

The Johns Hopkins Hospital 實習成果報告書

陽明醫學系六年級 劉士朋

學號19901006

2016/04/21-05/20

前言

感謝國立陽明大學醫學系、財團法人臺北市清井醫學關懷慈善基金會、榮陽卓越醫師人才培育計畫，以及老師、家人、學長姐、同學及各單位行政人員的幫忙，讓我能有這個出國實習的經驗。一路走來，有許多的前輩鼓勵我，不要因為自己來自經濟狀況不好的家庭就畫地自限，而母親近來病況穩定，父親也希望我趁年輕時多去增廣見聞，苗栗家裡的狀況要我放心。

我喜歡醫學，是因為享受像個偵探般抽絲剝繭的過程，臨床決策的思路總是引人入勝，因此這次我選了兩個風格迥異卻都需要大量鑑別診斷的科別——風濕科(rheumatology)與急診醫學(emergency medicine)。

行前劉瑞玲主任叮嚀，美國的醫療固然有許多令我們稱羨之處，但在讚賞之餘，也要反思在人力、資源與環境差異甚大的台灣，我們怎樣能讓醫療更好。除了自我的提升，我相信此行很重要的目的，是如何把這些經驗化為貢獻，回饋台灣！

急診醫學(emergency medicine)



(上圖) 這個月一起在E-med奮鬥的同學家睿，離開前穿scrub在醫院前合影。

這個月選修的是Advanced Clerkship in Emergency Medicine，不過由於醫院在發給國際學生寫病歷、開醫囑的行政作業上一直有困難，因此我們反而又像是 Emergency Medicine Basic Clerkship的學生。

差異在哪呢？前者的身份是sub-intern，在這個月內要完成16個普通急診班（每個shift為8hr，分為三個時段7-15,15-23,23-3），不用跟著住院醫師行動，而是自己接完病人後與主治醫師討論寫好的病歷與治療方向，並在最後有case presentation；後者的身份是medical student，這個月要完成12個普通急診班，與住院醫師行動，獨立看完病人後報告並討論治療方向，由住院醫師完成病歷與醫囑，此外有3種特殊班——purple shift（時間為13-22）、TONS（Teaching Obs/ Nursing Shift，學習各種技術如抽靜脈血、放鼻胃管與導尿管等等）、EMS ride along（跟著Emergency Medical System緊急救護醫療系統擔任觀察員的角色），並且每週三在simulation center（有點像是設備更高級的臨床技能訓練中心）用模擬病人訓練醫學生對病人的評估與治療決策。

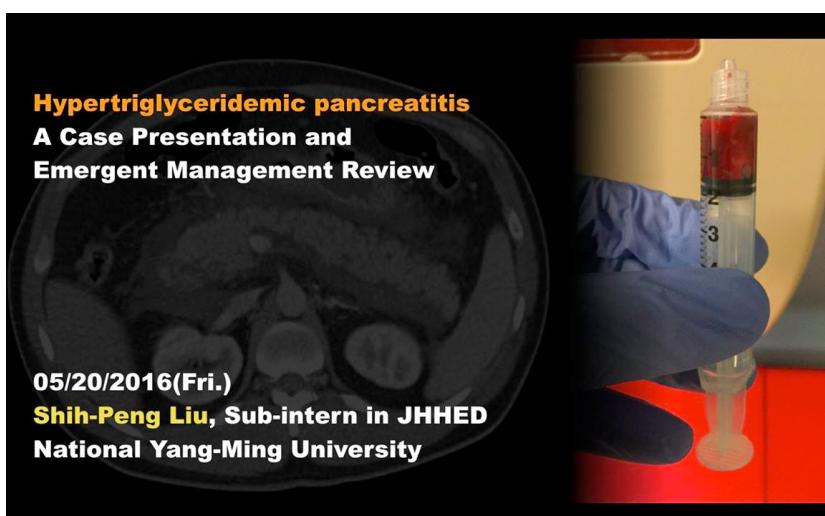
折衷之下，我們跟著住院醫師完成16個shifts，並在最後進行presentation，至於特殊班與臨床模擬訓練自由選擇是否參加。可以自由選擇要在downtown hospital 或是Bayview hospital（較遠，約10分鐘車程），downtown有6張重症床、13張精神科床、27 (north)+21(south)張普通床；Bayview則規模較小些。

Bayview的教學氣氛不錯，可能因為病人量沒那麼緊繃，住院醫師能花比較多時間與我們討論。儘管是對外科急診比較有興趣的學長，也願意花時間印論文細聊心室上心搏過速(SVT)的鑑別診斷與處置；其他常見的病症也不會少——酒精 / 類鴉片中毒(alcohol/ opioid intoxication)、慢性阻塞性肺病急性惡化(COPD AE)、以胸痛與喘表現的恐慌症發作等等。有一次年輕女性下腹痛，報完病史後評估病人有可能是輸卵管扭轉、卵巢囊腫破裂、性傳染病或骨盆腔發炎等等，除了超音波之外也覺得該做個內診，住院醫師就直接讓我去做了，並在雙合診(bimanual examination)做出明顯的單側壓痛。



（上圖）急診PGY2 Dr. Chung與Hopkins二年級醫學生Divine

至於在downtown hospital 遇到最有趣的case，莫過於下面這個了：

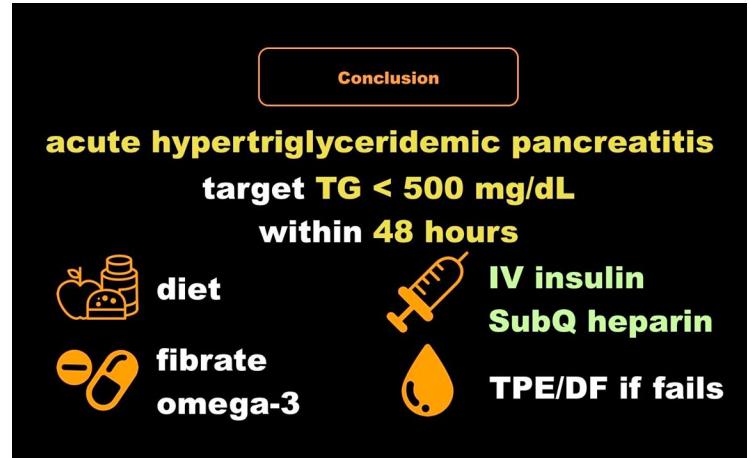


胰臟炎的病人TG高到7000多，上圖右邊為病人的血，巨觀下看起來就油膩膩的；也成為最後報告的主題，討論緊急降TG的方法。

講到胰臟炎會想到什麼呢？除了結石、酒精這兩大原因之外，高三酸甘油酯血症(hypertriglyceridemia)大約佔10%的原因，而近來的研究(Goyal et al. 2016)發現比起酒精胰臟炎，他們嚴重度較高、病

發症也較多。傳統降低TG的方法不是太慢就是不夠力，建議當輔助治療，因此目前建議使用靜脈注射胰島素與皮下注射肝素(IV insulin + SubQ heparin)，若失敗再改用血漿置換(plasmapheresis)，希望能在48小時內把TG降低到500mg/dL以下。

從一開始準備的緊張，到後來順利報完，老師跟同學都說喜歡我的報告（如附件），也算是為這趟實習做了不錯的結尾吧！



(右三)急診醫學的course director Dr. Jung、(左起)JHH的三年級醫學生Mark, Ben與(右一)Andrew

在這裡感受到最大的差異是，急診的班對於醫學生來說是純粹的「學習」，而不像在台灣的醫院有許多「勞動」的成分在，在Hopkins的醫學生參與的每個急診班中，只需要專心在接病人、報告病人與討論治療計劃，再來就是與住院醫師及主治醫師檢討剛才的鑑別診斷流程中，有沒有疏漏或可以改進的地方？因為他們的抽血有專門的phlebotomist，其他procedure也都有clinical technician的協助。

另一方面，是關於台灣民眾的看診習慣，動輒來拿處方籤或不緊急的情況就來就診，大量的看診人次讓看診的時間受到限縮，連帶著教學也會受到影響，這還是一個大環境的問題，需要從教育、政策、法律層面多管齊下，有辦法改變這樣的風氣。

結語

這兩個月以來，除了醫學知識上的增長、親眼見識到許多罕見的疾病（如anti-PL7 syndrome, relapsing polychondritis等等），更令我覺得值得學習的，是遇見的同學與老師都對知識充滿著熱忱——總是自動自發回去讀了兩三篇研究，隔天大家一起來討論。而又因為在團隊當中，可以感受到他們對醫學生的重視，更讓人願意回去讀更多東西，來共同修改決策讓病人的照護變得更好。

我記得有一次問fellow問題，說”Sorry it might be a stupid question.....”，她便直接回”Every question is welcome, no question is stupid.” 在這裡討論的氣氛很好，真的不了解就問，就算是資深的教授也不會露出不屑的眼神，而會很誠懇的與你討論問題。在台灣時我往往會戰戰兢兢，想著「這是不是一個聰明的問題 / 回答？」才敢發言，但在Hopkins，只要你有想法都很歡迎提出來討論，我認為這樣的風氣是很值得被推廣的。

這兩個月是珍貴且難忘的經驗，我會好好應用所學的這些，繼續在台灣的醫界努力！

附件—急診醫學期末個案報告（請見下頁）

Hypertriglyceridemic pancreatitis

A Case Presentation and

Emergent Management Review

05/20/2016(Fri.)

Shih-Peng Liu, Sub-intern in JHHED

National Yang-Ming University



JH46xxxx98 Mr. C

ER at 18:56 on 05/16/2016(Mon.)

A 35-year-old gentlemen



JH46xxxx98 Mr. C

ER at 18:56 on 05/16/2016(Mon.)

A 35-year-old gentlemen

HTN losartan

HLD
TC 568, TG 4265

T2DM
A1c 10.2%



diffuse abdominal pain

A 35-year-old gentlemen

HTN

HLD

T2DM



diffuse abdominal pain

A 35-year-old gentlemen

HTN

HLD

T2DM



acute onset

severe, 10/10

non radiating

nausea with emesis*2, nbnb

no CP, no SOB

no fever/ chills

last BM yesterday, nl

no alcohol use

no gallstone hx

HTN

HLD

T2DM

37.6 °C

109 bpm

18 bpm

124/84 mmHg

Sat 100%

diffuse abdominal pain

nausea with emesis*2, nbnb



HTN

HLD

T2DM

37.6 °C

109 bpm

18 bpm

124/84 mmHg

Sat 100%



diffuse abdominal pain

nausea with emesis*2, nbnb

no rales, no wheezing

**distended abdomen
epigastric tenderness
hypoactive BS**

**no peritoneal signs
no Murphy sign
no Rovsing sign, no obturator sign**



diffuse abdominal pain

nausea with emesis*2, nbnb

**109 bpm distended abdomen
epigastric tenderness
hypoactive BS**

35M

HTN

HLD

T2DM



diffuse abdominal pain

nausea with emesis*2, nbnb

**109 bpm distended abdomen
epigastric tenderness
hypoactive BS**

35M

HTN

HLD

T2DM

DKA

AGE

pancreatitis

appendicitis

bowel perforation

DKA
AGE
pancreatitis

appendicitis
bowel perforation



CBC, CMP, dexi, UA

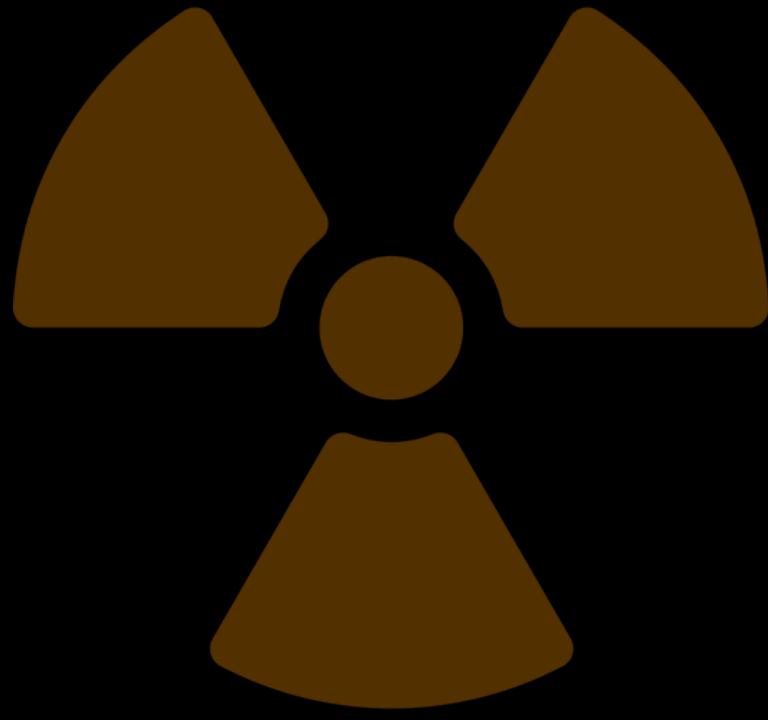
lipase, lipid panel

CXR

abd CT

DKA
AGE
pancreatitis

appendicitis
bowel perforation



CBC, CMP, dexi, UA

lipase, lipid panel

CXR

abd CT



diffuse abdominal pain

nausea with emesis*2, nbnb

109 bpm



distended abdomen
epigastric tenderness
hypoactive BS

35M

HTN

HLD

T2DM

WBC	13.79	Na	120	Lactate	2.8
Hgb	18.9	K	4.9	LDH	403
Hct	43.1	Cl	82	Lipase	6,910
Plt	201	Bicarb	22	T-Bil	0.6
		BUN	18	AST	hero
TC	995	Cr	1.4	ALT	45
TG	7347	Glc	438		



diffuse abdominal pain

nausea with emesis*2, nbnb

109 bpm



distended abdomen
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35M

HTN

HLD

T2DM

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HLD

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TC	995	Cr	1.4	ALT	45
TG	7347	Glc	438	Ca	7.8



diffuse abdominal pain
nausea

109 bpm



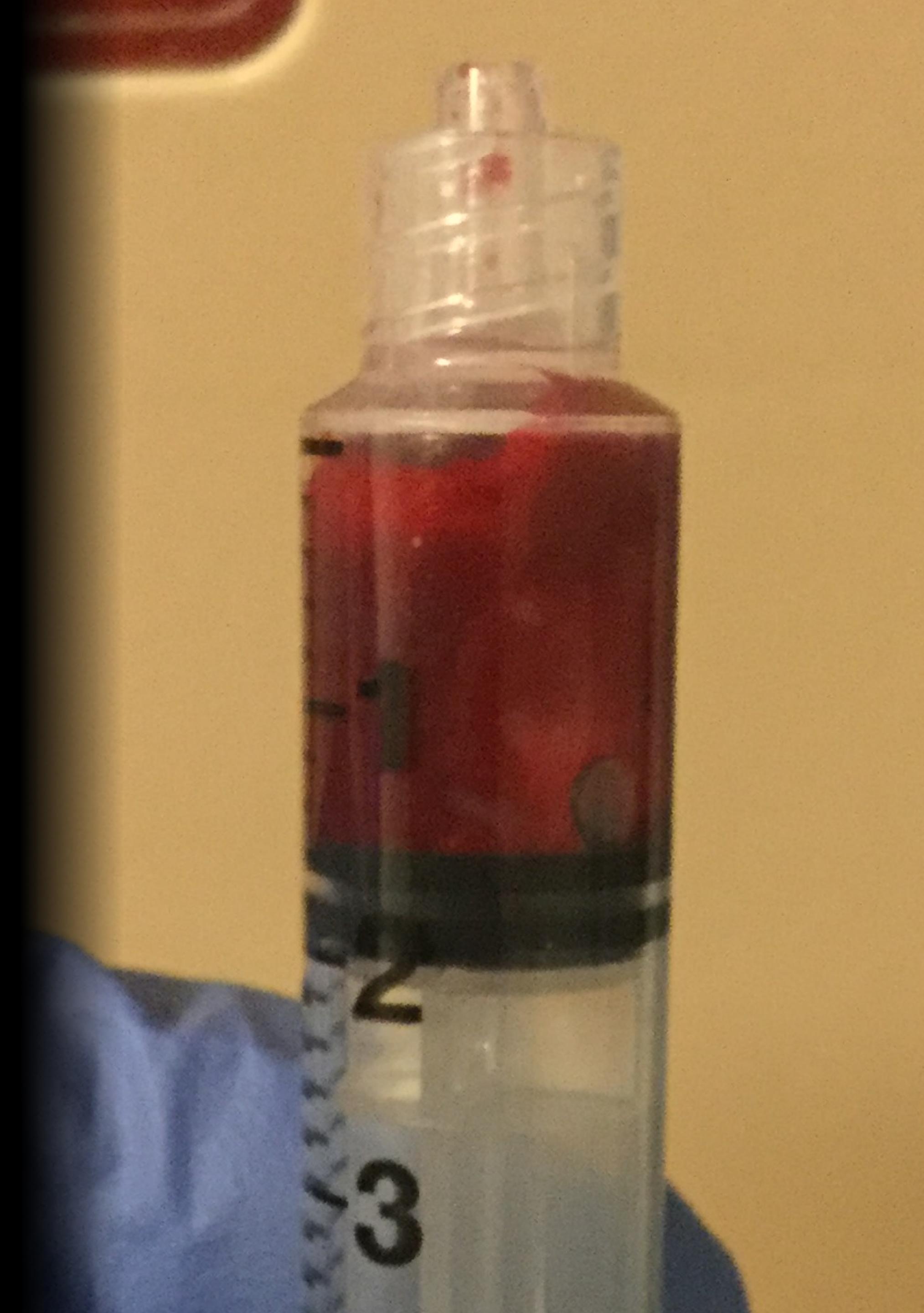
WBC	13.79
Hgb	18.9
Hct	43.1
Plt	201
TC	995
TG	7347

