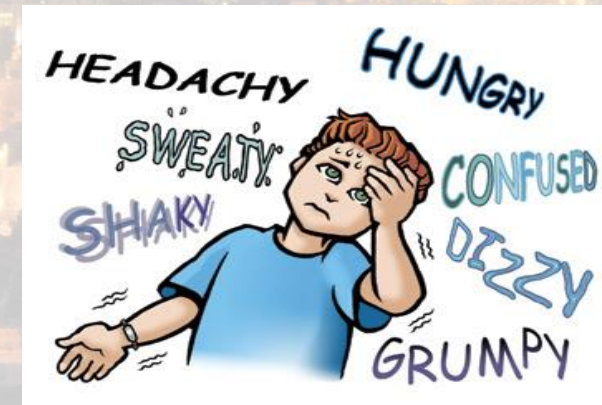




Recurrent PreSyncope

Batla Falah M.D
Hadassah Medical Center
Israel

- 57 y.o female patient.
- Recurrent PreSyncope, sweating, numbness, abdominal pain .



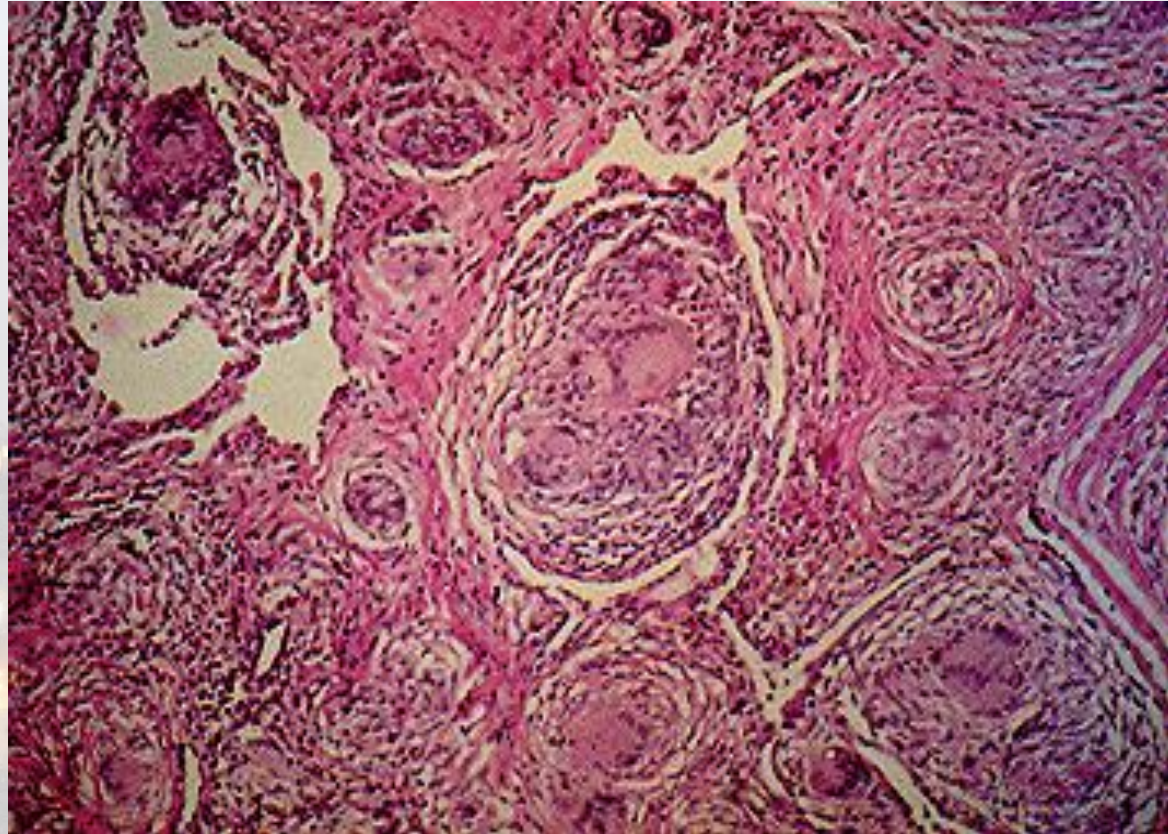
Abdominal Pain – Family physician

1

2

3

4



Still outpatient...

