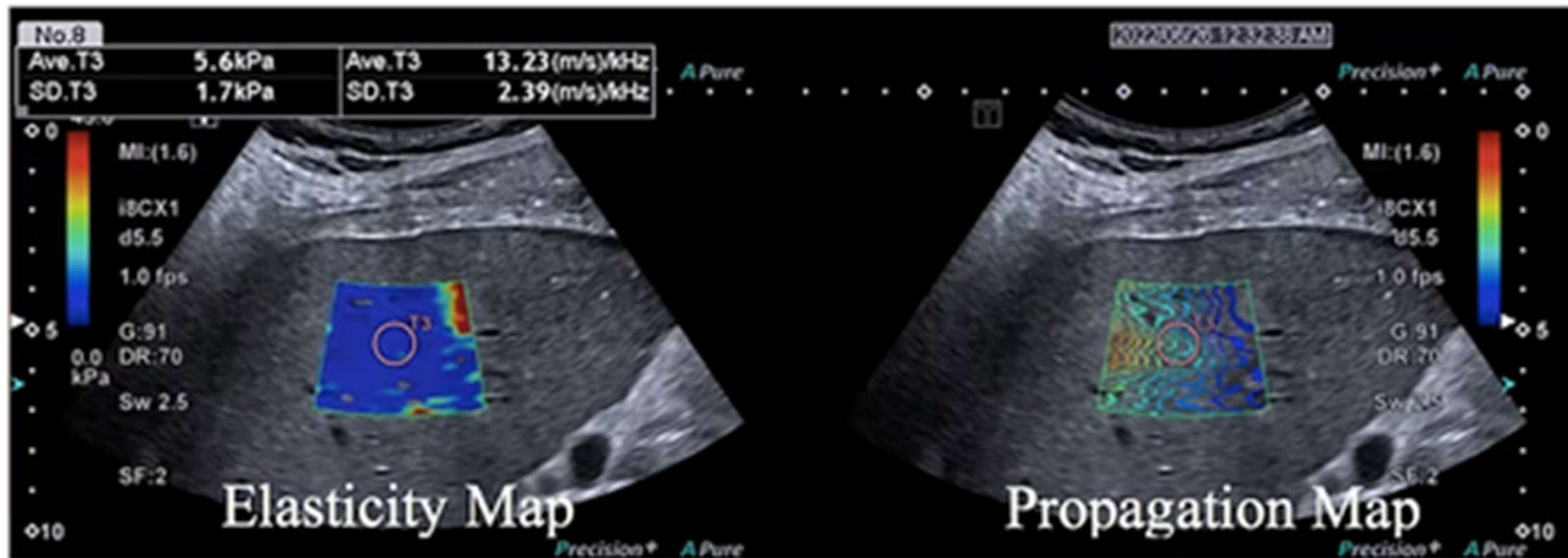


Gastrointestinal Imaging

Multiparametric US for Identifying Metabolic Dysfunction-associated Steatohepatitis: A Prospective Multicenter Study



A



Three parameters were found to have significant associations with MASH: attenuation coefficient (odds ratio [OR], 3.32 [with 1 as reference]; $p < 0.001$), alanine aminotransferase level (OR, 4.42; $p = 0.001$), and international normalized ratio (OR, 0.59; $p = 0.03$).

The researchers developed a combined model with these parameters, with the results being area under the curve (AUC) values of 0.85 and 0.77 for predicting MASH in the training ($n = 257$) and validation groups ($n = 167$), respectively.

The team also evaluated the model's performance on subgroups, including participants with and without diabetes and those with and without hepatitis B. It reported the following respective AUC