35M

HTN

HLD

T2DM





diffuse abdominal pain
nausea with emesis*2, nbnb
109 bpm distended abdomen
epigastric tenderness
hypoactive BS

35M

HTN

HLD

T2DM

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		BUN	18	AST	40
TC	995	Cr	1.4	ALT	45
TG	7347	Glc	438	Ca	7.8
				ΔNa 14.7	ΔNa 8.1



diffuse abdominal pain

nausea with emesis*2, nbnb

109 bpm



distended abdomen
epigastric tenderness
hypoactive BS

35M

HTN

HLD

T2DM

WBC	13.79	Na	143	Lactate	2.8
Hgb	18.9	K	4.9	LDH	403
Hct	43.1	Cl	82	Lipase	6,910
Plt	201	Bicarb	22	T-Bil	0.6
		BUN	18	AST	40
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35M
HTN
HLD
T2DM

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HTN

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T2DM

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diffuse abdominal pain

nausea with emesis*2, nbnb

109 bpm



distended abdomen
epigastric tenderness
hypoactive BS

35M

HTN

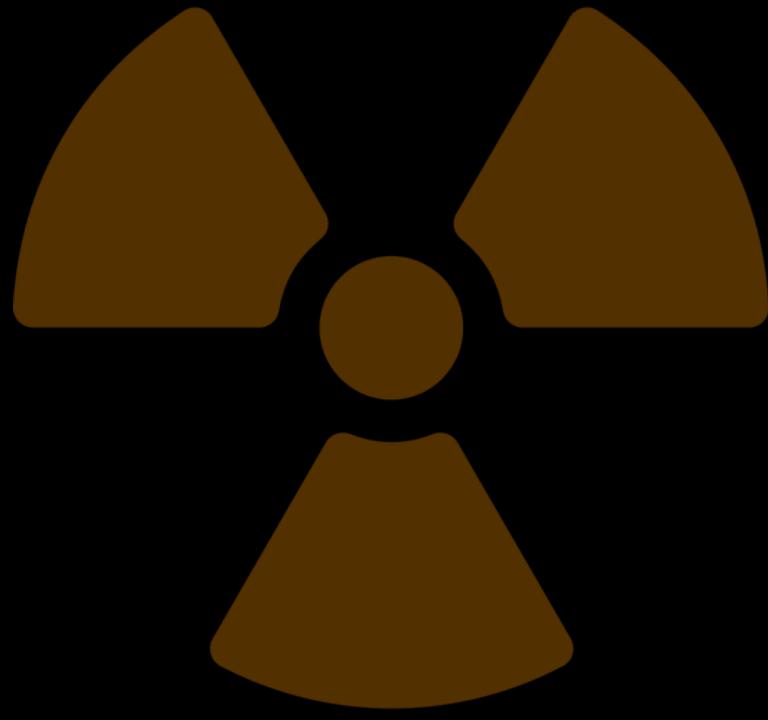
HLD

T2DM

U. pH	5.5
U. protein	2+
U. glucose	>1000
U. ketones	small
U. WBC	0/HPF
U. RBC	1/HPF

DKA
AGE
pancreatitis

appendicitis
bowel perforation



CBC, CMP, dexti, UA

lipase, lipid panel

CXR

abd CT

DKA
AGE
pancreatitis

appendicitis
bowel perforation



CBC, CMP, dxi, UA
lipase, lipid panel



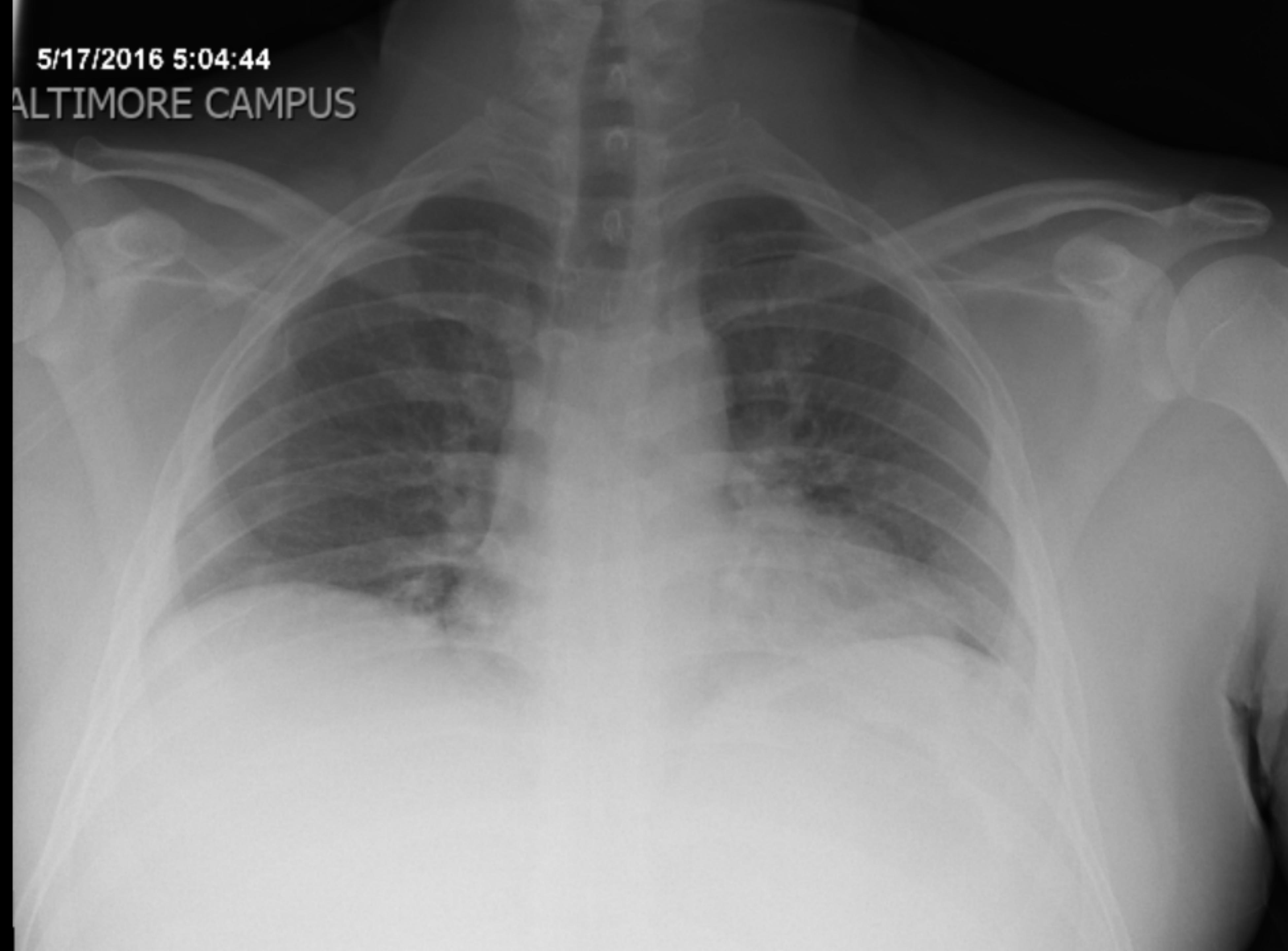
CXR
abd CT

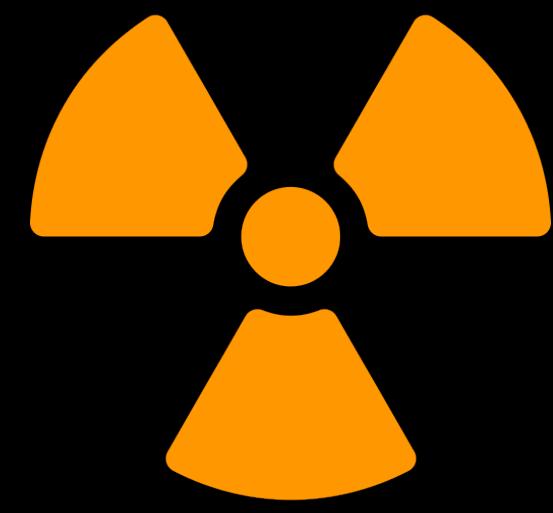


CXR

abd CT

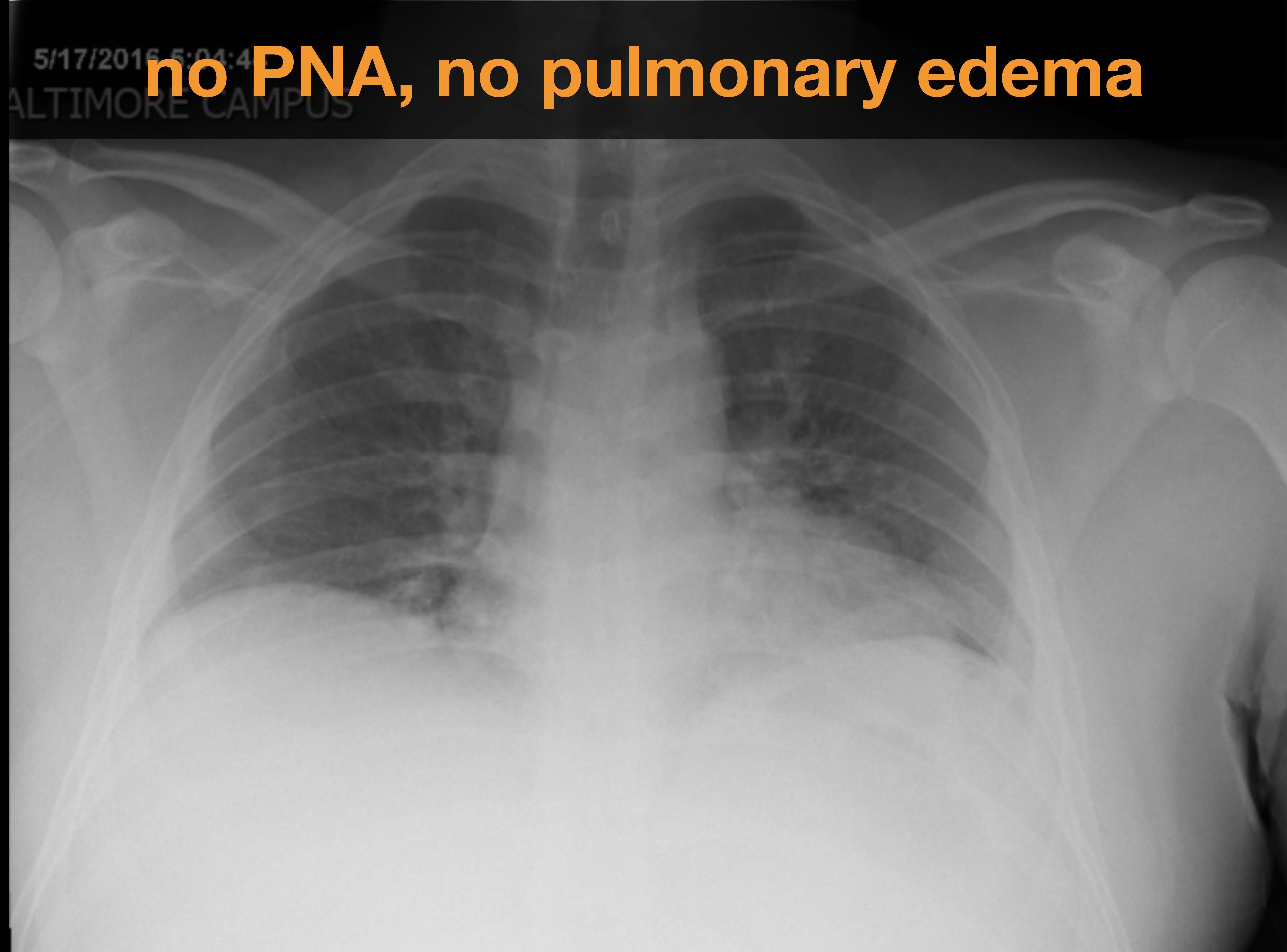
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BALTIMORE CAMPUS