HEADACHY

HUNGRY

SWEATY

CONFUSED

SHAKY

DIZZY

GRUMPY



CHEK[®]
forma

CEBIA Y DISPOSITIVO DE PUNCIÓN

Medición de Glucosa

MEDICIÓN SEGURA
Y CONFIABLE

MEDICIÓN RÁPIDA
EN 5 SEGUNDOS

PEQUEÑA MUESTRA
DE SANGRE

Roche



Admission

BP 144/72 35.9C , 98% Sat RA , Glucose- 70

Physical Exam- tenderness LUQ + LUQ

ביוכימיה

NA	K	CL	GLU	UREA	CRE	CA	P	TP	ALB	ALT	AST	ALK.P	חומר	תאריך שעה
139	4.8		<u>3.9</u>	4.9	69				38	15		67	דם	01/12/14 10:54
137	4.3	100	<u>3.4</u>	5.7	71	2.46	1.4						דם	02/12/14 08:34

ביוכימיה

GGTP	T.BIL	D.BIL	DIA	LDH	UR.AC	MG	CHOL	HDL	LDL	TG	CPK	TROT-HS	חומר	תאריך שעה
	7		82										דם	01/12/14 10:54

הורמונים

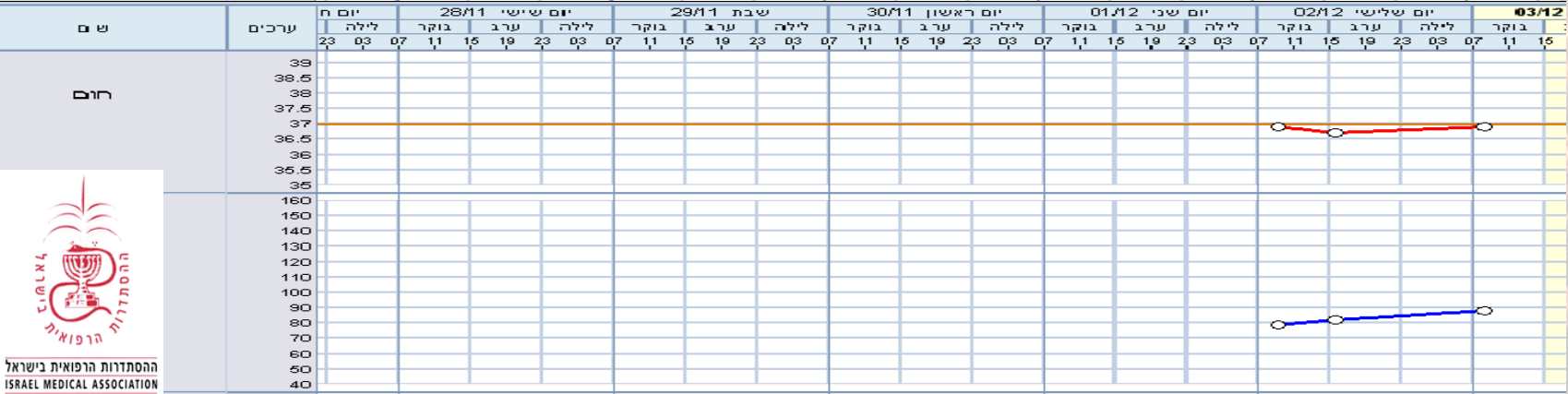
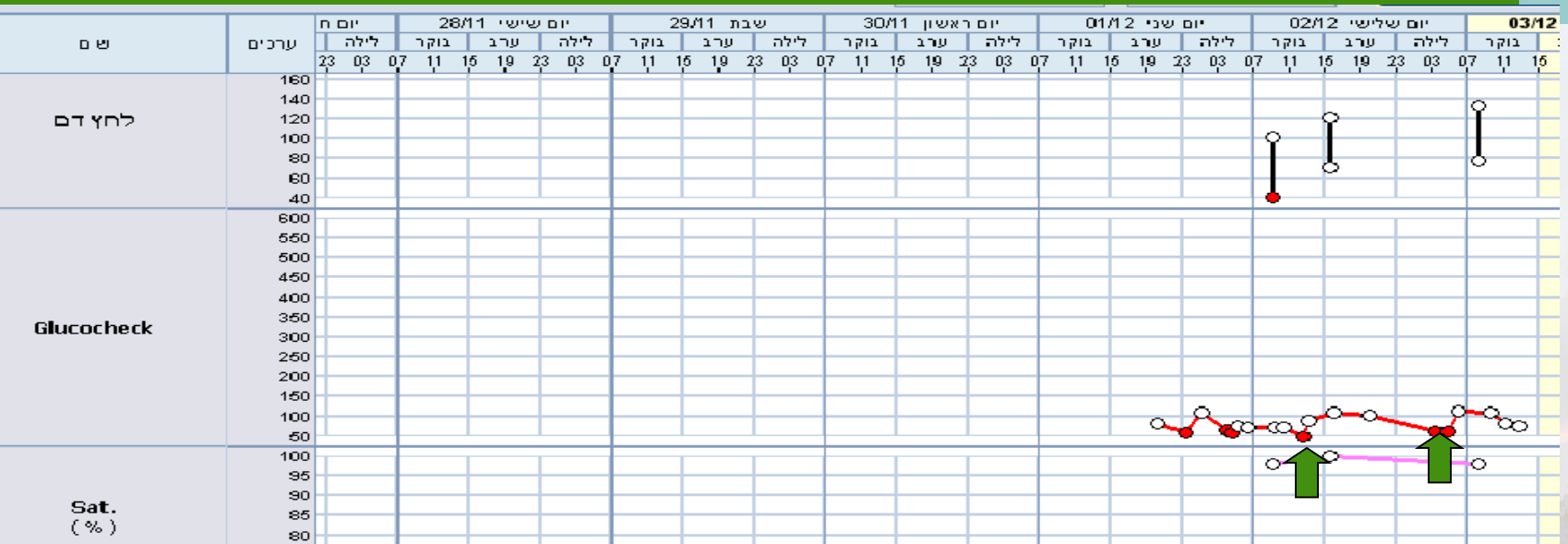
LH	FSH	TEST	ACTH	COR	DHEAS	PTH	חומר	תאריך שעה
					571.93		דם	02/12/14 08:35