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REVIEWS

Endometriosis: pathogenesis and treatment



Endometriosis: pathogenesis and treatment

Paolo Vercellini, Paola Viganò, Edgardo Somigliana and Luigi Fedele

Abstract | Endometriosis is defined as the presence of endometrial-type mucosa outside the uterine cavity. Of the proposed pathogenic theories (retrograde menstruation, coelomic metaplasia and Müllerian remnants), none explain all the different types of endometriosis. According to the most convincing model, the retrograde menstruation hypothesis, endometrial fragments reaching the pelvis via transtubal retrograde flow, implant onto the peritoneum and abdominal organs, proliferate and cause chronic inflammation with formation of adhesions. The number and amount of menstrual flows together with genetic and environmental factors determines the degree of phenotypic expression of the disease. Endometriosis is estrogen-dependent, manifests during reproductive years and is associated with pain and infertility. Dysmenorrhoea, deep dyspareunia, dyschezia and dysuria are the most frequently reported symptoms. Standard diagnosis is carried out by direct visualization and histologic examination of lesions. Pain can be treated by excising peritoneal implants, deep nodules and ovarian cysts, or inducing lesion suppression by abolishing ovulation and menstruation through hormonal manipulation with progestins, oral contraceptives and gonadotropin-releasing hormone agonists. Medical therapy is symptomatic, not cytoreductive; surgery is associated with high recurrence rates. Although lesion eradication is considered a fertility-enhancing procedure, the benefit on reproductive performance is moderate. Assisted reproductive technologies constitute a valid alternative. Endometriosis is associated with a 50% increase in the risk of epithelial ovarian cancer, but preventive interventions are feasible.

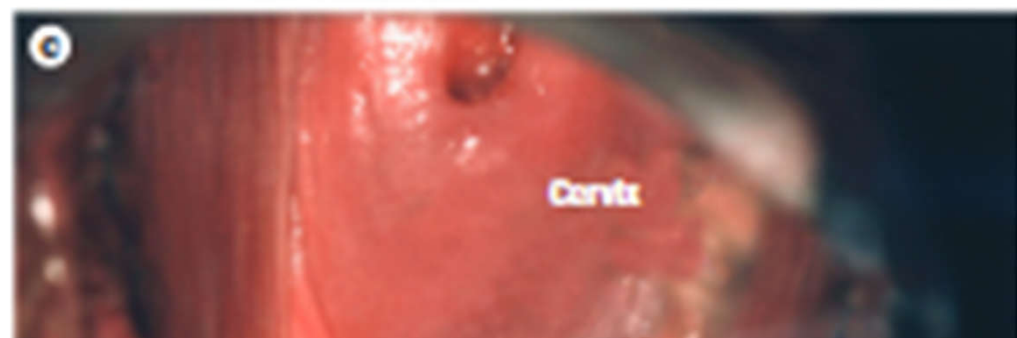
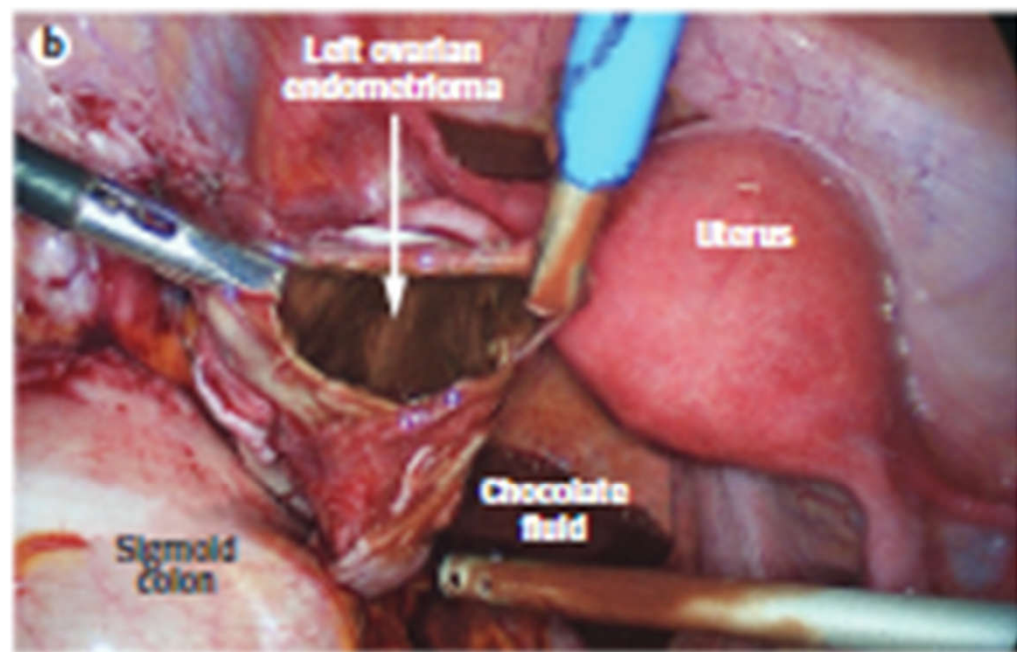
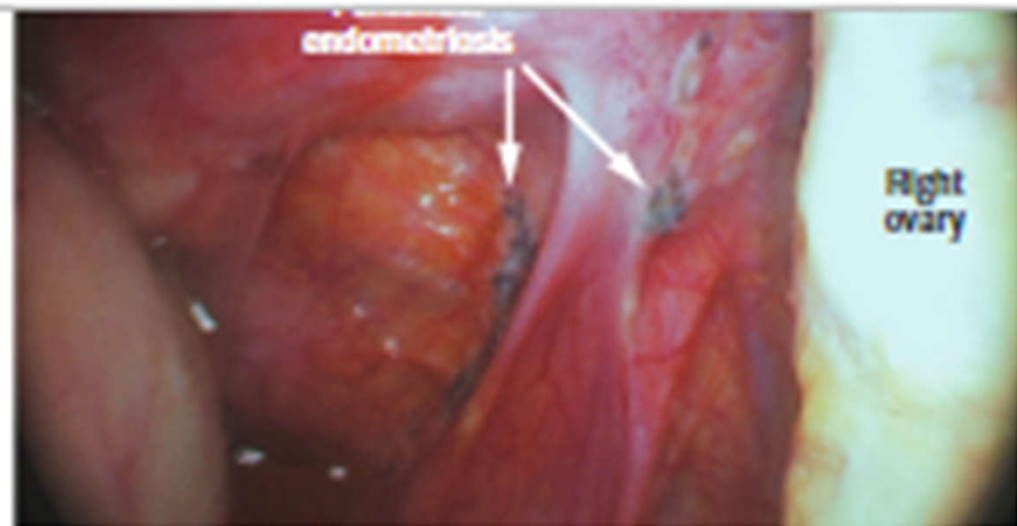
Vercellini, P. et al. *Nat. Rev. Endocrinol.* 10, 261–275 (2014); published online 24 December 2013; doi:10.1038/nrendo.2013.255