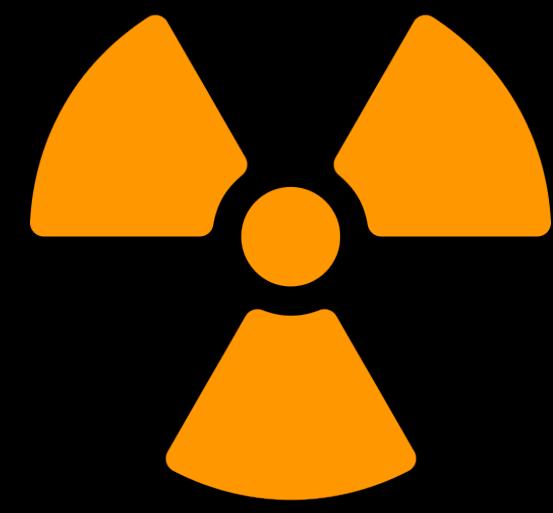




CXR

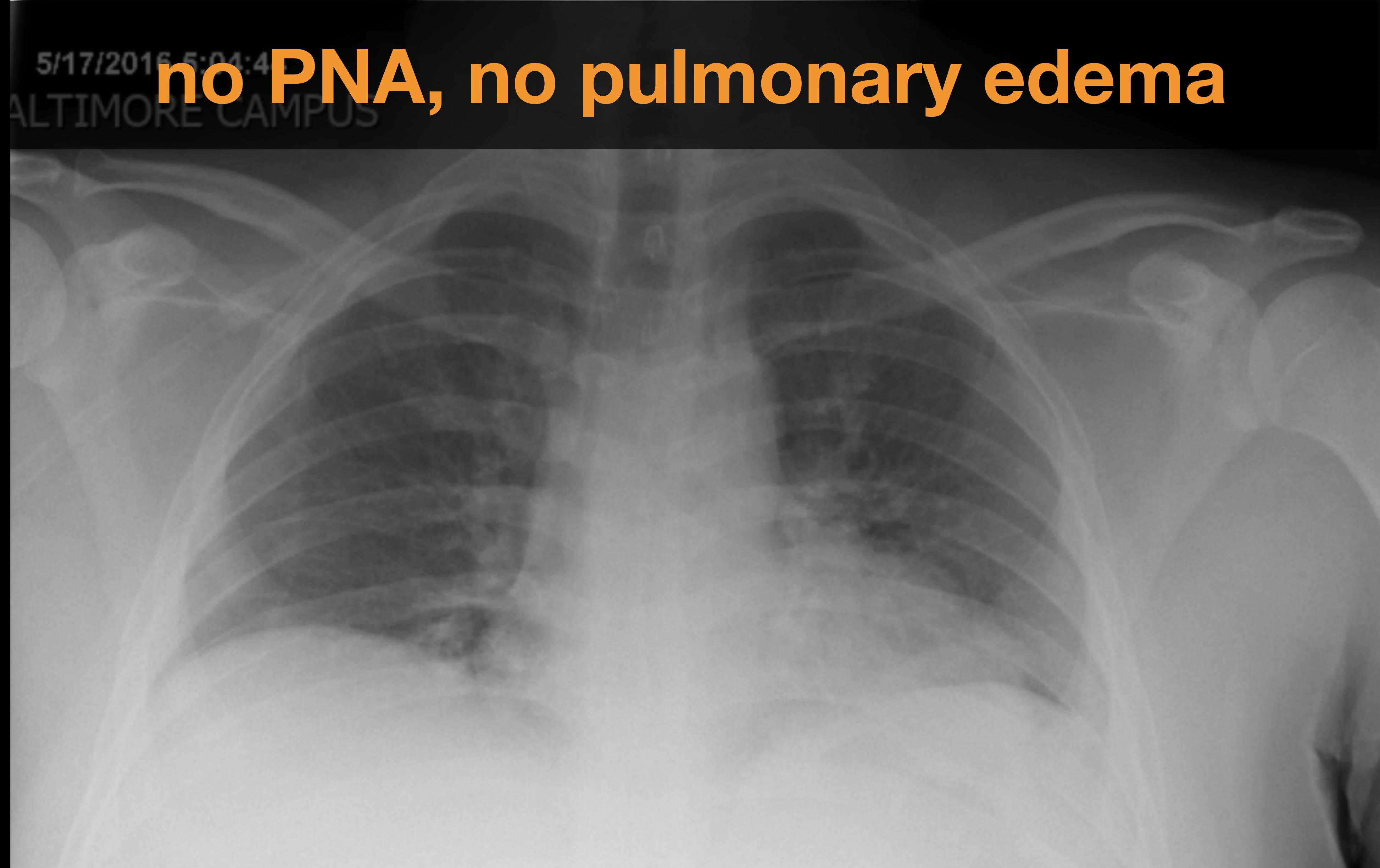
abd CT



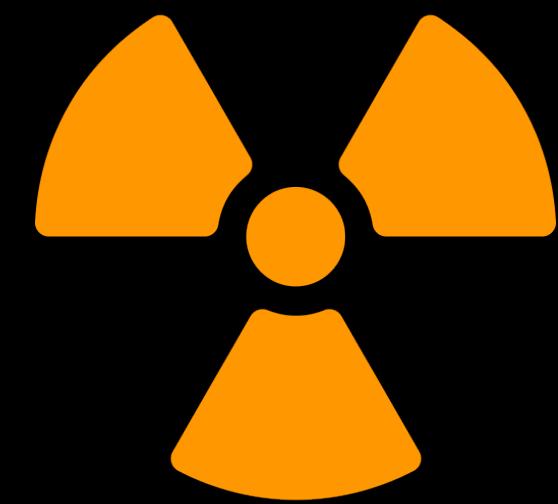


CXR

abd CT



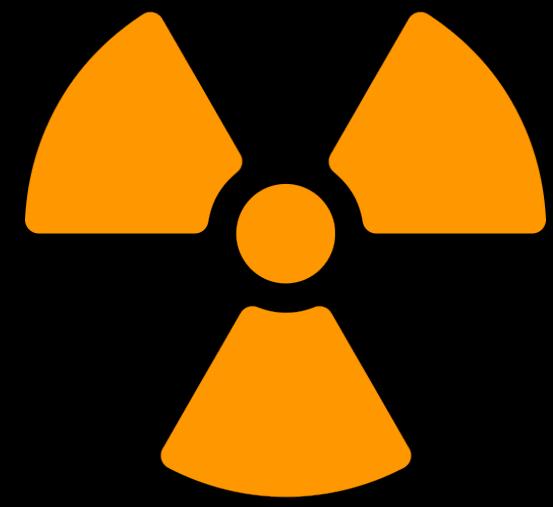
no PNA, no pulmonary edema
no pleural effusion, no free air



CXR

abd CT

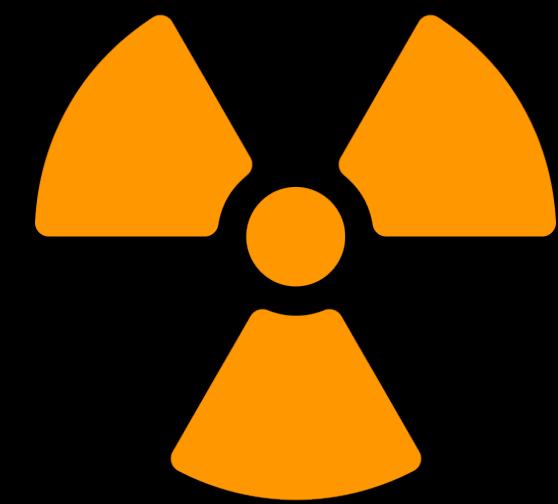




CXR

abd CT

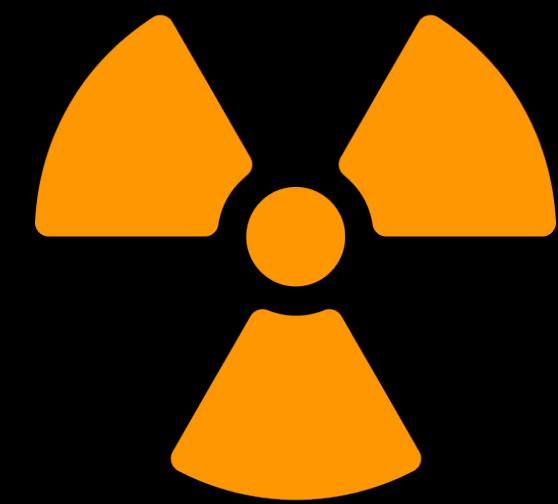




CXR

abd CT

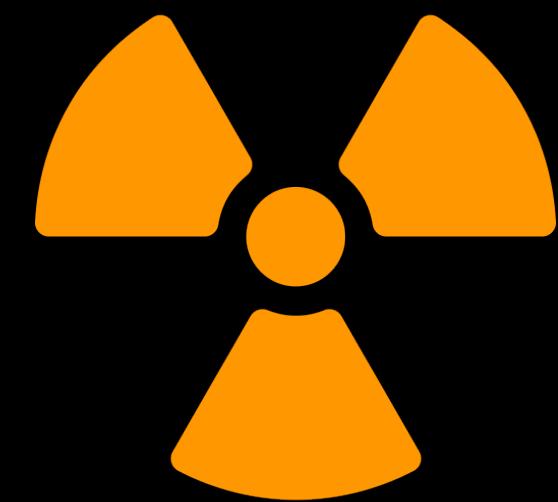




CXR

abd CT

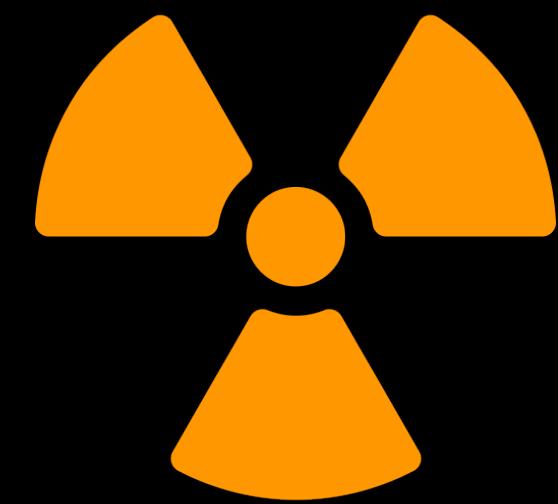




CXR

abd CT





CXR

abd CT

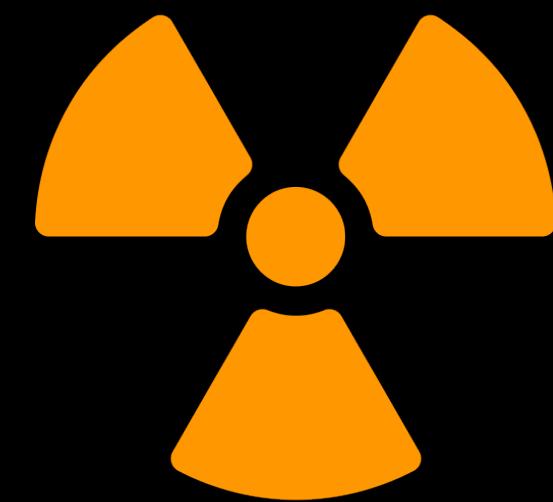




CXR

abd CT

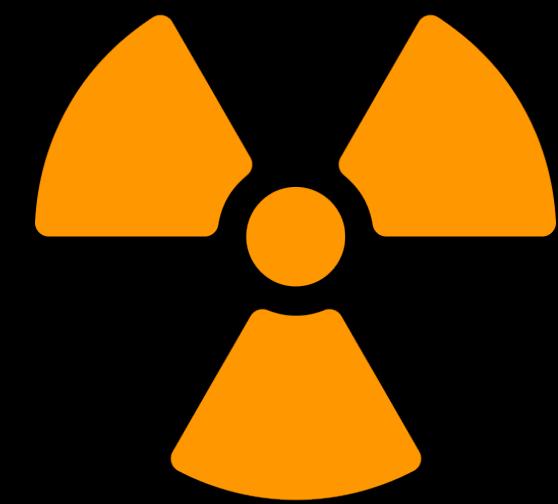




CXR

abd CT

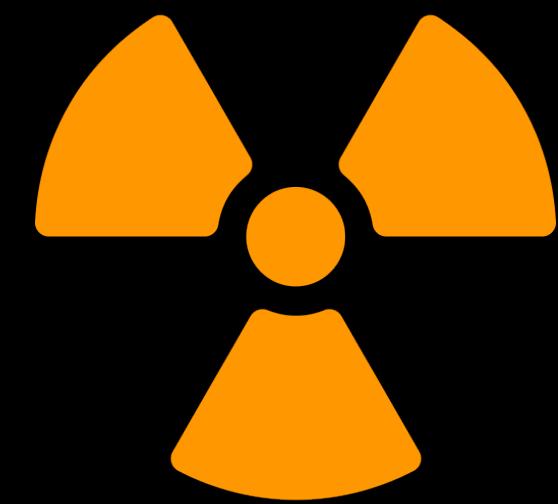




CXR

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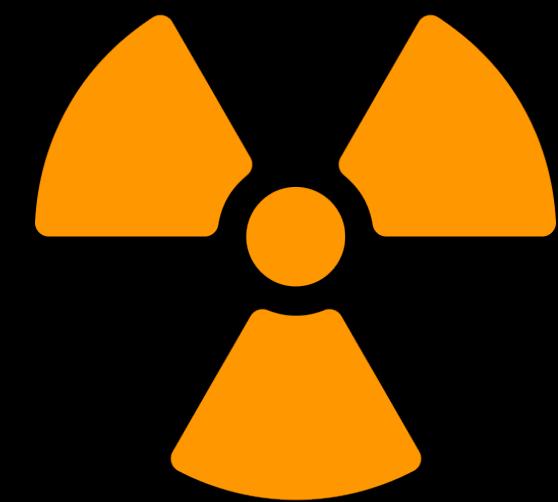