ספירה

WBC	RBC	HGB	HCT	MCV	MCH	MCHC	PLT	MPV	RETIC	RDW	חומר	תאריך שעה
5.6	5.05	12.6	40.0	79.2	<u>24.9</u>	<u>31.5</u>	290.0	8.3		<u>15.1</u>	דם	01/12/14 10:53
6.2	4.97	12.5	38.5	77.4	<u>25.2</u>	32.6	260.0	8.9		<u>16.4</u>	דם	02/12/14 08:28



On the ward



72 Hours fasting test

Hypoglycemia: Interpretation of laboratory tests*

Symptoms, signs, or both	Glucose (mg/dL)/ (mmol/L)	Insulin (microU/mL)/ (pmol/L)	C-peptide (nmol/L)/ (ng/mL)	Proinsulin (pmol/L)	Beta-hydroxybutyrate (mmol/L)	Glucose increase after glucagon (mg/dL)/ (mmol/L)	Circulating oral hypoglycemic agent	Antibody to insulin	Diagnostic interpretation
No	<55/3	<3/20.8	<0.2/0.6	<5	>2.7	<25/1.4	No	No	Normal
Yes	<55	>>3	<0.2	<5	≤2.7	>25	No	Neg (Pos)	Exogenous insulin
Yes	<55	≥3	≥0.2	≥5	≤2.7	>25	No	Neg	Insulinoma, NIPHS, PGBH
Yes	<55	≥3	≥0.2	≥5	≤2.7	>25	Yes	Neg	Oral hypoglycemic agent
Yes	<55	>>3	>>0.2*	>>5*	≤2.7	>25	No	Pos	Insulin autoimmune
Yes	<55	<3	<0.2	<5	≤2.7	>25	No	Neg	IGF ^Δ
Yes	<55	<3	<0.2	<5	>2.7	<25	No	Neg	Not insulin (or IGF)-mediated



