Ministry of Community Safety and Correctional Services

Office of the Regional Supervising Coroner – Eastern Region
366 King Street East, Suite 440
Kingston, ON K7K 6Y3
Telephone: 613-544-1596
Facsimile: 613-544-3473

Ministère de la Sécurité communautaire et des Services correctionnels

Bureau du coroner régional principal – Région de l'Est
366, rue King Est, Bureau 440
Kingston, ON K7K 6Y3
Téléphone: 613-544-1596
Télécopieur : 613-544-3473

July 11, 2017

Mr. Edward Marrocco
Stockwoods LLP Barristers
77 King Street West, Suite 4130
Toronto, ON M5K 2A1

Dear Mr. Marrocco:

Re: Name of Deceased: Faqiri, Soleiman
Date of Death: December 15, 2016
OCC File No: **2016-14763**

As authorized by Mr. Yusuf Faqiri, I have enclosed a copy of our letter and the coroner's reports which I forwarded to him concerning his late brother, Soleiman Faqiri.

If I can be of further assistance, please do not hesitate to contact my office.

Sincerely,

Paul E. Dungey, MD, FRCPC
Regional Supervising Coroner
East Region – Kingston Office

PED/lr

Enclosures: copy of letter to Mr. Yusuf Faqiri
copy of Coroner's Investigation Statement
copy of Post Mortem Examination Report
copy of Toxicology Report & Cardiology Genetics Report (attached to postmortem)

c: *Dr. Eric Ready*

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July 11, 2017

Mr. Yusuf Faqiri
21 Marsh Lane
Ajax, ON L1T 3W2

Dear Mr. Faqiri:

Re: Name of Deceased: Faqiri, Soleiman
Date of Death: December 15, 2016
OCC File No: **2016-14763**

On behalf of the Office of the Chief Coroner and the Ontario Forensic Pathology Service, please accept our condolences on the loss of your *brother* **Soleiman.**

Our investigation, which included a postmortem examination (autopsy), showed that a genetic mutation (change) or condition affecting the heart or blood vessels may have contributed to this death. Since genetic mutations can be passed down ("inherited") from one or both parents, close family members such as brothers, sisters, children or parents may be at risk of developing the same disease.

When a family member has died from one of these conditions, there is good scientific evidence that appropriate screening and testing of close relatives may be worthwhile to determine whether those relatives are at risk. We recommend that you and possibly other immediate family members, with the assistance of your family doctor, consider consulting an appropriate medical specialist (cardiologist, electrophysiologist and/or geneticist) with expertise in this area. The specialist can discuss with you important considerations including the likelihood of risk, the potential for and benefit of testing, and preventative steps to help reduce the possibility of developing the disease or complications. To assist you, attached is a list of the Heart Rhythm Disorder / Hereditary Heart Disease clinics located across Canada with expertise in this field of medicine.

As part of the postmortem examination of **Soleiman Faqiri**, a sample of blood or tissue was processed to obtain purified DNA, which will be stored indefinitely to allow for future genetic testing. If a cardiologist or geneticist requests this DNA to test for specific genetic mutations, we will make the DNA available to them for testing with the consent of the family. A copy of the Report of Post Mortem Examination is enclosed.

We encourage you to share the contents of this letter with your immediate family members by:

- Showing or providing them a copy;
- Providing our contact information so they can contact our office directly; or
- Providing our office with their names and contact information so that we can contact them directly.

I have also enclosed copies of reports referenced below, to which you are entitled under Section 18(4) of the *Coroners Act.*

The *Coroner's Investigation Statement* is submitted electronically and therefore does not include the investigating coroner's signature. The *Coroner's Investigation Statement* is released under my authority after my review for completeness and accuracy.

If you believe that the information documented in the enclosed reports is factually incorrect, please contact my office. If you have any questions about the investigation, you may contact the investigating coroner, Dr. Eric Ready at 705-324-3221. Your family doctor may also be able to assist you in understanding medical terms utilized.

Please contact me if you have any questions or require further information in this regard.

Sincerely,

Paul E. Dungey, MD, FRCPC
Regional Supervising Coroner
East Region – Kingston Office

PED/lr

Enclosures: copy of Post Mortem Report
copy of Coroner's Investigation Statement
copy of Toxicology Report & Cardiology Genetics Report (attached to postmortem)