CXR

abd CT





CXR

abd CT



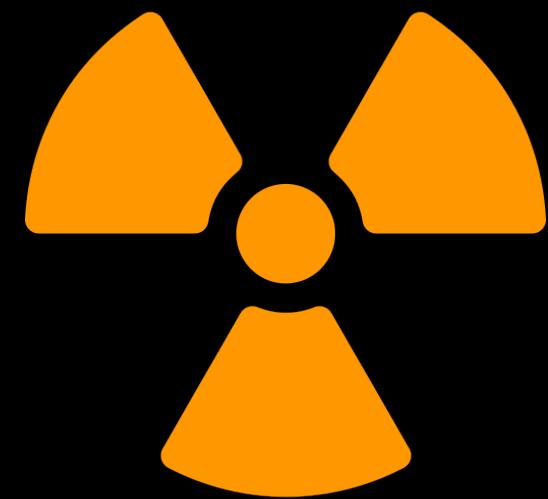


CXR

abd CT



enlarged pancreas, homogenous



CXR

abd CT



enlarged pancreas, homogenous



CXR

abd CT

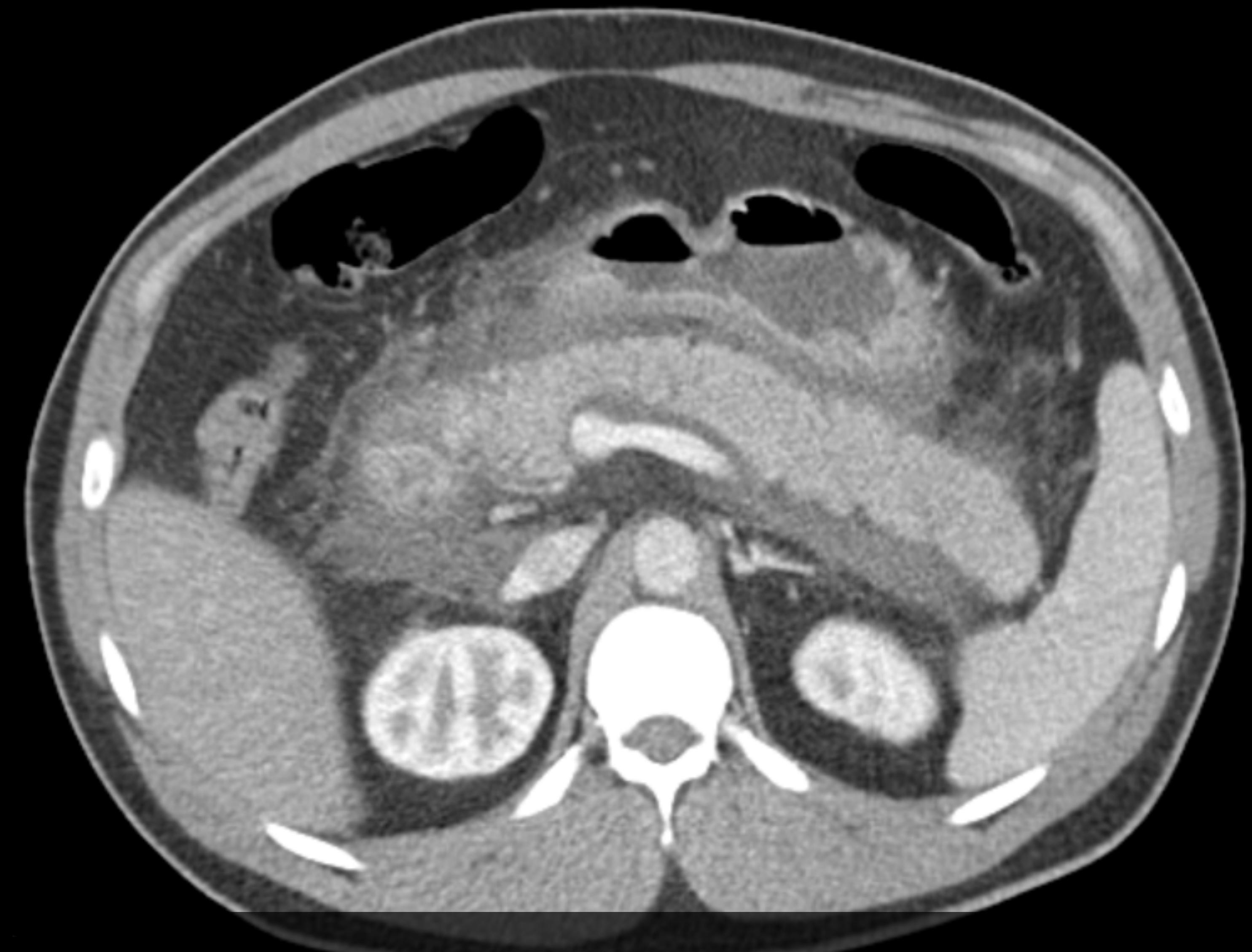


enlarged pancreas, homogenous

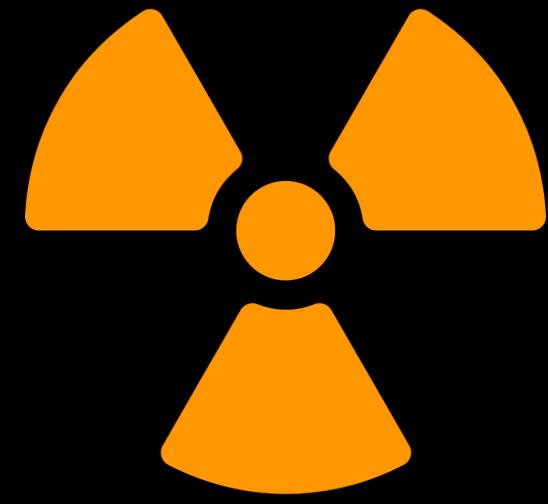


CXR

abd CT



enlarged pancreas, homogenous



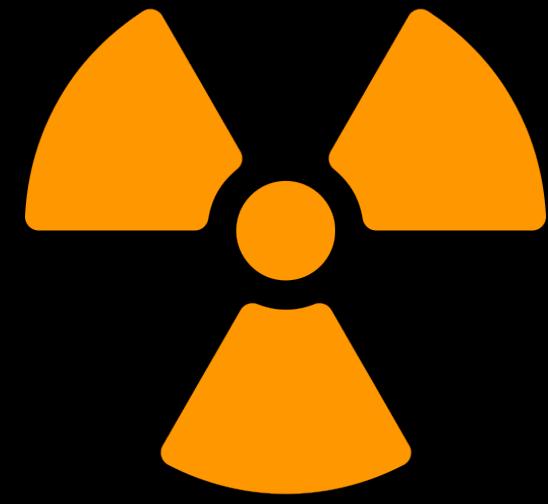
CXR

abd CT



peripancreatic edema

enlarged pancreas, homogenous



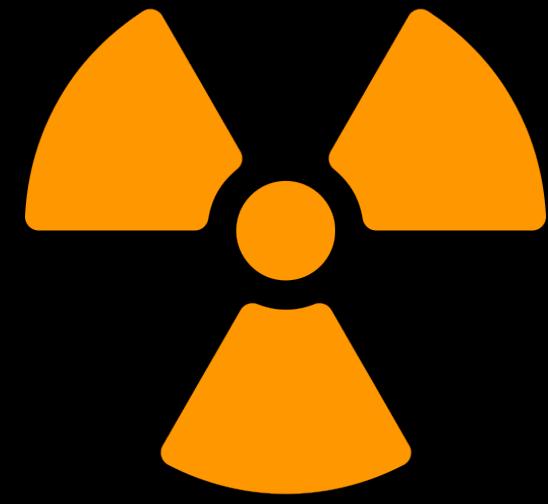
CXR

abd CT



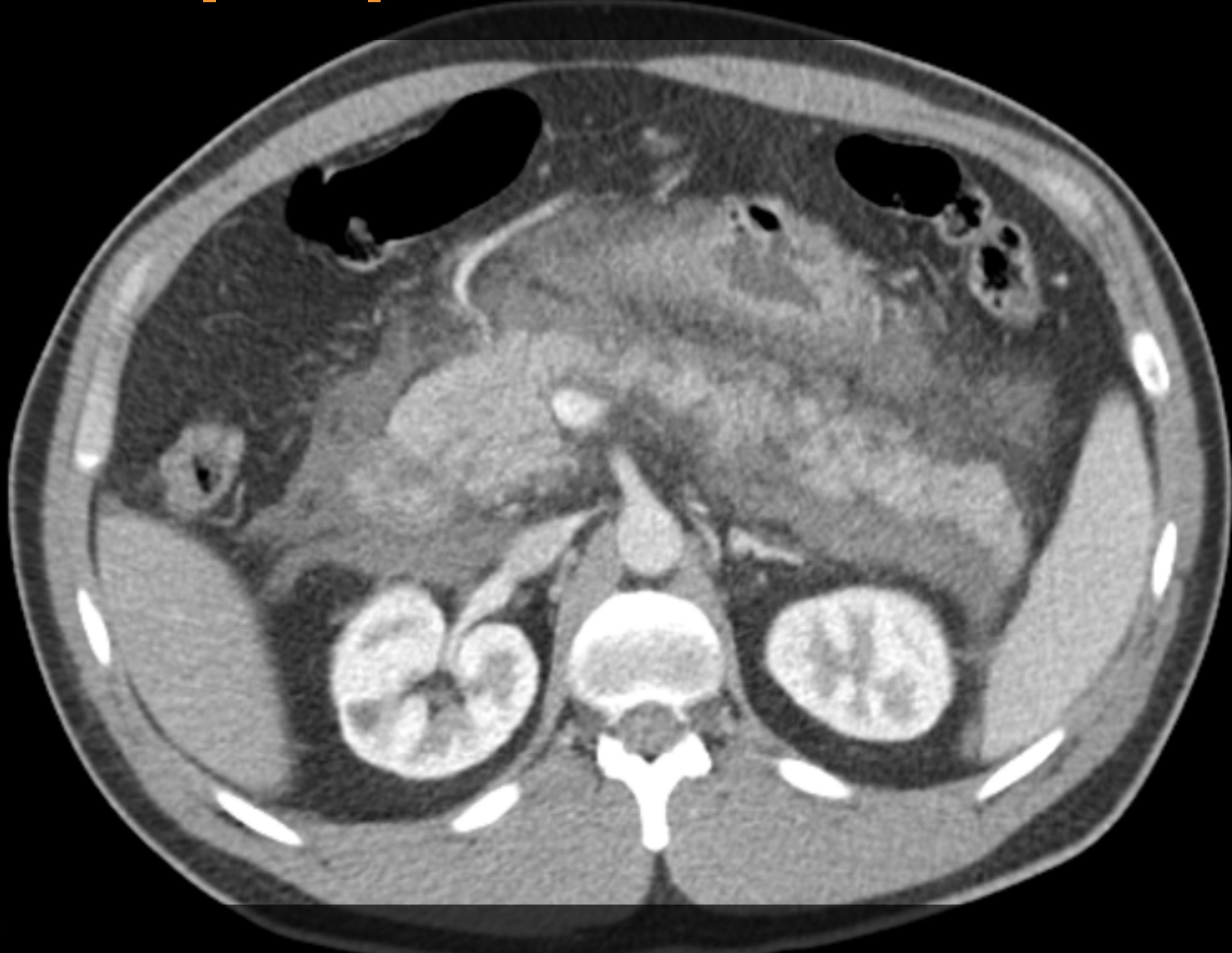
peripancreatic edema

enlarged pancreas, homogenous



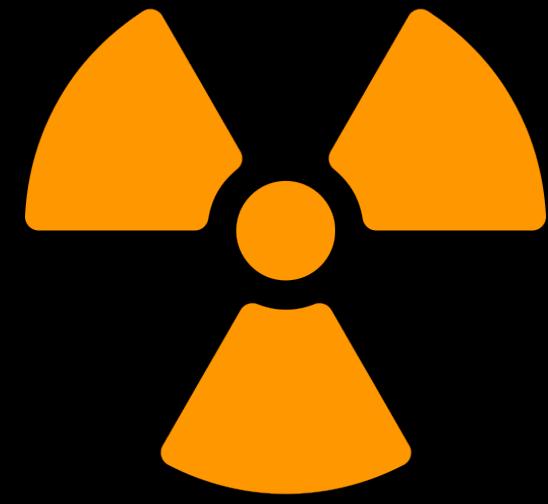
CXR

abd CT



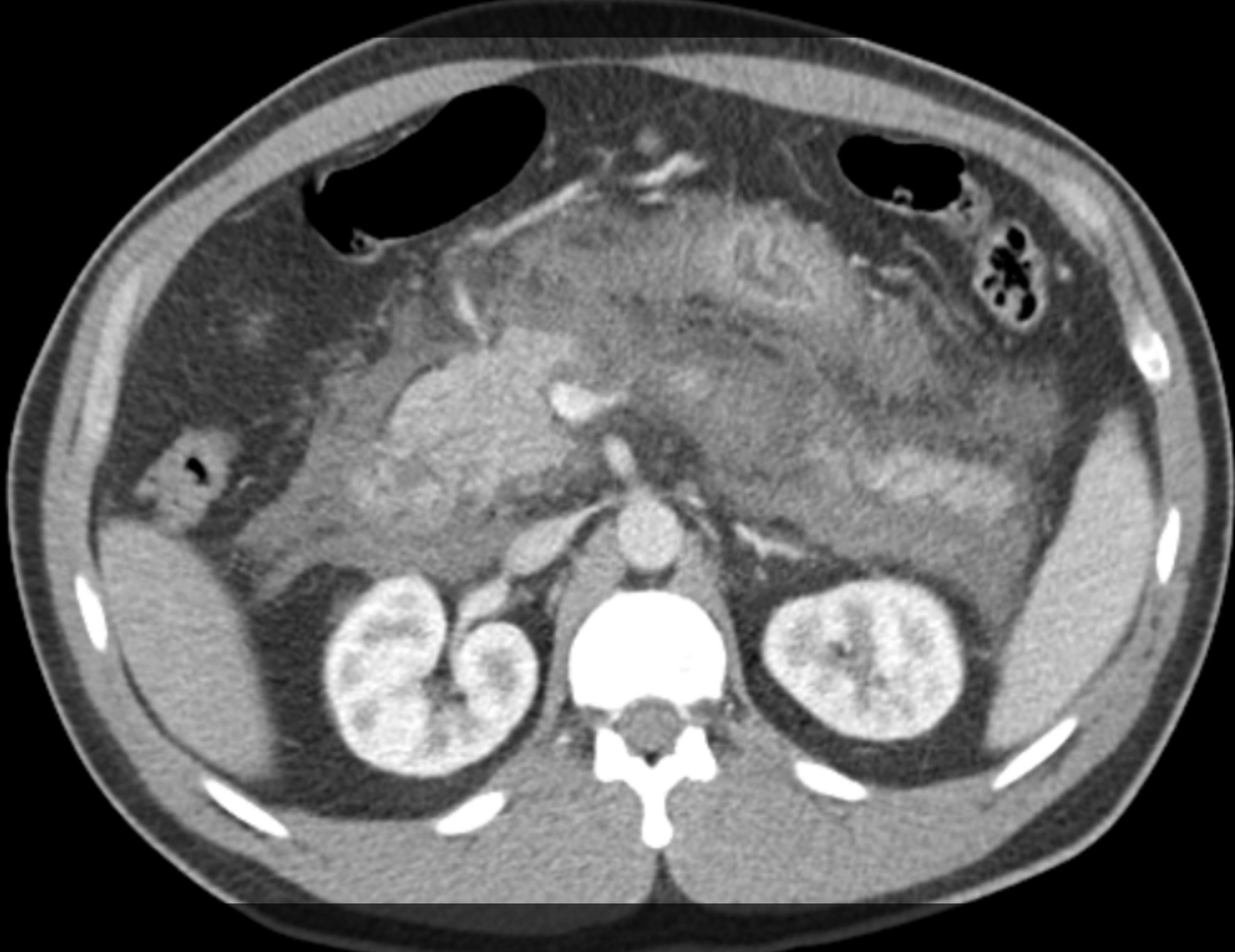
peripancreatic edema

enlarged pancreas, homogenous



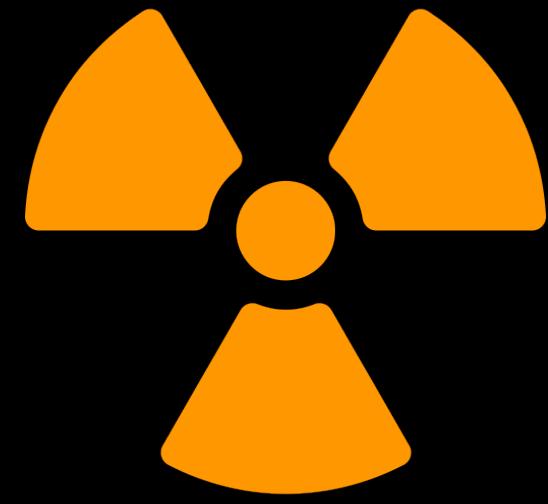
CXR

abd CT



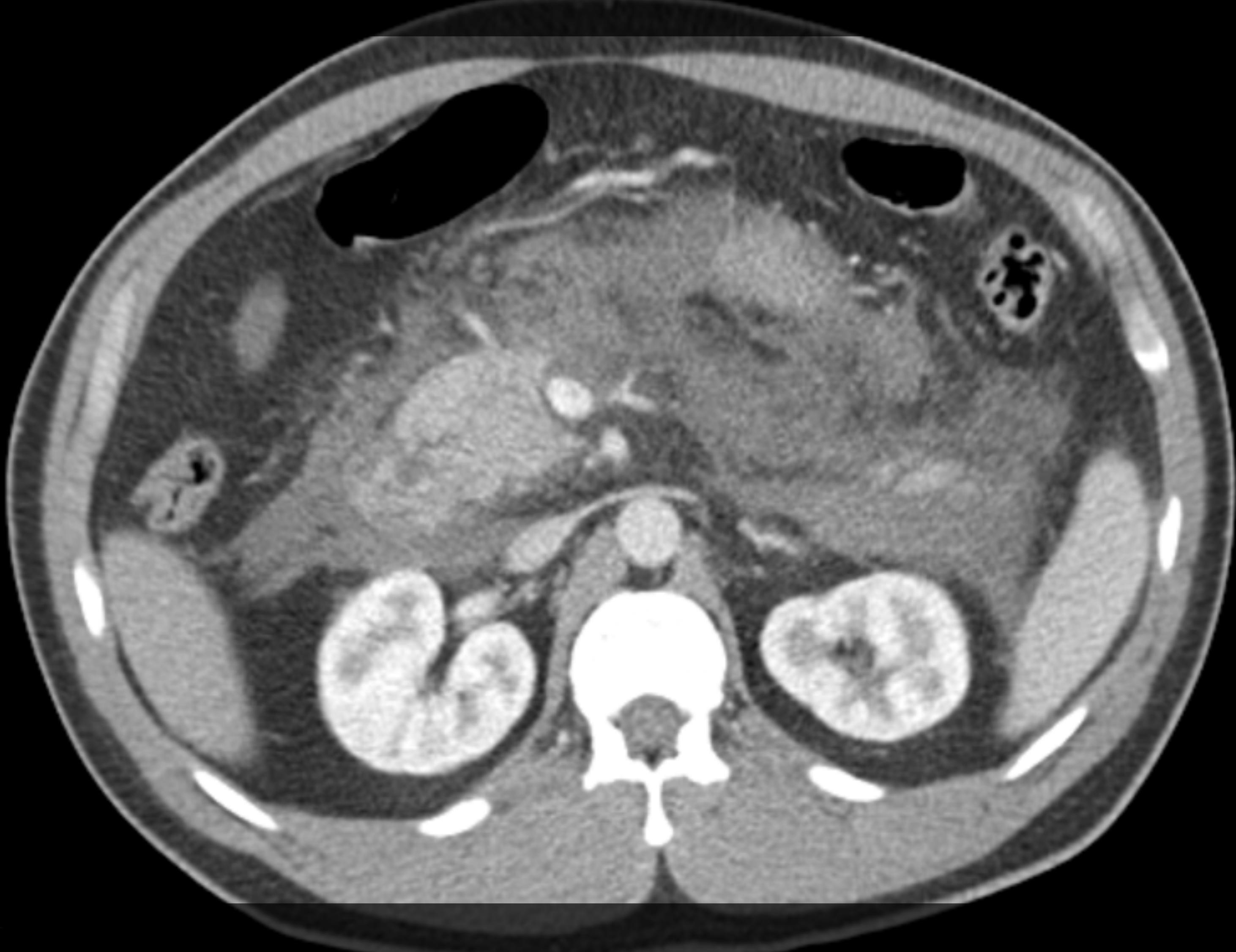
peripancreatic edema

enlarged pancreas, homogenous



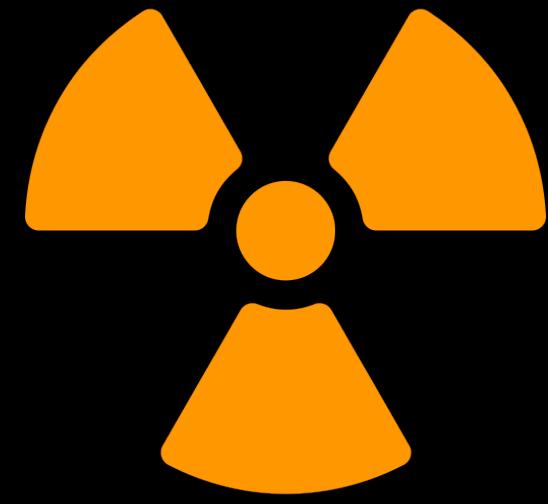
CXR

abd CT



peripancreatic edema

enlarged pancreas, homogenous



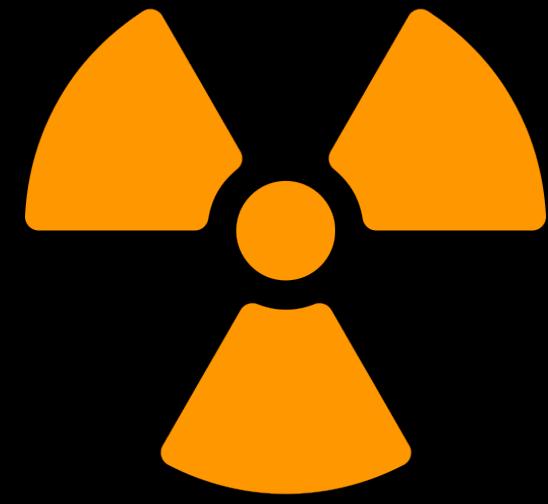
CXR

abd CT



enlarged pancreas, homogenous

peripancreatic edema



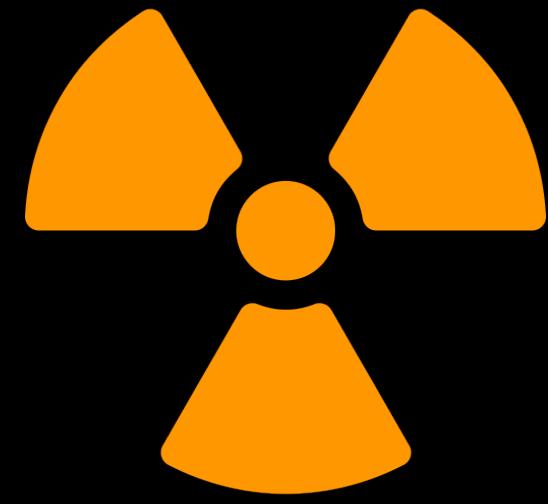
CXR

abd CT



peripancreatic edema

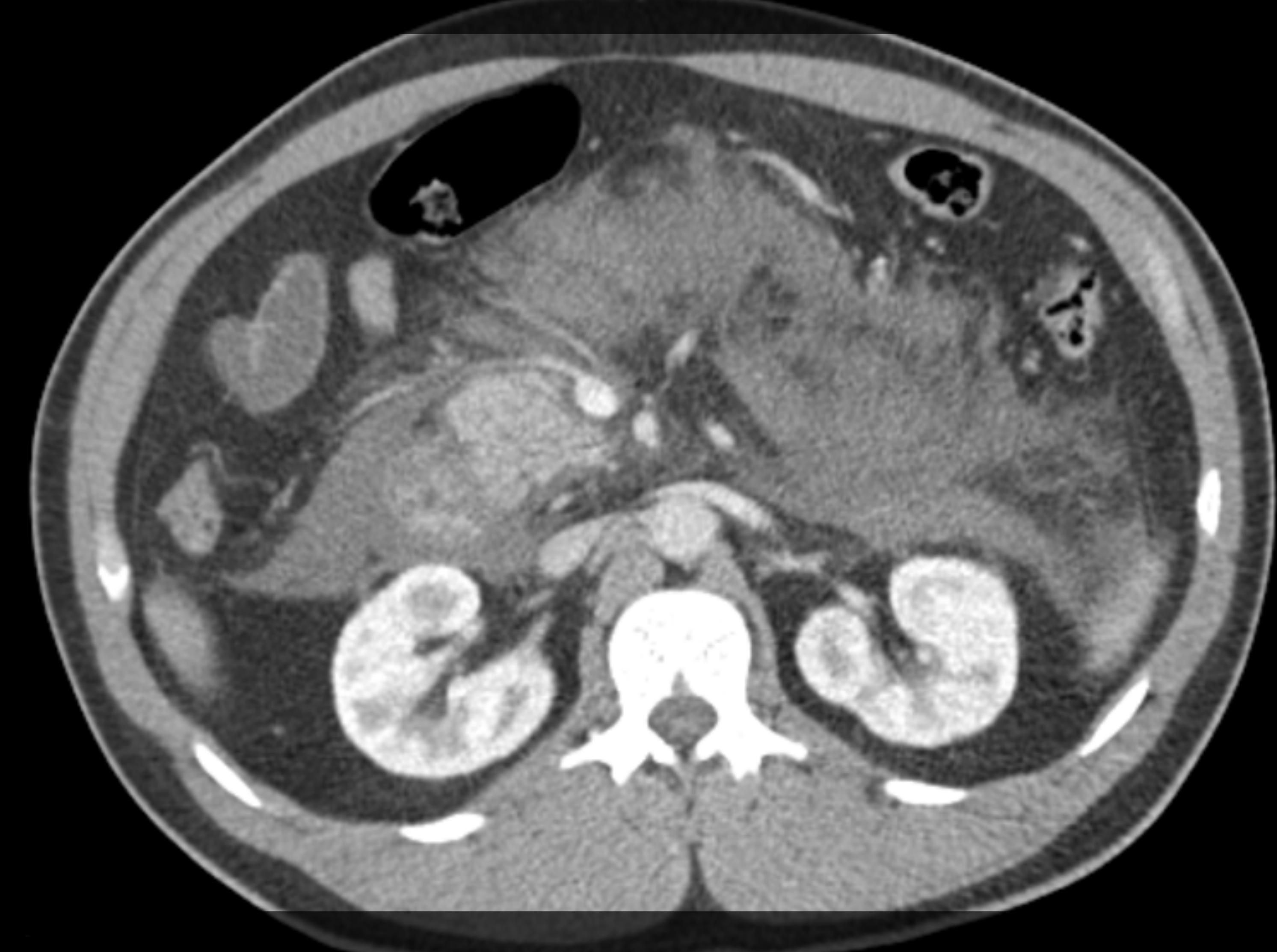
enlarged pancreas, homogenous



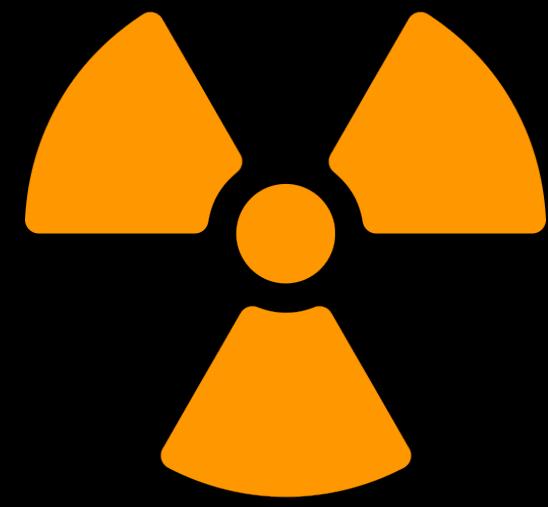
CXR

abd CT

peripancreatic edema



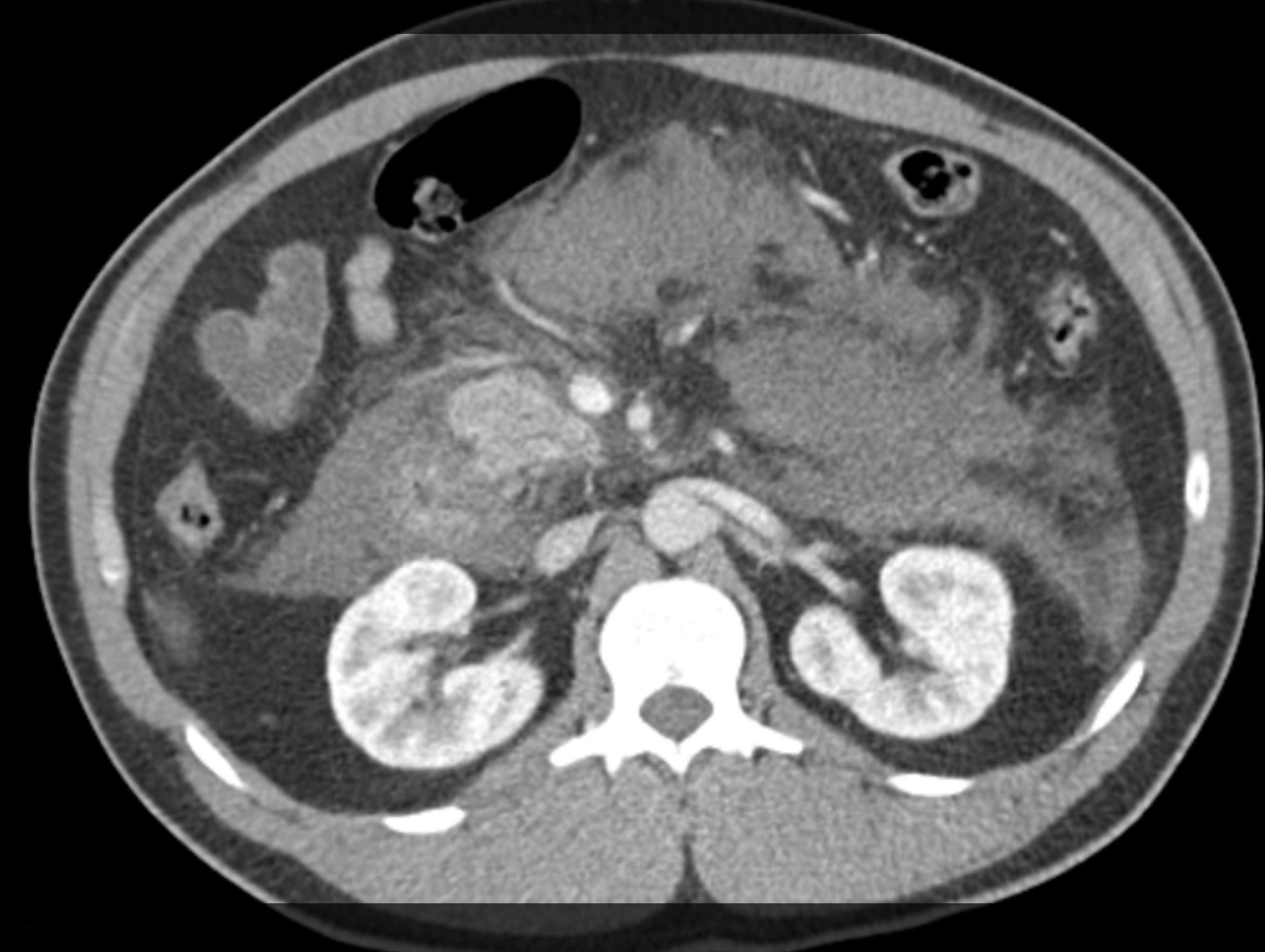
enlarged pancreas, homogenous



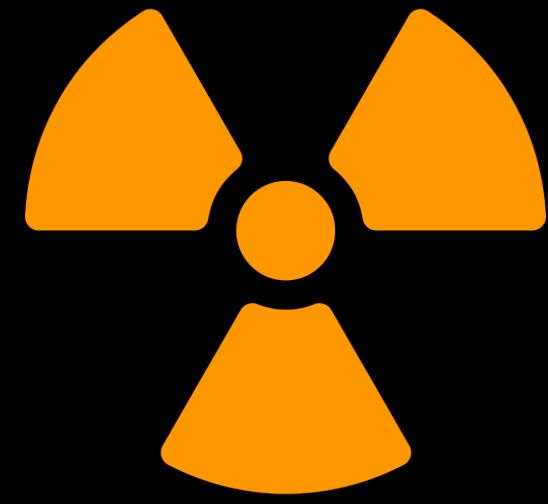
CXR

abd CT

peripancreatic edema

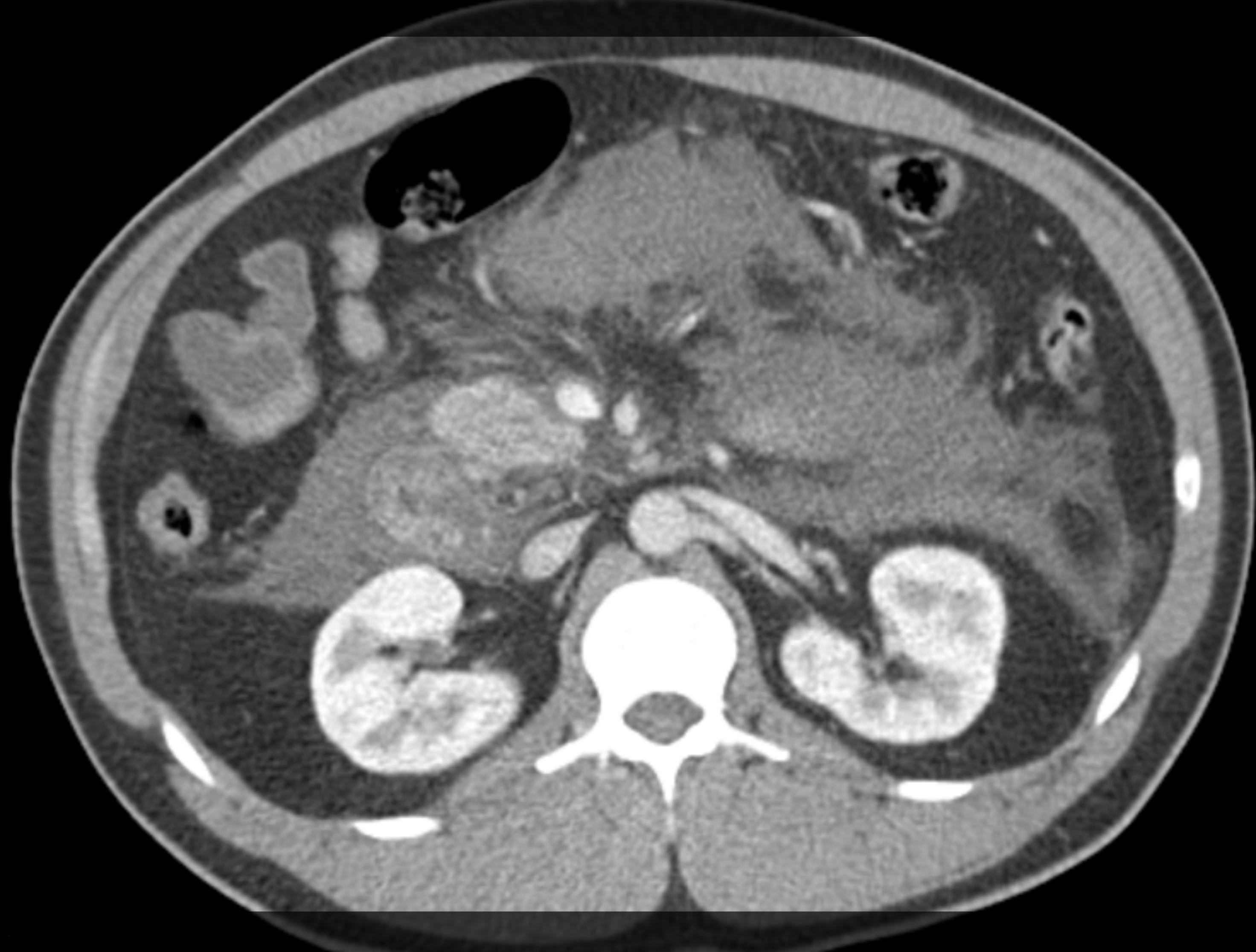


enlarged pancreas, homogenous



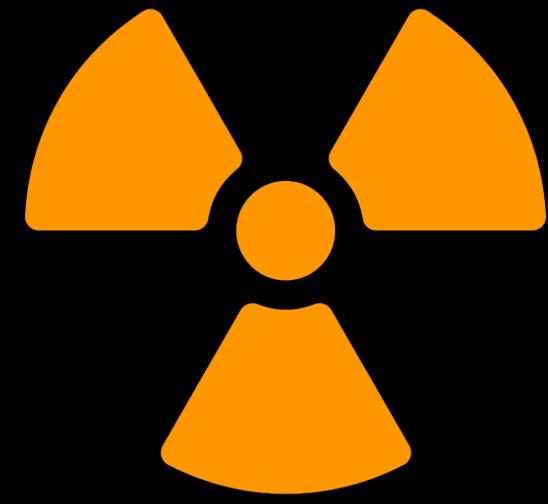
CXR

abd CT



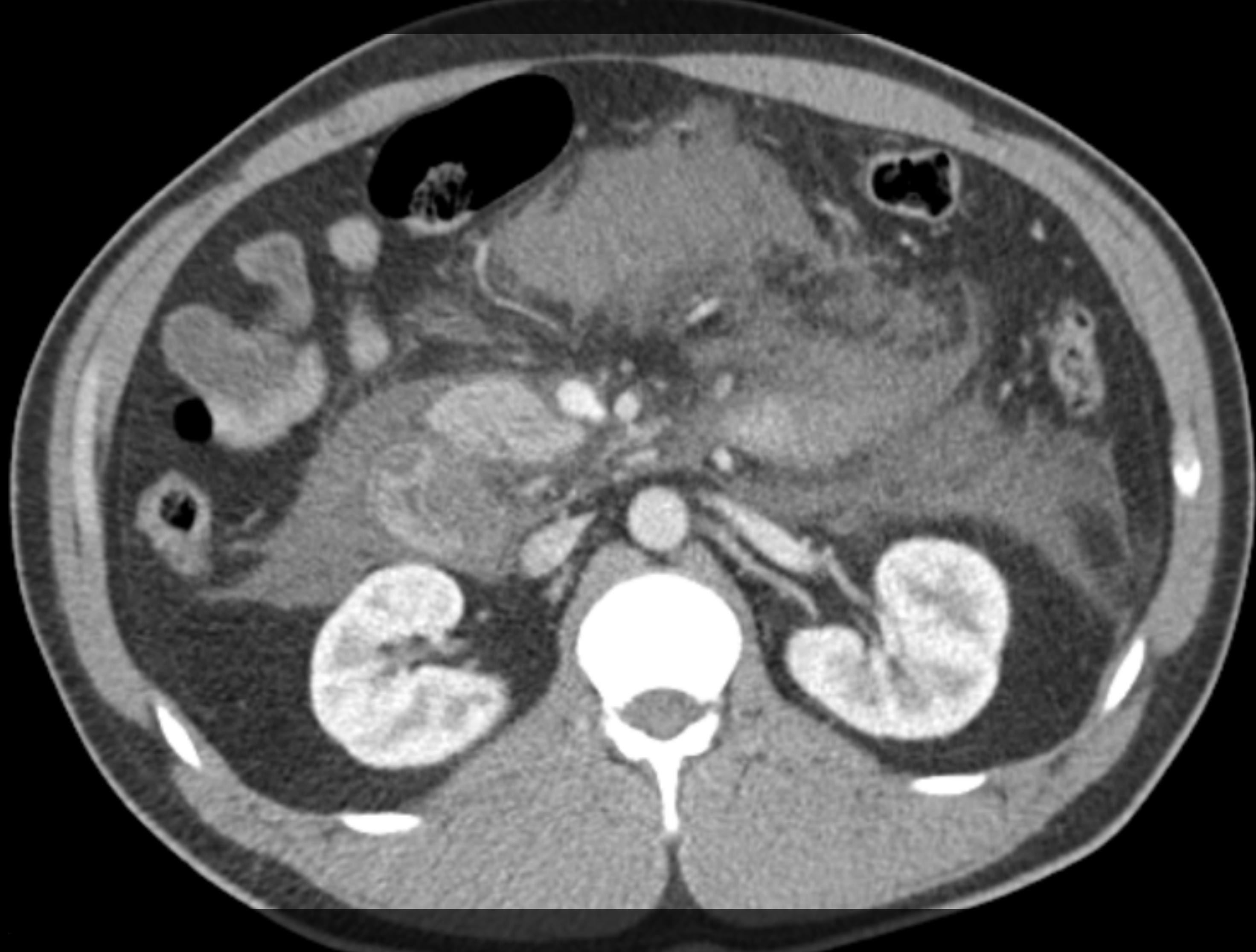
peripancreatic edema

enlarged pancreas, homogenous

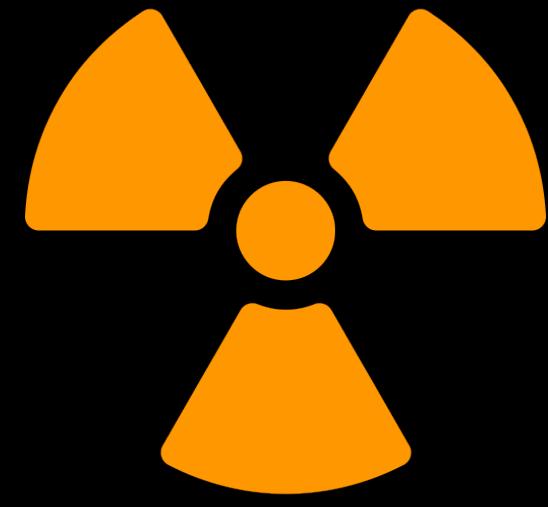


CXR

abd CT



peripancreatic edema
enlarged pancreas, homogenous



CXR

abd CT

no pneumoperitoneum

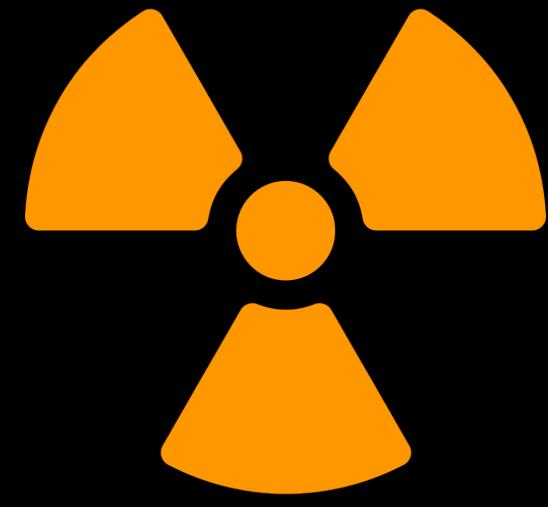
GB nl

appendix not visualized

no RLQ inflammation



enlarged pancreas, homogenous



CXR

abd CT

no pneumoperitoneum

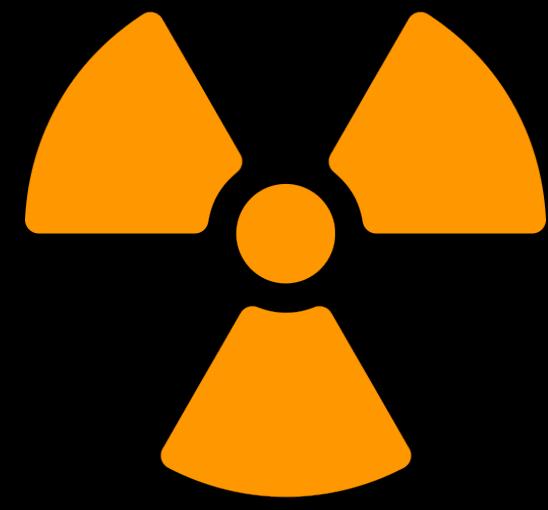
GB nl

appendix not visualized

no RLQ inflammation