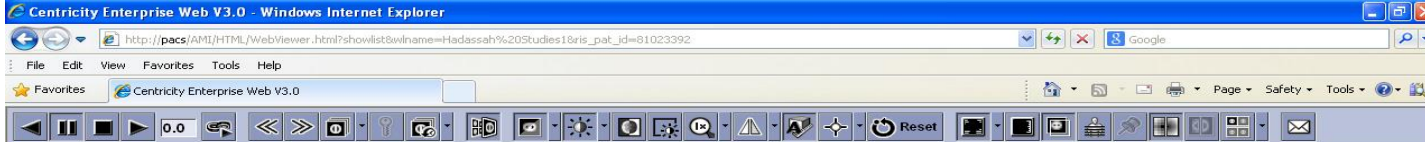
≥; Pos: positive; PGBH: post gastric bypass hypoglycemia; NIPHS: noninsulinoma pancreatogenous hypoglycemia syndrome; IGF: insulin-like growth factor. findings during fasting or after a mixed meal in normal individuals with no symptoms or signs despite relatively low plasma glucose concentrations (ie, Whipple's triad :ed) and in individuals with hyperinsulinemic (or IGF-mediated) hypoglycemia or hypoglycemia caused by other mechanisms. ide and proinsulin concentrations are low. ro-IGF-II, free IGF-II, IGF-II/IGF-I ratio.

modified for this publication to include mmol/L equivalent data. Reproduced with permission from: Cryer PE, Axelrod L, Grossman AB, et al. Evaluation and management of adult

CT- Abdomin

בדיקת ס.ט. סקן-בטן, נאג

KAROT NA



KAROT, NA
Acq Date:04/12/2014
Acq Time:09:19:06
Order Date:04/12/2014
MRN:81023392

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CTS.
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[L]

G: 20MMX 14MM

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PORTAL
CONTRAST

C102
W461

Nuclear Somatostatin Scan

Hyp
 lyn

Centricity Enterprise Web V3.0 - Windows Internet Explorer

http://pacs/AMI/HTML/WebViewer.html?showlist&wname=Hadassah%20Studies1&ris_pat_id=81023392

File Edit View Favorites Tools Help

Centricity Enterprise Web V3.0

KAROT, NA(2) 07:59:54 23/12/2014 (81023392) גאנאם קארוט/גאנאם קארוט of 2 images)

ScreenCap05_255 Volumetric MI 23/12/2014

Discovery ST dslbay86 KAROT, NA
 Acq Date:26/12/2014
 Acq Time:13:45:00
 Order Date:23/12/2014
 MRN:81023392

Se:1005
 Im:0

0.98 mm
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 SFOV 50.0 cm
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0.98 mm
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0.98 mm
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 SFOV 50.0 cm
 -20.27

W: 400
 L: 401.172

HYBRID_CT Coronal

PT Coronal

HYBRID_CT + PT Coronal

HYBRID_CT Axial

PT Axial

HYBRID_CT + PT Axial

HYBRID_CT Sagittal

PT Sagittal

HYBRID_CT + PT Sagittal

HYBRID_CT Transaxial

PT Transaxial

HYBRID_CT + PT Transaxial

HYBRID_CT Sagittal

PT Sagittal

HYBRID_CT + PT Sagittal

HYBRID_CT Axial

PT Axial

HYBRID_CT + PT Axial

HYBRID_CT Sagittal

PT Sagittal

HYBRID_CT + PT Sagittal

HYBRID_CT Axial

PT Axial

HYBRID_CT + PT Axial

C117
 W282

Local intranet

start

http://virgo.hadas...

Document1 - Micro...

Centricity Enterpris...

Hadassah Medical ...

קבוצת ב"ב חולקה ...

http://oraweb6:80...

EN

19:14

Biopsy

ממצא מיקרוסקופי

Blood and small fragments of granulomas without central necrosis artifact present. Chromogranin endocrine/neuroendocrine staining negative for fungi and acid fast bacteria. The findings are morphologically consistent with sarcoidosis.
Clinical/radiological correlation



with well-formed epithelioid granuloma tissue with crush preparation. Special stains demonstrate negative results for Ziehl-Neelsen stains negative for tuberculosis.
Sarcoidosis.



Pancreatic Sarcoidosis?

PANCREATIC — Clinically apparent pancreatic sarcoidosis is rare [21,47-49]. One autopsy series of 92 patients with sarcoidosis documented incidental pancreatic involvement in five [4]. A different report reviewed nine major series and found pancreatic granulomas in 3 (1 percent) of 287 postmortem examinations [3].