Introduction

Endometriosis: pathogenesis and treatment

Paolo Vercellini, Paola Viganò, Edgardo Somigliana and Luigi Fedele

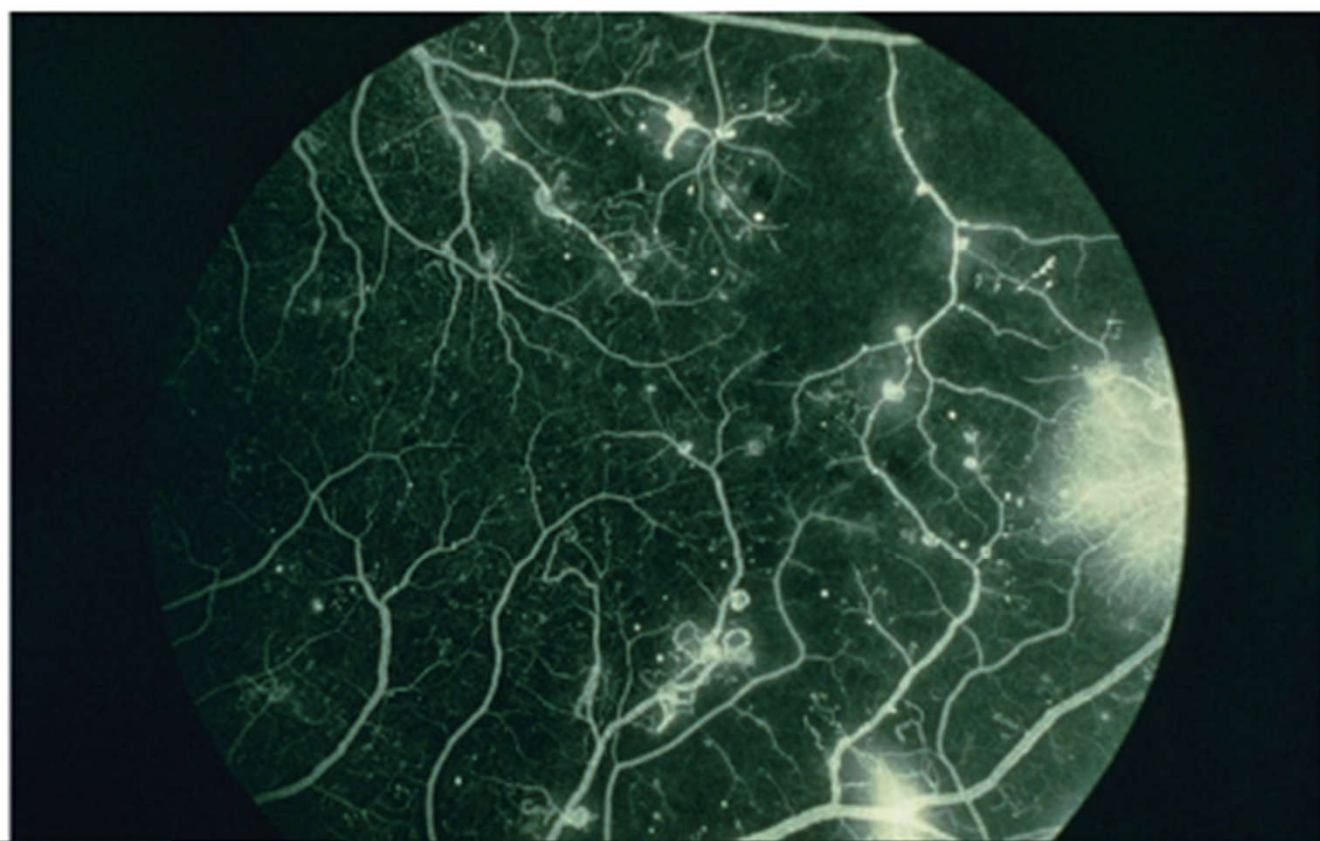
Abstract | Endometriosis is defined as the presence of endometrial-type mucosa outside the uterine cavity. Of the proposed pathogenic theories (retrograde menstruation, coelomic metaplasia and Müllerian remnants), none explain all the different types of endometriosis. According to the most convincing model, the retrograde menstruation hypothesis, endometrial fragments reaching the pelvis via transtubal retrograde flow, implant onto the peritoneum and abdominal organs, proliferate and cause chronic inflammation with formation of adhesions. The number and amount of menstrual flows together with genetic and environmental factors determines the degree of phenotypic expression of the disease. Endometriosis is estrogen-dependent, manifests during reproductive years and is associated with pain and infertility. Dysmenorrhoea, deep dyspareunia, dyschezia and dysuria are the most frequently reported symptoms. Standard diagnosis is carried out by direct visualization and histologic examination of lesions. Pain can be treated by excising peritoneal implants, deep nodules and ovarian cysts, or inducing lesion suppression by abolishing ovulation and menstruation through hormonal manipulation with progestins, oral contraceptives and gonadotropin-releasing hormone agonists. Medical therapy is symptomatic, not cytoreductive; surgery is associated with high recurrence rates. Although lesion eradication is considered a fertility-enhancing procedure, the benefit on reproductive performance is moderate. Assisted reproductive technologies constitute a valid alternative. Endometriosis is associated with a 50% increase in the risk of epithelial ovarian cancer, but preventive interventions are feasible.



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Milestone 5

A pioneering study of diabetes complications



of this triopathy correlated robustly with long duration of diabetes and poor glycaemic control, particularly in the year leading up to the examination. By contrast, the degree of glycaemic control seemed to have little influence on other vascular manifestations, such as coronary and peripheral atherosclerosis.

Owing to a meticulous approach and a high level of patient engagement, Pirart's study was unprecedented in terms of size and duration. Earlier studies often relied on single measurements taken at baseline to determine diabetes severity, but the longitudinal assessments performed by Pirart and his colleagues reflected the true dynamic nature of glycaemic control status and raised the prospect of modifying this status to reduce the risk of degenerative complications.

Pirart acknowledged that his data could not prove the existence of a causal link between long-term hyperglycaemia and the diabetic triopathy. However, his work laid the foundations for further investigations, such as the UK Prospective Diabetes Study, which set out to determine whether improved glycaemic control could prevent the development of complications from type 2 diabetes. Researchers are also exploring the mechanisms underlying glucose-mediated vascular damage, including defects in the mitochondrial electron transport chain.

By the middle of the twentieth century, the link between diabetes and degenerative conditions such as neuropathy, retinopathy and nephropathy was already well established. However, the precise relationship between these complications and glycaemic control was not fully appreciated until the publication of a ground-breaking longitudinal study, which was initiated by the Belgian physician Jean Pirart in 1947 and continued for over three decades.