enlarged pancreas, homogenous



CXR

abd CT

no pneumoperitoneum

GB nl

appendix not visualized

no RLQ inflammation



enlarged pancreas, homogenous



diffuse abdominal pain

nausea with emesis*2, nbnb

109 bpm

distended abdomen

epigastric tenderness

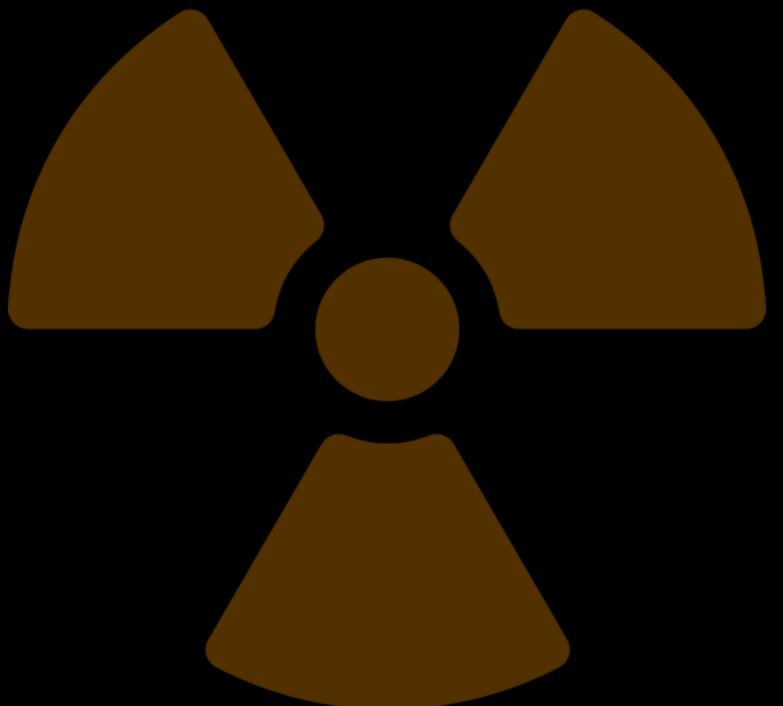
hypoactive BS

35M

HTN

HLD

T2DM





diffuse abdominal pain

nausea with emesis*2, nbnb

**109 bpm distended abdomen
epigastric tenderness
hypoactive BS**

35M

HTN

HLD

T2DM



WBC 13.79

Lipase 6910

TG 7347

Glc 438

Cr 1.4 (bl 0.9)

Ca 7.8





diffuse abdominal pain

nausea with emesis*2, nbnb

109 bpm distended abdomen
epigastric tenderness
hypoactive BS

35M

HTN

HLD

T2DM



WBC 13.79

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Cr 1.4 (bl 0.9)

Ca 7.8



enlarged
pancreas

peri-
pancreatic
inflammation

**acute hypertriglyceridemic pancreatitis
with AKI ($0.9 \rightarrow 1.4$)**

35M

HTN

HLD

T2DM

diffuse abdominal pain

nausea with emesis*2, nbnb

109 bpm distended abdomen

epigastric tenderness

hyporeactive BS

with hyperglycemia (438)

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TG 7347

Glc 438

Cr 1.4 (bl 0.9)

Ca 7.8

enlarged
pancreas

peri-
pancreatic
inflammation

acute hypertriglyceridemic pancreatitis

Emergent TG-lowering therapy for HTGP

Hepatogastroenterology. 2015 Mar-Apr;62(138):429-34.

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Emergent TG-lowering therapy for HTGP

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acute pancreatitis



acute pancreatitis



alcoholic



gallstones



acute pancreatitis



10% HTG



alcoholic



acute pancreatitis



10% HTG

high severity
complications



alcoholic



10% HTG

high severity
complications

Goyal et al. 2016

Table 2: Severity of pancreatitis as per modified Atlanta Criteria

Severity of pancreatitis	HTGP (N = 30)	AAP (N = 147)	P value*
Mild acute pancreatitis	3 (10.00)	55 (37.41)	<0.0001
Moderately severe pancreatitis	14 (46.67)	79 (53.74)	
Severe acute pancreatitis	13 (43.33)	13 (8.84)	



10% HTG

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complications

Goyal et al. 2016