A literature review in 1996 found only 13 patients with biopsy-proven granulomas in the pancreas or peripancreatic nodes [47]. They added six of their own cases, one involving the body of the pancreas and five infiltrating the peripancreatic nodes. A clinically helpful profile of pancreatic sarcoidosis emerged from their limited review of 18 patients:

- Clinically apparent pancreatic sarcoidosis appears to be more common in women
- Bilateral hilar adenopathy was present on chest radiograph in three-fourths
- Two-thirds of patients have acute abdominal pain, but other typical findings of acute pancreatitis are uncommon

Laboratory, radiographic, and nuclear imaging features are not definitive. Serum amylase and lipase elevation may or may not be present [50,51]. Computed tomography (CT) and ultrasound of the abdomen are useful in demonstrating a pancreatic "mass," which is in the head of the pancreas in about half the cases [21,49]. A diffusely nodular pancreas is noted in the other half. However, imaging tests are unable to distinguish the granulomatous involvement from focal pancreatic inflammation or a carcinomatous mass. Thus, either a CT-guided or laparoscopic biopsy is needed to identify noncaseating granulomas and exclude cancer and mycobacterial or fungal infection [21,50].

TREATMENT — The decision to treat gastrointestinal (GI) sarcoidosis is based upon the activity and extent of disease. Asymptomatic patients generally do not require treatment. For patients who are symptomatic and have a substantial amount of granulomatous inflammation on tissue biopsy, glucocorticoids are the treatment of choice, based on extrapolation from the treatment of pulmonary sarcoidosis [21].

- **Immunosuppressive therapy** — For most forms of gastrointestinal sarcoidosis, when therapy is indicated, we initiate [prednisone](#) 30 to 40 mg given as a single daily dose, and then gradually taper to a maintenance dose of 10 to 15 mg daily, over a period of approximately six months. The proper length of therapy for those who respond to treatment is not known. We usually aim for a duration of therapy of at least one year.

The prognosis of pancreatic sarcoidosis is good; 80 percent of patients improve either spontaneously or with glucocorticoids [47-49].

Endocr J. 2010;57(4):325-30. Epub 2010

Hypoglycemia due to ectopic secretion of IGF-II by the spleen.

Ogiwara Y¹, Mori S, Iwama M, Sawada Y, Kimbara Y, Tamura Y, Chiba Y, Araki A, Yokote K, Maruyama N, Ito H.

Author information

Abstract

Hypoglycemia is reported to be associated with ectopic secretion of insulin-like growth factor-II (IGF-II) by the spleen in patients with an isolated sarcoidosis of the spleen. During hypoglycemia, serum insulin-like growth factor-I (IGF-I) and IGF-II were measured. A high molecular weight form of IGF-II, termed "big" IGF-II, was identified by Western blotting. The objective was to investigate whether the patient's hypoglycemia was caused by the splenic sarcoidosis. Serum insulin-like growth factor-I, serum IGF-II and serum visfatin were measured. After splenectomy, hypoglycemia did not recur and serum insulin-like growth factor-I, serum IGF-II and serum visfatin levels were reduced by half the preoperative level. RT-PCR revealed that the mRNA level of IGF-II in the splenic sarcoidosis was suppressed. These data suggest that ectopic secretion of IGF-II by the splenic sarcoidosis might cause fasting hypoglycemia mainly by suppressing hepatic gluconeogenesis.



"I've brought my own bed with me."

1 a patient with an isolated sarcoidosis of

do Y, Kimbara Y, Tamura Y, Chiba Y, Araki A, Yokote K,

splenic sarcoid infiltrates due to impaired counter-regulation of insulin. We recently identified a patient with hypoglycemia which completely disappeared after splenectomy. There was no abnormality. The objective was to investigate whether the patient's hypoglycemia was caused by the splenic sarcoidosis. Serum insulin-like growth factor-I (IGF-I) and IGF-II were measured. A high molecular weight form of IGF-II, termed "big" IGF-II, was identified by Western blotting. The objective was to investigate whether the patient's hypoglycemia was caused by the splenic sarcoidosis. Serum insulin-like growth factor-I, serum IGF-II and serum visfatin were measured. After splenectomy, hypoglycemia did not recur and serum insulin-like growth factor-I, serum IGF-II and serum visfatin levels were reduced by half the preoperative level. RT-PCR revealed that the mRNA level of IGF-II in the splenic sarcoidosis was suppressed. These data suggest that ectopic secretion of IGF-II by the splenic sarcoidosis might cause fasting hypoglycemia mainly by suppressing hepatic gluconeogenesis.



"WHEN I ASKED IF YOU WERE FLEXIBLE,
MRS. HARKNESS, I WAS TALKING
ABOUT YOUR HOURS!"