"In 1947, I had the good fortune to take up practice at the Cesar de Paepe Clinic in Brussels, which treated hundreds of diabetic patients," explained Pirart in a key paper charting the first 25 years of the study, which was first published in French in 1977 and was translated into English in 1978. "The faithful attendance of these patients and the quality of their files over the preceding years gave me the idea to attempt a longitudinal study, which I have been able to follow for more than 30 years because of my long association with the clinic."

The 1977 paper included data from 4,398 patients with diabetes, each of whom underwent a series of examinations, including blood

tests and neurovascular evaluations, at least once a year for a period of up to 25 years. In total, around 21,000 such examinations were conducted over the first 25 years of the study. At each assessment, the patients were rated according to their level of glycaemic control (good, fair or poor).

In light of the frequent co-occurrence of neuropathy, retinopathy and nephropathy, Pirart adopted the practice of referring to these manifestations as a 'diabetic triopathy', as originally proposed by Root and colleagues in 1954. The risk of developing one or more components

"Owing to a meticulous approach and a high level of patient engagement, Pirart's study was unprecedented in terms of size and duration"

tes and more precisely on hyperglycaemia, concluded Pirart in his paper. "Of course, this can be lessened by treatment. This fact should encourage physicians to strive toward normoglycaemia in diabetes therapy, while at the same time recommending various methods of hygiene which could slow down the development of atheromatosis and of hypertension."

Heather Wood Chief Editor,
Nature Reviews Neurology

Milestone studies

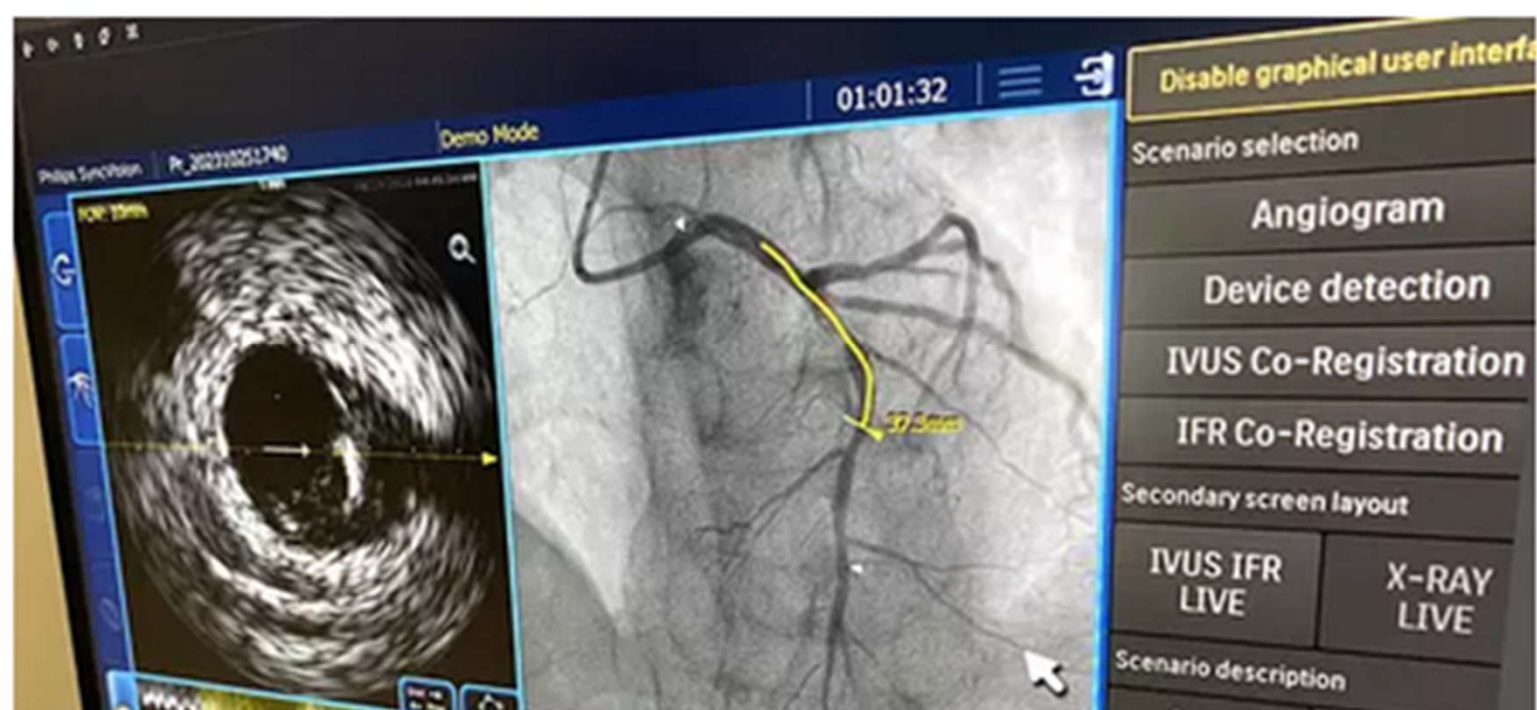
Pirart, J. Diabetes mellitus and its degenerative complications: a prospective study of 4,400 patients observed between 1947 and 1973. Part 1. *Diabetes Care* **1**, 168–188 (1978) | Pirart, J. Diabetes mellitus and its degenerative complications: a prospective study of 4,400 patients observed between 1947 and 1973. Part 2. *Diabetes Care* **1**, 252–263 (1978)