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Severe acute pancreatitis	13 (43.33)	13 (8.84)	

43.33% vs 8.84%



10% HTG

high severity complications

Goyal et al. 2016

Variable	HTGP (N = 30) N (%)	AAP (N = 147) N (%)	P value*
Hospital stay			0.043
<4 days	7 (23.33)	64 (43.54)	
≥4 days	23 (76.67)	83 (56.46)	
ICU care			0.00030
Yes	10 (33.33)	10 (6.80)	
No	20 (66.67)	137 (93.20)	
Surgical intervention			0.016
Yes	6 (20.00)	8 (5.44)	
No	24 (80.00)	139 (94.56)	
Mortality			0.31
Yes	1 (3.33)	1 (0.68)	
No	29 (96.67)	146 (99.32)	
Charges**			0.00030
Median (IQR)	\$28320.50 (19502.00-50673.00)	\$19304.00 (14234.00- 29448.00)	
Charlson comorbidity index			0.30
0	27 (90.00)	117 (79.59)	
1,2,3	3 (10.00)	30 (20.41)	



10% HTG

high severity complications

Goyal et al. 2016

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>4 days 76.67% vs 54.46%



10% HTG

high severity complications

Goyal et al. 2016

Table 4: Outcome comparisons between the patients with HTGP and AAP			
Variable	HTGP (N = 30) N (%)	AAP (N = 147) N (%)	P value*
Hospital stay			
<4 days	7 (23.33)	6 (4.13)	
≥4 days	23 (76.67)	83 (56.46)	0.043
ICU care			
Yes	10 (33.33)	10 (6.80)	
No	20 (66.67)	137 (93.20)	0.00030
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Charlson comorbidity index			
0	27 (90.00)	117 (79.59)	0.30
1,2,3	3 (10.00)	30 (20.41)	

>4 days 76.67% vs 54.46%

ICU care 33.33% vs 6.08%



10% HTG

high severity complications

Goyal et al. 2016

Variable	HTGP (N = 30) N (%)	AAP (N = 147) N (%)	P value*
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10% HTG

high severity complications

Goyal et al. 2016

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Charlson comorbidity index			0.30
0	27 (90.00)	117 (79.59)	
1,2,3	3 (10.00)	30 (20.41)	

surgery 20.00% vs 5.44%



HTGP



**high severity
complications**



HTGP



high severity
complications

Tsuang et al. 2009

rapid reduction
 $\text{TG} < 500\text{mg/dL}$



HTGP



high severity
complications

Tsuang et al. 2009

rapid reduction

TG < 500mg/dL

Etiology

Emergent therapy of HTGP

Emergent TG-lowering therapy for HTGP

Hepatogastroenterology. 2015 Mar-Apr;62(138):429-34.

