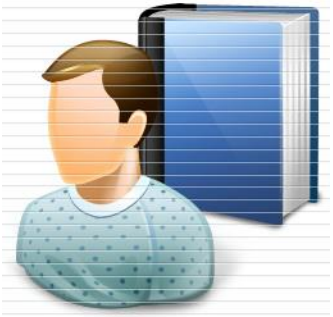
Case report study of patient with type 2 DM and chronic virus hepatitis C

Natia Vashakmadze,

Endocrinologist, MD

Tbilisi, Georgia

Patient History



- 44 years old male was diagnosed with diabetes Mellitus type 2 and Chronic virus hepatitis C coincidentally during screening tests in 2011 year.
- On first visit: No history of Hypertension, dyslipidemia or obesity
- Poor diet, sedentary lifestyle, Current Smoker (1 package per day)
- Genetic predisposition toward DMT2 – father has DMT2

On first visit: 08/2015

- Weight - 81 kg; height – 1.78 m;
  BMI – 25.5 kg/m2 Abdominal circumference – 95 cm

- T/A 120/80 mmhg; HR – 104’ / per minute
- Pulmo et Cor auscultation is normal
- Deep breath test is negative for autonomic neuropathy

- Pulses on a. dorsalis pedis and a. tibialis posterior are preserved on both feet symmetrically.

- he is able to feel the semmes Weinstein 10 g monofilament sensation on the plantar surface of both feet.
  Temperature sensation and nociception are also preserved.

Laboratory examination – 08/2015

- **Glucose profile:**
- Fasting glucose – 150-170 mg/dl,
- postprandial glucose – 220-250 mg/dl;
- HbA1c – 9.2%

- **Fasting lipids:**
- Tot. cholesterol – 121 mg/dl LDL – 67 mg/dl HDL – 37 mg/dl; TG –85.0 mg/dl

- **Kidney function test**
- Crea – 81.3 μmol/l eGFR -101 ml/min/1.73m2 according to CKD-EPI
- microalbuminuria – 129mg/l (<15 mg/l)

- **Liver function test:**
- ALT - 69 U/l; AST - 39.0 U/l; γ-GT 69 U/l; T. Bil – 5.6 μmol/l; total protein - 69 g/l; Albumin – 51.0 g/l

- **CBC:** Hb – 158 g/l; RBC – 4.9x10*9; PLT - 235.0x10*9; WBC – 9.75x10*9/L; ESR – 13 mm/hr;

- **Urinalysis** – glycosuria, other parameters without changes.

- **Thyroid function:** TSH – 1.5 μmol/L (0.4-4.4)

Instrumental examination: 08/2015

- Resting ECG: sinus tachycardia
- Treadmill stress test: negative for ischemia
- Thyroid ultrasound – total V = 28 m3, no nodules
- Abdominal ultrasound – fatty liver disease; stones in gallbladder;
- Fibro scan – F3 stage
- The fundoscopic examination – no diabetic retinopathy

Association between hepatitis C virus and IR/DM T2

- T2D is a common complication of all liver diseases, independently of the etiology, especially at the advanced stage. However, clinical and experimental data suggest a direct role of HCV in the perturbation of glucose metabolism.

- The Third National Health and Nutrition Examination Survey (NHANES III) showed that among persons ≥40 years of age, those with HCV infection were more than three times more likely than those without HCV infection to have type 2 diabetes.

- Ageing, obesity, family history of diabetes, African-American origin, and HIV coinfection are recognized influencing factors associated with diabetes development among HCV-infected patients.

World J Gastroenterol. 2009 Apr 7; 15(13): 1537–1547.
http://care.diabetesjournals.org/content/29/5/1140.full