Further reading

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Expert calls for education, research to spur adoption of intravascular ultrasound

Chad Van Alstin | March 21, 2024 | Health Imaging |
Ultrasound





Standards and Guidelines

Intravascular Ultrasound Use in Peripheral Arterial and Deep Venous Interventions: Multidisciplinary Expert Opinion From SCAI/AVF/AVLS/SIR/SVM/SVS



Eric A. Secemsky, MD, MSc^{a,b,*}, Herbert D. Aronow, MD, MPH^{c,d},
Christopher J. Kwolek, MD, MBA^{b,e}, Mark Meissner, MD^f, Patrick E. Muck, MD^g,
Sahil A. Parikh, MD^h, Ronald S. Winokur, MDⁱ, Jon C. George, MDⁱ, Gloria Salazar, MD^k,
Erin H. Murphy, MD^l, Mary M. Costantino, MD^m, Wei Zhou, MDⁿ, Jun Li, MD^o,
Robert Lookstein, MD^p, Kush R. Desai, MD^q

What makes gouty inflammation so variable?

Robert Terkeltaub^{1,2}

variable?

Robert Terkeltaub^{1,2}

Abstract

Acute gout arthritis flares contribute dominantly to gout-specific impaired health-related quality of life, representing a progressively increasing public health problem. Flares can be complex and expensive to treat, partly due to the frequent comorbidities. Unmet needs in gout management are more pressing given the markedly increasing gout flare hospital admission rates. In addition, chronic gouty arthritis can cause joint damage and functional impairment. This review addresses new knowledge on the basis for the marked, inherent variability of responses to deposited urate crystals, including the unpredictable and self-limited aspects of many gout flares. Specific topics reviewed include how innate immunity and two-signal inflammasome activation intersect with diet, metabolism, nutritional biosensing, the microbiome, and the phagocyte cytoskeleton and cell fate. The paper discusses the roles of endogenous constitutive regulators of inflammation, including certain nutritional biosensors, and emerging genetic and epigenetic factors. Recent advances in the basis of variability in responses to urate crystals in gout provide information about inflammatory arthritis, and have identified potential new targets and strategies for anti-inflammatory prevention and treatment of gouty arthritis.



certain small molecules, including colchicine, invert decreases in ABCG2 141 K cell surface localization and function [78]. Finally, it will be interesting to ascertain whether the high frequency of nocturnal onset of acute gout flares [79] is driven by decreases in endogenous inflammation suppressors beyond altered activity of the hypothalamic-pituitary-adrenal corticosteroid axis.

Conclusions

Multiple recent discoveries have revealed that urate crystals form at a variety of unexpected sites, but without clear inflammatory sequelae. Work in the last few years has elucidated factors involved in the marked, inherent variability of responses to urate crystals and onset, extent, and duration of flares of acute gouty inflammation. Knowledge of how innate immunity intersects with diet, metabolism, the microbiome, genetics and epigenetics, and phagocyte fate in shaping inflammatory responses to urate crystal deposits in gout provides valuable lessons about inflammation, as well as potential new biomarkers and targets for therapy in gout.

THE END