Introduction

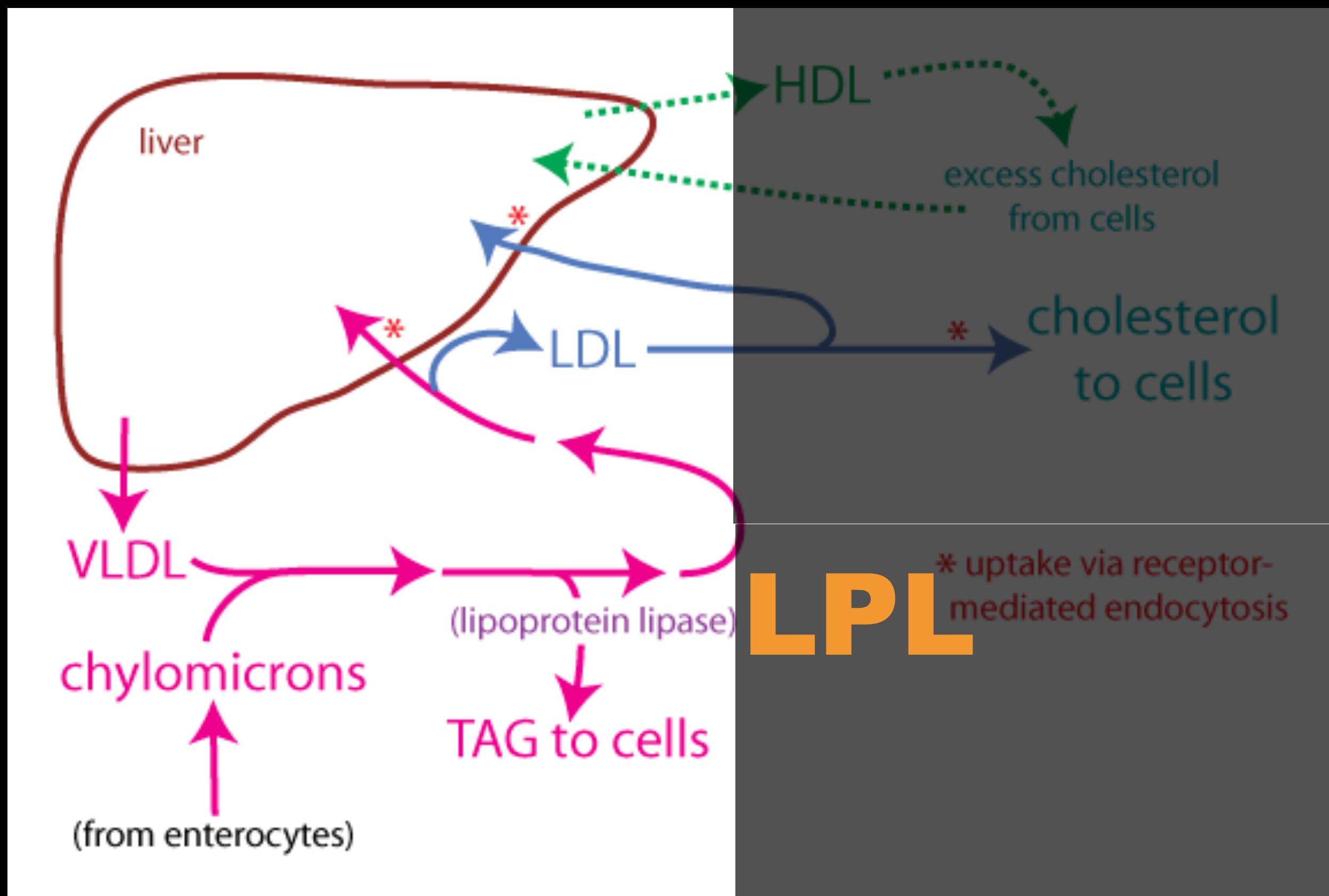
Etiology

Emergent therapy of HTGP

Conclusion

Etiology

hypertriglyceridemia



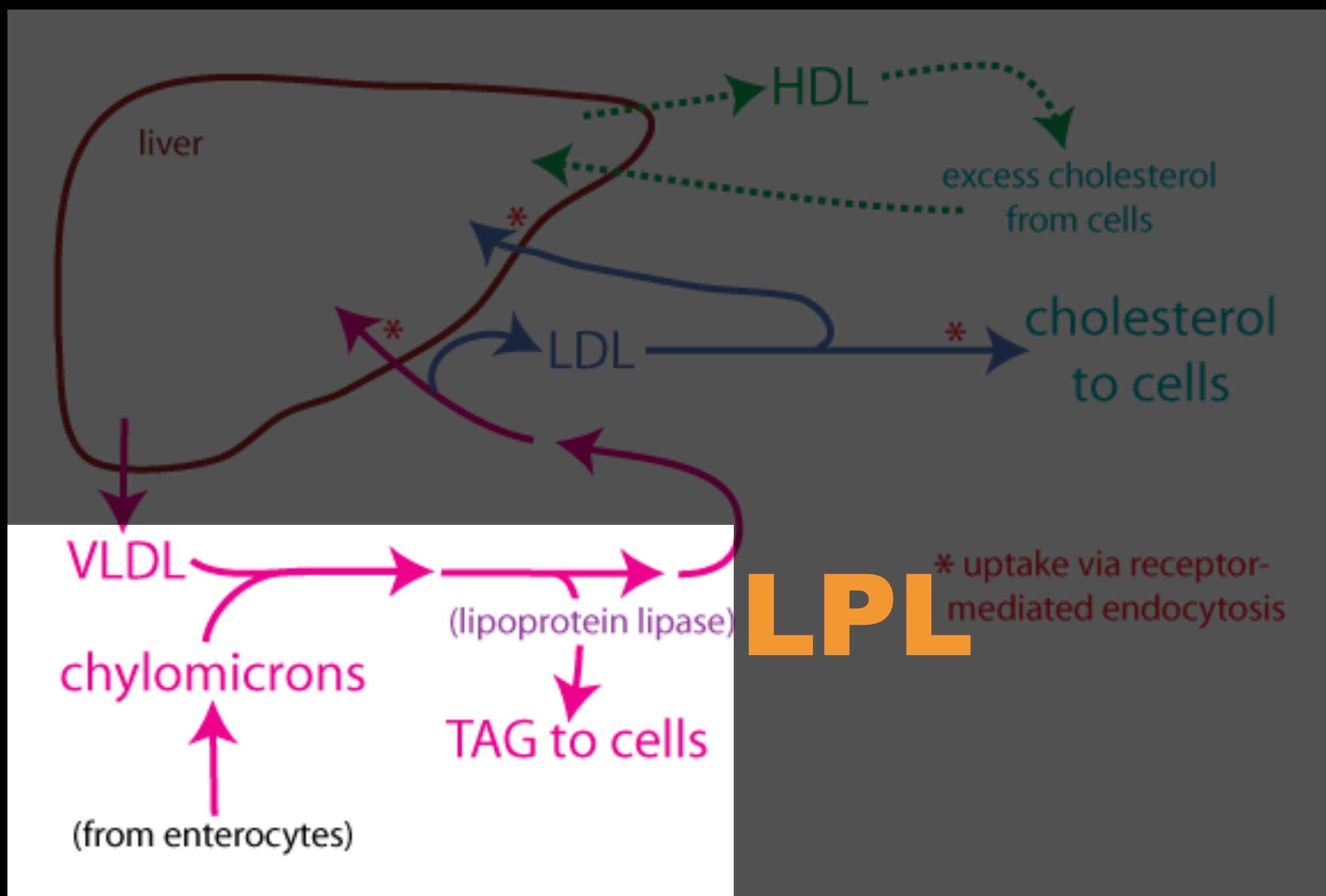
↑ of VLDL

↓ clearance of VLDL/ chylomicron

Etiology

hypertriglyceridemia

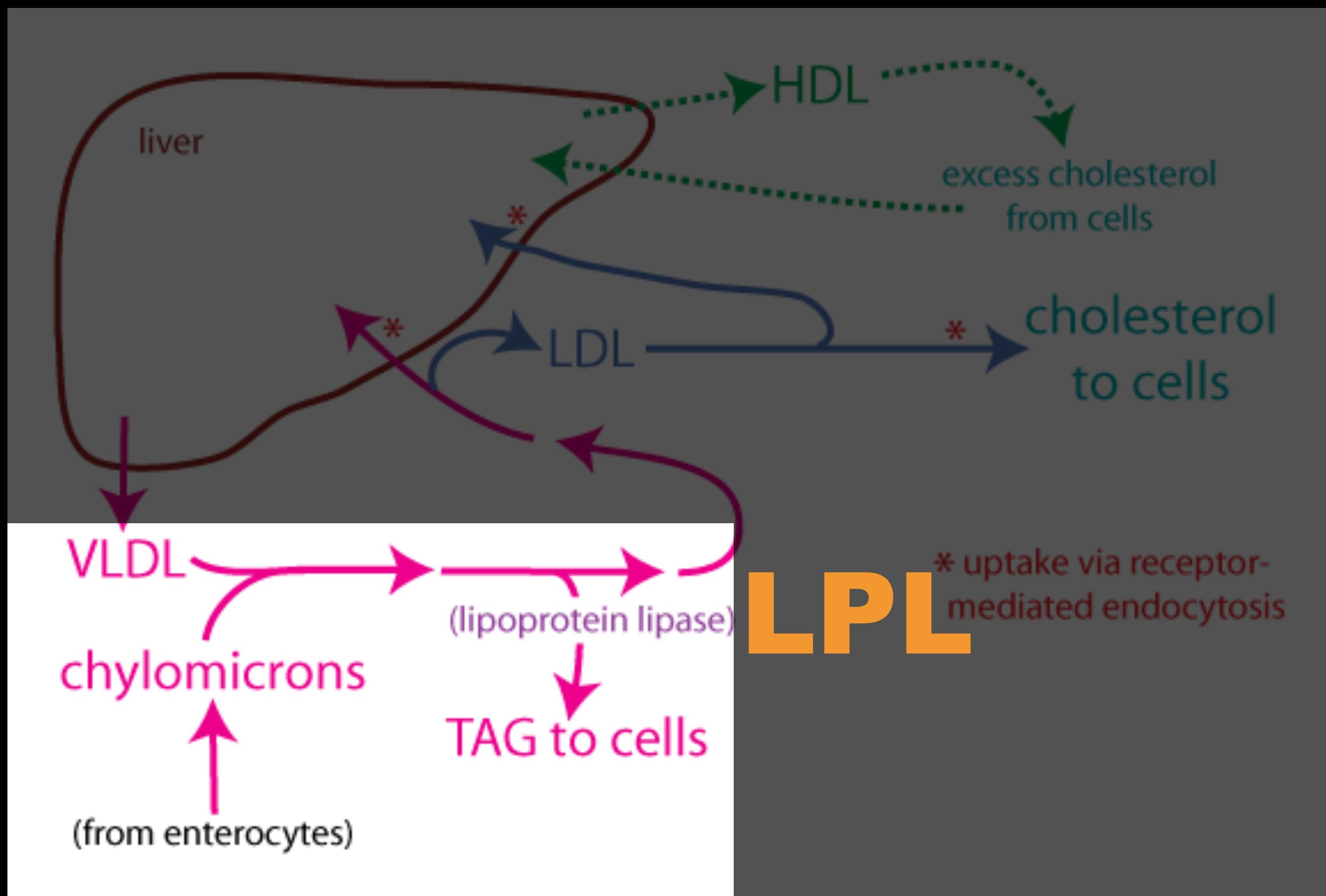
primary <5%



secondary

Etiology

hypertriglyceridemia



primary <5%

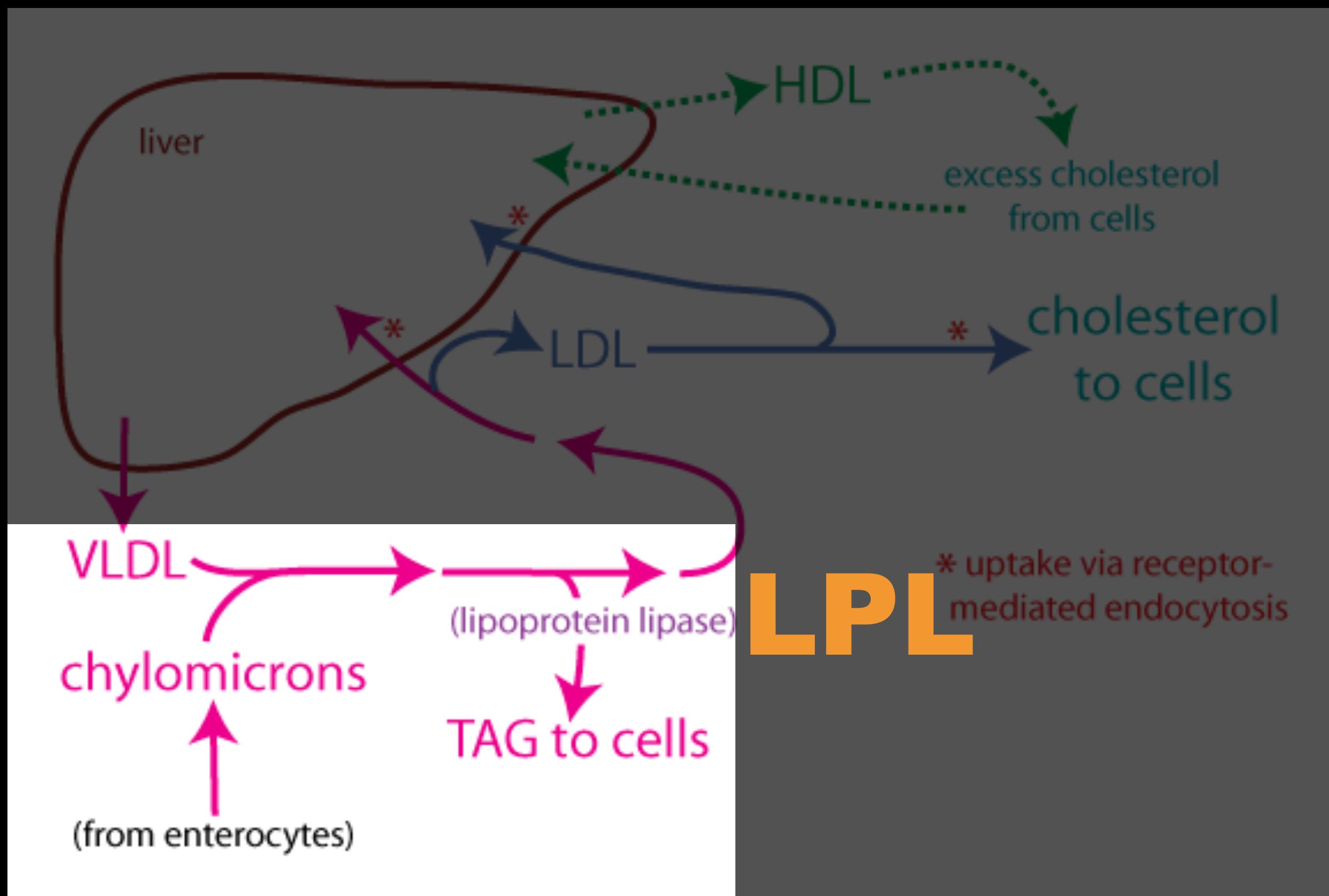
LPL def.

ApoC-II def.

secondary

Etiology

hypertriglyceridemia



primary <5%

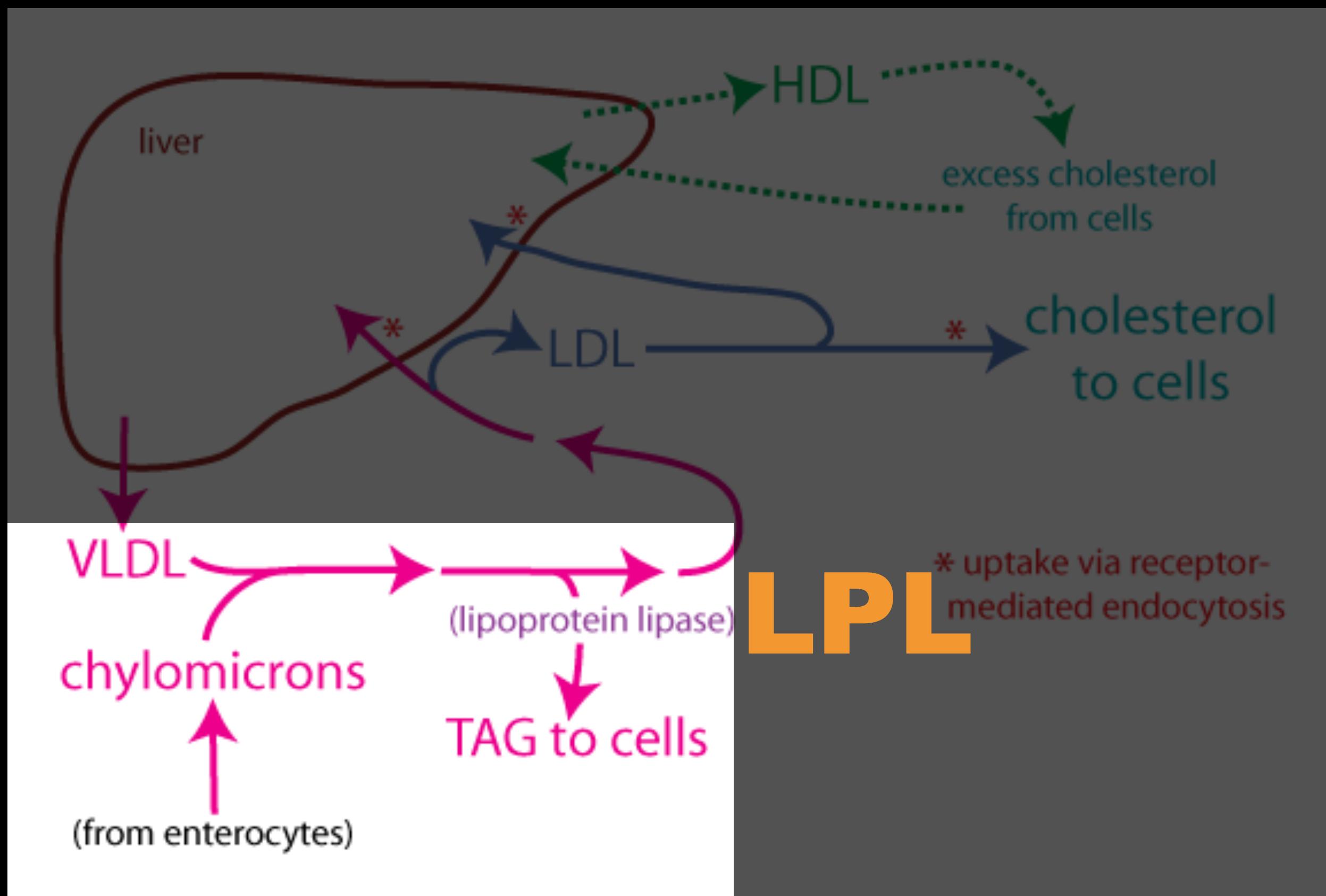
LPL def.

ApoC-II def.

secondary

Etiology

hypertriglyceridemia



primary <5%

LPL def.

ApoC-II def.