Regulation of gluconeogenesis and glycolysis in the HCV-infected cells

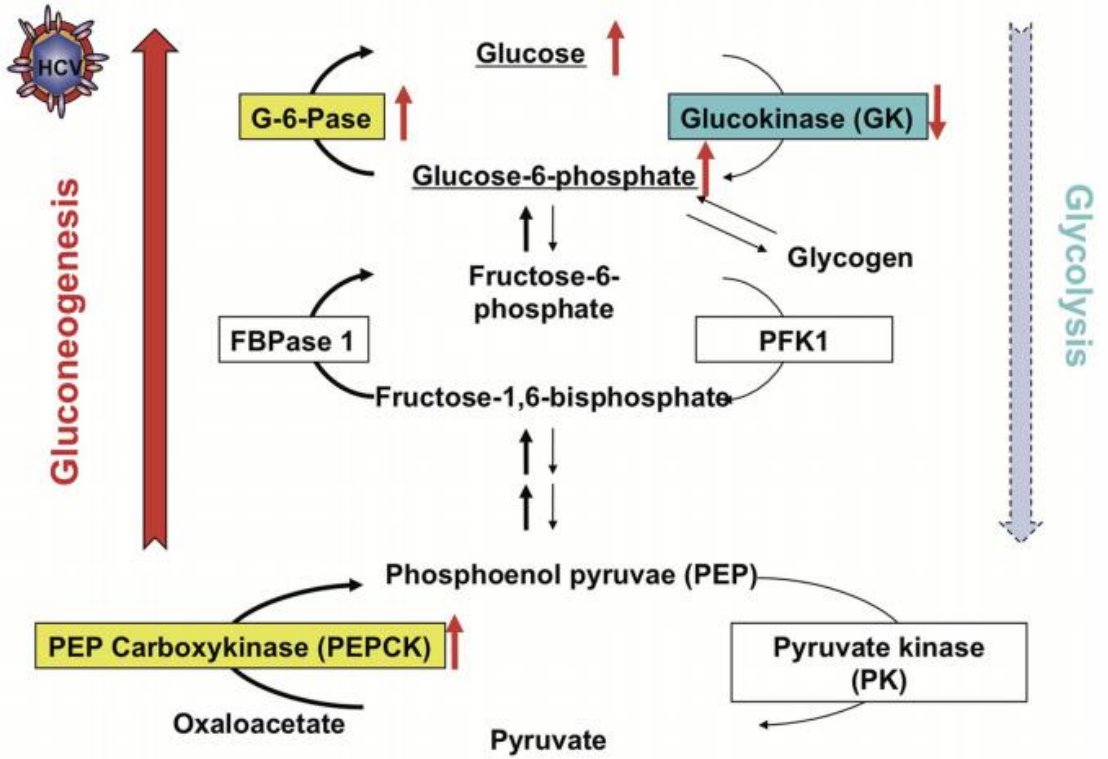
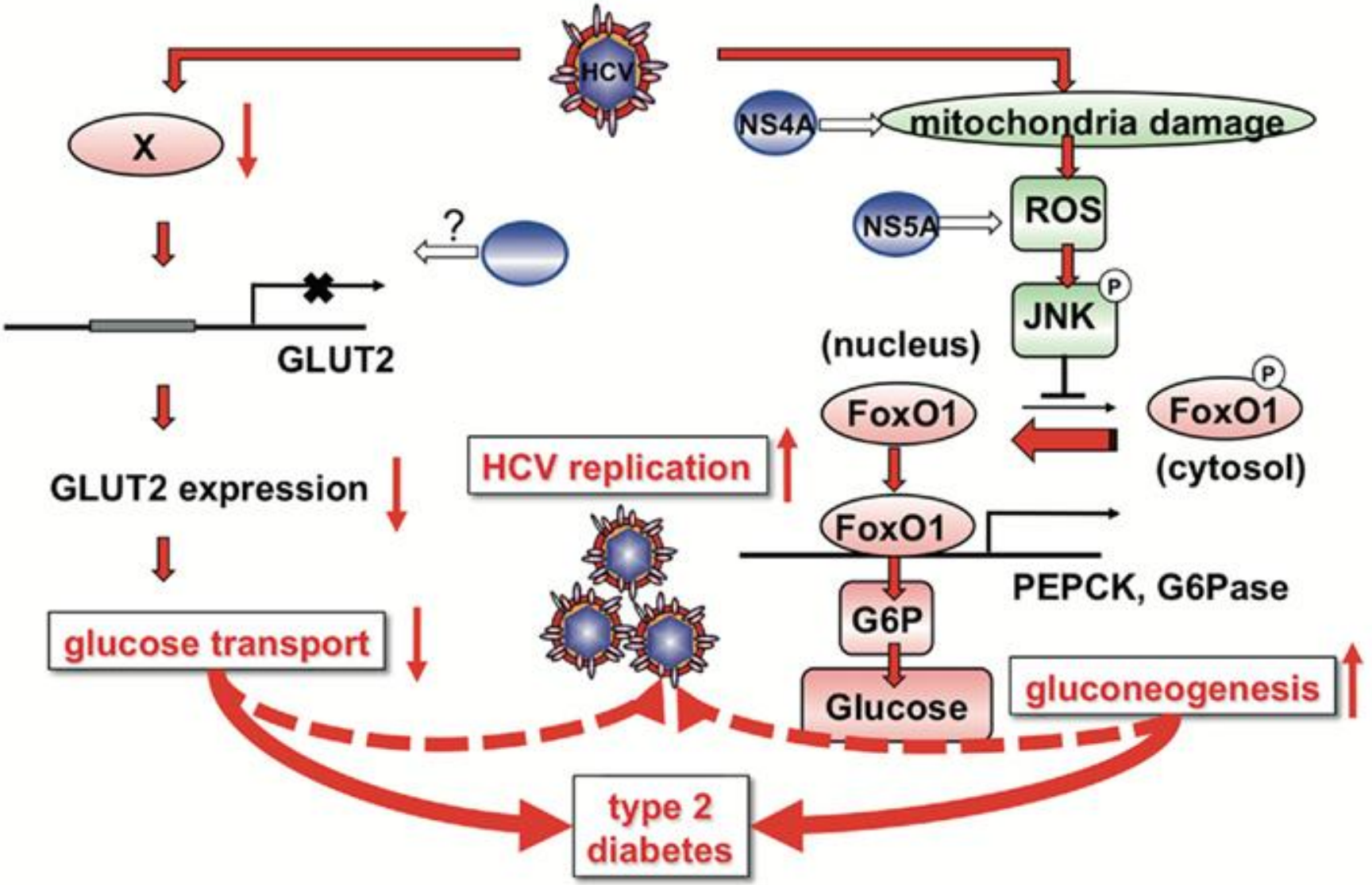