secondary

obesity

DM

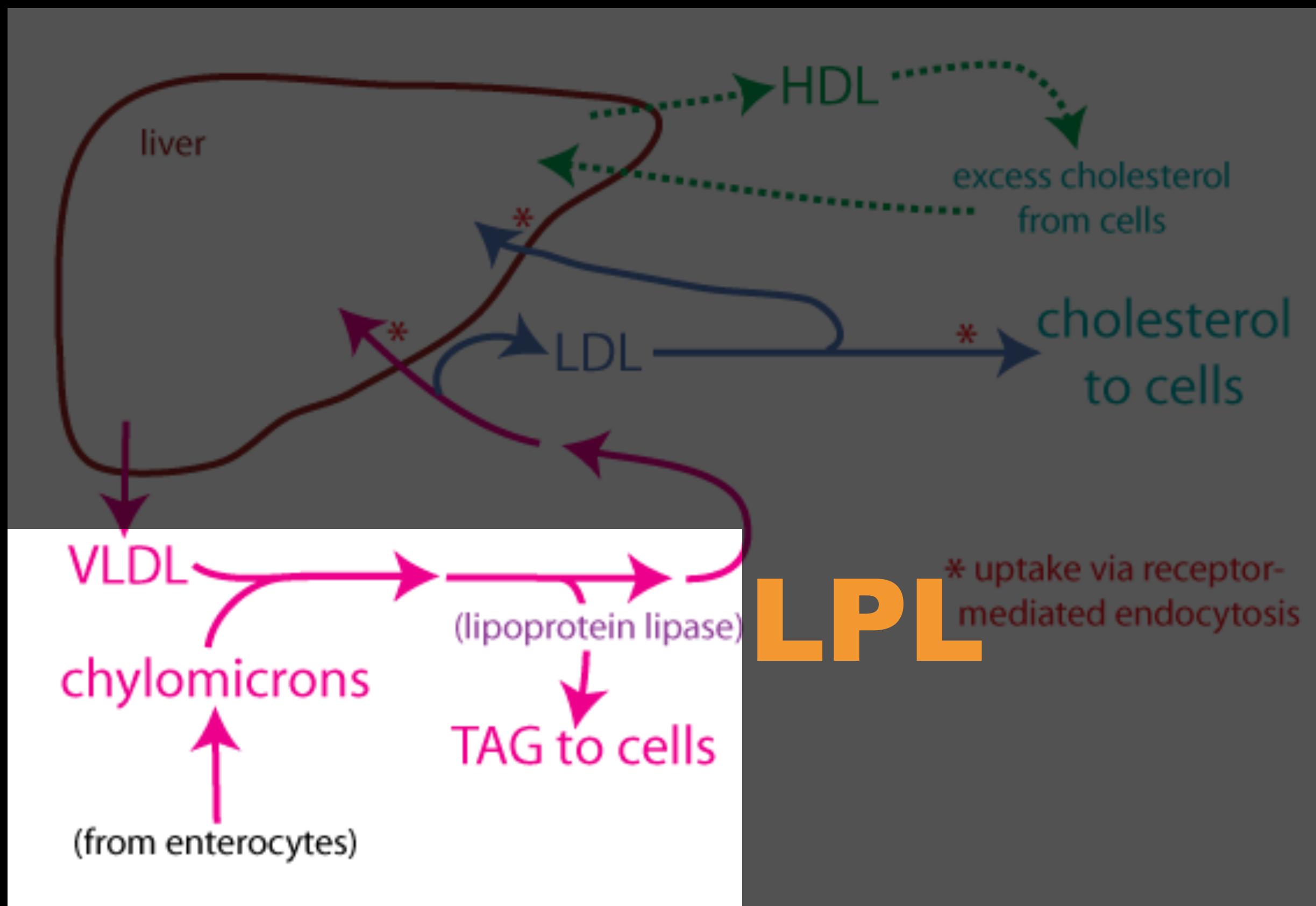
alcohol

pregnancy

medication

Etiology

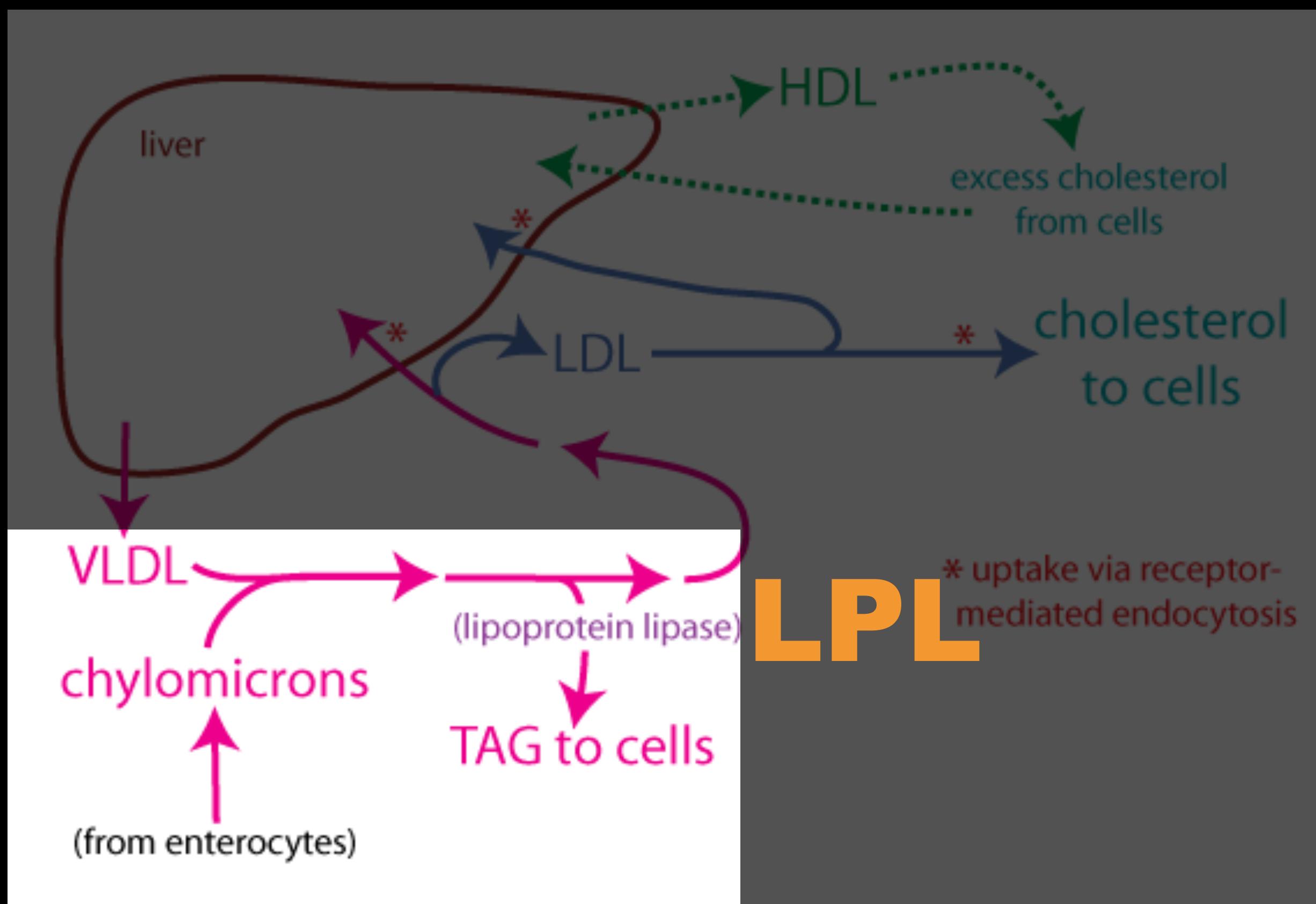
hypertriglyceridemia



secondary
obesity
DM
alcohol
pregnancy
medication
insulin
resistance

Etiology

hypertriglyceridemia



secondary
obesity
DM
alcohol
insulin
resistance

VLDL ↑
LPL ↓

Emergent TG-lowering therapy for HTGP

Hepatogastroenterology. 2015 Mar-Apr;62(138):429-34.

Introduction

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Emergent therapy of HTGP

Conclusion

Emergent therapy of HTGP

**target TG < 500 mg/dL
within 48 hours**

Emergent therapy of HTGP

**target TG < 500 mg/dL
within 48 hours**



diet restrictions



**fibrates
omega-3**

Emergent therapy of HTGP

**target TG < 500 mg/dL
within 48 hours**



diet restrictions

**not effective
slow onset**



**fibrates
omega-3**

Choice	Pros	Cons	Recommend
Insulin	non-invasive economic and safe DM pts	limited efficiency	IV insulin, monitor Glc
Insulin + heparin	non-invasive economic and safe	depletion of LPL	SubQ LMWH < 3 days
TPE	directly and rapidly	invasive expensive infection, allergic	for poor respond
DF	selective removal TGs	invasive expensive filter saturation	for poor respond
MCTs	non-invasive economic and safe	relatively weak	combination use

Choice	Pros	Cons	Recommend
Insulin	non-invasive economic and safe DM pts	limited efficiency	IV insulin, monitor Glc
Insulin + heparin <i>Anderson et al. 2011</i>	non-invasive economic and safe	depletion of LPL	SubQ LMWH < 3 days
TPE	TG < 500	invasive expensive infection, allergic	for poor respond
DF	26% in day3 selective removal TGs 79% in day5	invasive expensive filter saturation	for poor respond
MCTs	non-invasive economic and safe	relatively weak	combination use

Choice	Pros	Cons	Recommend
Insulin	non-invasive economic and safe DM pts	limited efficiency	IV insulin, monitor Glc
Insulin + heparin	non-invasive economic and safe	depletion of LPL	SubQ LMWH < 3 days
<i>Lu et al. 2009, 2010</i>	directly and rapidly	invasive expensive infection, allergic	for poor respond
MCTs	non-invasive economic and safe	relatively weak	combination use

Choice	Pros	Cons	Recommend
Insulin	non-invasive economic and safe DM pts	limited efficiency	IV insulin, monitor Glc
Insulin + heparin	non-invasive economic and safe	depletion of LPL	SubQ LMWH < 3 days

Lu et al. 2009, 2010

IV insulin 0.1-0.3 U/kg/hr, Glc 140-180

SubQ LMWH 5000U Q12H, < 3days

lower complications
mortality, mean hospital stay

Choice	Pros	Cons	Recommend
<i>Bettering et al. 1978</i>	non-invasive economic and safe	limited efficiency	IV insulin, monitor Glc
TG 7120 → normal level in 2.5 hrs <small>DM pts</small>			
Insulin + heparin	non-invasive economic and safe	depletion of LPL	SubQ LMWH < 3 days
TPE	directly and rapidly	invasive expensive infection, allergic	for poor respond
DF	selective removal TGs	invasive expensive filter saturation	for poor respond
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Choice	Pros	Cons	Recommend
<i>Bettering et al. 1978</i>	non-invasive economic and safe	limited efficiency	IV insulin, monitor Glc
TG 7120 → normal level in 2.5 hrs	DM pts		
Insulin + heparin	non-invasive economic and safe	depletion of LPL	SubQ LMWH < 3 days
TPE	directly and rapidly	invasive expensive infection, allergic	for poor respond
<i>Yeh et al. 2003</i>	DE selective Removal TGs filter saturation	invasive	for poor respond
MCTs	non-invasive economic and safe	relatively weak	combination use

TPE

directly and rapidly

invasive
expensive
infection, allergic

for poor respond

Yeh et al. 2003

decrease TG up to **66.3%**

Syed et al. 2010

decrease TG up to **70.4%**

uncertain mobility
length of stay

TPE

directly and rapidly

invasive
expensive
infection, allergic

for poor respond

DF

selective removal TGs

invasive
expensive

for poor respond

MCTs

Gubensek et al. 2009, a retrospective cohort

non-invasive
economic and safe

relatively weak

combination use

Timing ?

decrease TG up to 80%
decrease mortality 15%

TPE

directly and rapidly

invasive
expensive
infection, allergic

for poor respond

DF

selective removal TGs

invasive
expensive

for poor respond

MCTs

non-invasive
economic and safe

Infection
allergic reaction
expensive

combination use

Choice	Pros	Cons	Recommend
Zhang et al. 2008	non-invasive economic and safe DM pts	depletion of LPL	IV insulin, monitor Glc
Insulin + heparin	non-invasive economic and safe	invasive expensive infection, allergic	SubQ LMWH < 3 days
TPE	directly and rapidly	invasive expensive filter saturation	for poor respond
DF	selective removal TGs no need for plasma	invasive expensive filter saturation	for poor respond
MCTs	non-invasive economic and safe	relatively weak	combination use

Choice	Pros	Cons	Recommend
Zhang et al. 2008 Insulin + heparin Yeh et al. 2003; Giannini et al. 2005	non-invasive economical DM pts TPE filter saturation	depletion of LPL expensive infection, allergic	IV insulin, monitor Glc SubQ LMWH < 3 days for poor respond
DF	selective removal TGs no need for plasma	invasive expensive filter saturation	for poor respond
MCTs	non-invasive economic and safe	relatively weak	combination use

Choice	Pros	Cons	Recommend
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Conclusion

acute hypertriglyceridemic pancreatitis

target TG < 500 mg/dL

within 48 hours

Conclusion

acute hypertriglyceridemic pancreatitis

target TG < 500 mg/dL

within 48 hours



diet



fibrate
omega-3

Conclusion

acute hypertriglyceridemic pancreatitis

target TG < 500 mg/dL

within 48 hours



diet



fibrate
omega-3



IV insulin
SubQ heparin

TPE/DF if fails

**acute hypertriglyceridemic pancreatitis
with AKI ($0.9 \rightarrow 1.4$)**

35M

HTN

HLD

T2DM

diffuse abdominal pain

nausea with emesis*2, nbnb

109 bpm distended abdomen

epigastric tenderness

hyporeactive BS

with hyperglycemia (438)

Lipase 6910

TG 7347

Glc 438

Cr 1.4 (bl 0.9)

Ca 7.8

enlarged
pancreas

peri-
pancreatic
inflammation



- 18:38 **arrived ED**
- 20:09 **NS bolus 1000mL**
odansetron 4 mg, diet NPO
- 21:11 **morphine 2 mg**
- 01:11 **Lipase 6910, NS bolus 1000mL**
- 01:38 **abd-CT acute pancreatitis**
- 04:16 **LR infusion 250 mL/hr**
morphine 4 mg
- 05:00 **TG 7374**
- 05:30 **NovoLog 5U, Glc 514—>464**
- 09:16 **Admit to MICU**

35M

HTN

HLD

T2DM



hypertriglyceridemic pancreatitis

09:16 **Admit to MICU**

Ranson score 2

Q6H BCS, Mg, Phos, Ca

Keep UOP > 0.5cc/hr/kg

Target TG < 500

35M

HTN

HLD

T2DM

morphine 4 mg IV PRN

gemfibrozil 600mg BID

insulin IVD 13.5 U/hr

heparin 5000U SubQ

05/17 05:00

37.2 °C

102 bpm

18 bpm

128/78 mmHg

Sat 97%

WBC	13.79	Na	143	Lactate	2.8
Hgb	18.9	K	4.9	LDH	403
Hct	43.1	Cl	82	Lipase	6,910
Plt	201	Bicarb	22	T-Bil	0.6
		BUN	18	AST	40
TC	995	Cr	1.4	ALT	45
TG	7347	Glc	438	Ca	7.8

05/17 14:08

38 °C

133 bpm

33 bpm

105/79 mmHg

Sat 97% on 6L/min TG

WBC	12.49	Na	136	Lactate	2.5
Hgb	12.8	K	4.4	LDH	-
Hct	34.2	Cl	94	Lipase	-
Plt	265	Bicarb	22	T-Bil	0.6
		BUN	26	AST	40
TC	-	Cr	1.9	ALT	45
	630	Glc	305	Ca	6.3

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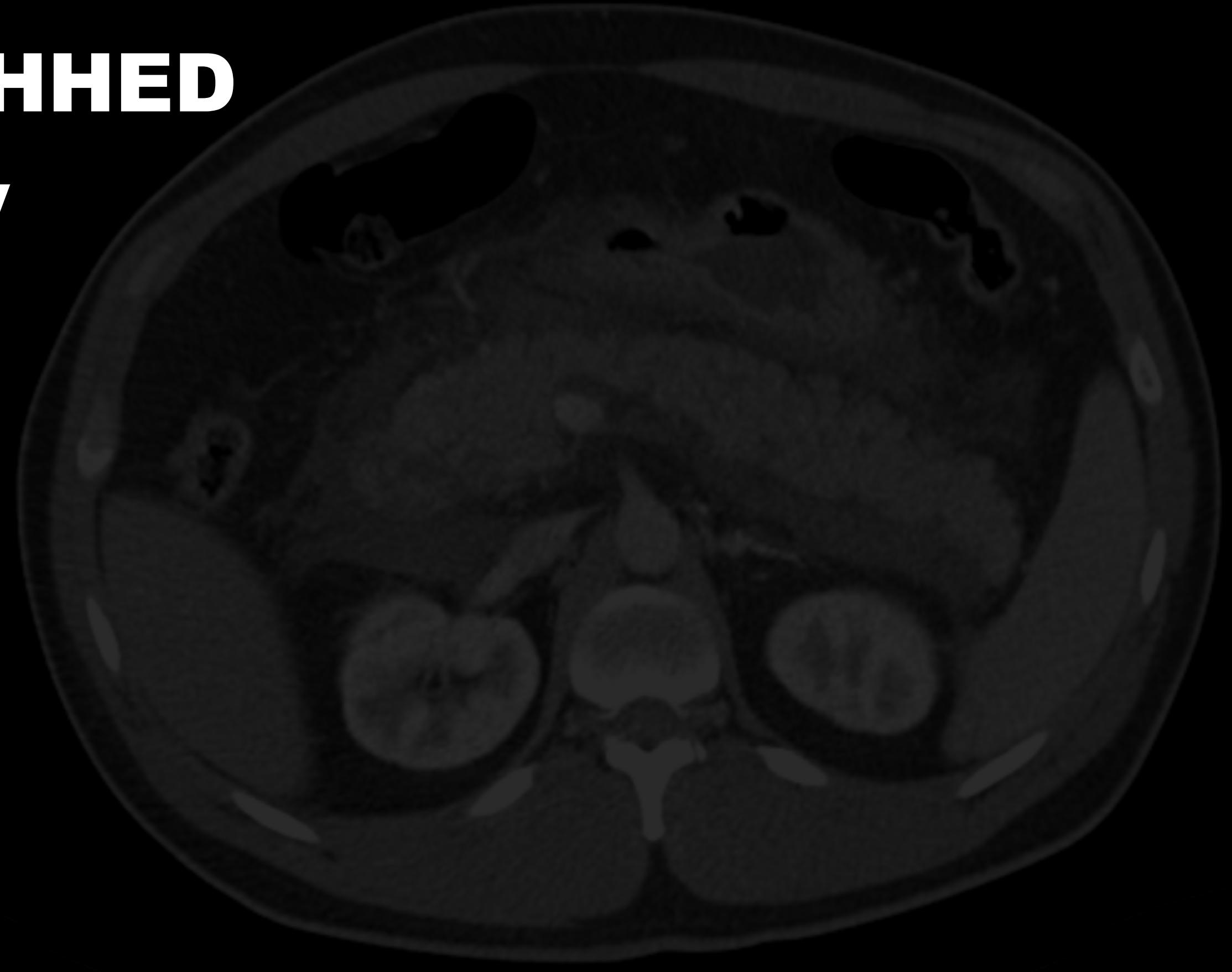
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Shih-Peng Liu, Sub-intern in JHHED

National Yang-Ming University

Thanks for your attention



**Emergent
TG-lowering therapy
for HTGP**

Hepatogastroenterology. 2015 Mar-Apr;62(138):429-34.