FIGURE 1 | Regulation of Gluconeogenesis and Glycolysis in the HCV-infected cells. HCV infection promotes gluconeogenesis via transcriptional up-regulation of the genes for PEPCK and G6Pase, the rate-limiting enzymes for hepatic gluconeogenesis, and transcriptional down-regulation of the gene for GK, the rate-limiting enzyme for hepatic glycolysis.

Front. Microbiol., 10 January 2012 | http://dx.doi.org/10.3389/fmicb.2011.00278



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Diagnosis

- **Main diagnosis**:

  Diabetes mellitus type 2, target HbA1c $\leq$ 6.5%
- **Complications**:

  Diabetic nephropathy, microalbuminuria, CKD1
- **Co-morbidities:**

  Chronic virus hepatitis C

  Chronic calculous cholecystitis

  Diffuse goiter, Euthyroidism

  Overweight

  Sinus tachycardia

Treatment 08/2015

Previous treatment

- Metformin 1000 mg t.i.d.
- Ins. Glargine 30 U at bedtime
- Repaglinide 2 mg before each main meal

New treatment

- Metformin 1000 mg b.i.d.
- Pioglitazone 45 mg per day
- Liraglutide 1.8 mg at bedtime
- Perindopril 2.5 mg at bedtime
- Statin initiation was deliberately delayed

Outcome 03/2016

- **Body weight** 76 kg (-5kg x 6 month) **BMI – 24** kg/m2
- Abdominal circumference – 92 cm

- **Glucose metabolism:** Fasting glucose – 100-140 mg/dl,
- postprandial glucose – 160-180 mg/dl;
- HbA1c – **9.2%** - baseline - **8.3%** 3 month later - **7.4%** several days ago

- **Fasting lipids:** Tot. cholesterol – 140 mg/dl LDL – 85 mg/dl HDL – 42 mg/dl; TG –120 mg/dl

- **Kidney function tests:** Crea – 69 μmol/l eGFR -110 ml/min/1.73m2 according to CKD-EPI Microalbuminuria – 110 mg/l (<15 mg/l)

- **Liver function test**: ALT - 63 U/l; AST - 27.0 U/l; γ-GT - 59 U/l; T. Bil – 18.8 μmol/l;

- **Thyroid ultrasound** – total volume 28.5 m3; in right lobe 0.5x0.3x0.4 mm and 0.7x0.5x0.6 mm hyperechogenic nodules are visualized and in the left lobe 1.1x0.8x0.9 mm hyperechogenic nodule is visualized, with increased perinodular vascularization
- **TSH** -3.5 μmol/l (0.4-4.4)

08/2016 – 12/2016

- Patient was involved in Hepatitis C elimination program. He was taking following medications: Harvoni and Ribavirini
  According to investigations conducted in 02/2017 hepatitis C was fully eliminated and patient is free of disease

- Generally elimination of HCV infection has dramatic positive influence on glucose metabolism.

  However glucose and lipid metabolism was seriously deteriorated after treatment with new medications.

02/12/2016

- HbA1c - 8,1%

- Fasting lipids:
- Tot. chol – 276 mg/dl, LDL – 184 mg/dl; HDL – 44 mg/dl; Tg – 222 mg/dl;

- Liver function test – without changes

- Kidney function - stable,
- Microalbuminuria – negative - perindopril was stopped

- Statin was added to treatment: atorvastatin 40 mg

18/05/2017

Test	Result	Target
HbA1c	8,01%	The lower the better
Tot. cholesterol	302 mg/dl	
HDL	38,4 mg/dl	>45 mg/dl
LDL	203 mg/dl	<100 mg/dl
Tg	299 mg/dl	<150 mg/dl
Non-HDL	263,6 mg/dl	<130 mg/dl
eGFR	89 ml/min/1.73 m2	>60 ml/min/1.73m2
TSH	1.6 mkIu/ml	0.4-4.4 mkIU/ml

- Current therapy:

Metformin (Siofor) 1000 mg X2
Pioglitazone (Actos) 45 ∂ϑ X 1
Liraglutide (Victoza) 1,8 ∂ϑ
Atorvastatin (Liprimar) 40 mg
Spontaneously Stopped intake 1 month ago

Thyroid US without changes in comparison with 2015 results

Questions for discussion:

- What could be a reason for deterioration of lipid and glucose metabolism after elimination of HCV infection?

- Is SGLT2 inhibitor (dapagliflozin) rational combination with existing therapy? Especially with pioglitazone? (according to potential risk of bladder cancer of both drugs)

- Does this patient need aspirin initiation for primary CVD prevention?