copy: *Dr. Eric Ready*

Heart Rhythm Disorder / Hereditary Heart Disease Clinics across Canada

Hospital	Telephone	Fax
BC Inherited Arrhythmia Clinic – Victoria Site	250-595-1551	250-595-1551
BC Inherited Arrhythmia Clinic – Vancouver Site*	604-682-2344 x 63260 604-875-2836	604-806-8723 604-875-3454
BC Children's Hospital (Vancouver)	604-875-3619	604-875-3463
Foothills Hospital (Calgary)*	403-955-7368 403-955-2289 403-220-2656	403-955-2701 403-210-9350
University of Alberta Hospital	780-407-6946 780-407-7333	780-407-6845
London Health Sciences Centre	519-663-3746	519-663-3782
Hamilton Health Sciences Centre	905-577-8004	905-523-9165
Hospital for Sick Children (Sick Kids) – Toronto*	416-813-5850	416-813-5582
Inherited Arrhythmia Clinic - Toronto General Hospital *	416-340-3535 416-340-4282	647-693-7543
St. Michael's Hospital (Toronto)	416-864-5152	416-864-5348
Sunnybrook Health Sciences Centre (Toronto)	416-480-6100 x 7383	416-480-5069
Southlake Regional Health Centre and Credit Valley Hospital	905-895-4521 x 2572 905-813-4104	905-830-5806 905-813-4347
The Inherited Heart Rhythm Clinic – Kingston	613-549-6666 x 3377	613-548-1387
Ottawa Heart Institute Inherited Arrhythmia Clinic	613-761-5016	613-761-5060
Children's Hospital of Eastern Ontario	613-737-7600 x 2804 613-737-2275	613-738-4835 613-738-4822
Montreal Heart Institute	514-593-2498	514-593-2499
Quebec Heart Institute (Quebec City)	418-656-4598 418-656-8711 x 5925/3488	418-656-4574 418-656-4649
QEII Health Sciences Centre (Halifax)	902-473-2187 902-470-8754	902-473-3158 902-470-8709
Health Sciences Centre (St. John's Nfld.)	709-777-4788	709-777-4190

Statement #:	Coroner:	CIS Case #:
2016-2677-B	20332 - DR Ready, Eric	2016-14763

Personal Details of Deceased

Name: Faqiri, Soleiman **Gender:** Male **Date of Birth :** 01/Jan/1986 **Age:** 30 yrs
Address: 152 Atherton AVE
City: AJAX **Province:** ON **Postal Code:** L1S 7N3

Investigation Details

Status: Final **Inquest Required:** No **Death Pronounced:** 15/Dec/2016
By what means: Undetermined **Death Presumed:**

Environments

Environment(1) PRIMARY

Date: 15/Dec/2016
Municipality: LINDSAY
Institution:
Environment: Custody - Provincial Jail / Detention Center
Death Factor: Category Not Ascertained
Address: 541 36 HWY
City: LINDSAY

Involvements

912 Psychiatric Treatment - past / present

Reports Expected

Police: Y Min. of Labour: N
Laboratory: Y Other: N
Fire Marshal: N

Pathologist	Hospital
22062 DR Bellis, Maggie	

Medical cause of death: Unascertained
Due to / as a consequence of:

Contributing Factors: Mental Illness (Schizoaffective Disorder)

Narrative

This case was referred by the Central Dispatch Unit of the Office of the Chief Coroner and Forensic Pathology Service of Ontario. The case had been reported by the Kawartha Lakes Police Services who were attending at a death scene within the Central East Correction Centre in Lindsay. The case was accepted under Section 10 of the Coroners Act.

The Coroner was asked to contact the local Police Service to receive a report about a death occurring in the segregation cells of the Central East Correctional Centre. The Coroner notified the Regional Supervising Coroner of this death immediately after the call from Dispatch and the initial discussion with the Police.

The Coroner attended the Central East Correctional Centre and met with local Police Officers in their offices at this centre.

The initial information provided indicated that a young male inmate had been found dead in his segregation cell after being escorted back to his cell from the shower cell. The death was discovered after an "altercation" had occurred between the inmate and a number of Correctional Officers.

The Central East Correctional Centre supervisory staff provided the Coroner and the Police with some initial documentation about the inmate. This information included demographic information and next-of-kin information and a very limited account of health information. This information indicated that there was a mental health history and that there had been some psychiatric medications being prescribed and administered in 2014. These medications included Seroquel, Lithium and Epival. The Coroner was able to see some video of the minutes preceding the death. This video showed the inmate being escorted to his cell down a cellblock hallway. There was a group of

Statement #:	Coroner:	CIS Case #:
2016-2677-B	20332 - DR Ready, Eric	2016-14763

Correctional Officers surrounding the inmate. The inmate appeared to be wearing some form of an undergarment. There appeared to be some resistance on the part of the inmate to be returned to the cell and there appeared to be an episode when one of the Correctional Officers seemed to strike out at the inmate. Unfortunately, there was no video available of what transpired within the cell after the inmate was returned to the cell by the Corrections Officers. Shortly after the inmate was returned to the cell, some Corrections Officers were seen to leave the cell and some contents of the cell were seen to be removed from the cell as well. At some point after this, a large number of Correctional Officers were seen to respond and some returned to the cell. Paramedics were seen to enter the cell as well.

At some point, the base hospital physician pronounced the death from the Peterborough Regional Health Centre.

The Coroner visited the cell with the Forensics Officer from the Kawartha Lakes Police Service. The Coroner was requested to don a full forensic cover with a face mask prior to entering the cell. The Coroner found a young male lying supine on the floor of the cell. An identification tag was applied to the wrist. The body was not disturbed as the Coroner was planning a full forensic autopsy at the main Provincial Forensic Pathology Unit in Toronto. The Coroner did see resuscitation equipment scattered about the cell and the body was seen lying in a supine position. The body was bearded and there was material covering the mid-section. There were linear marks noted on the wrist and there was a mark on the forehead. There was in intravenous present in the right hand. There was an oral airway present. There was rigor mortis felt in the left elbow. Lividity was not assessed as the body was not rolled.

The Coroner had discussions with the Police, Regional Supervising Coroner, and later with the brother of the decedent on the evening of the death.

The discussion with the family involved discussions about why an autopsy was being requested as there was no clear explanation for the death at that time. The family expressed concerns about an autopsy in general and indicated that due to religious issues, they wanted the body released to them within twenty four hours of the death. The Coroner was anticipating that the Forensic Pathologist might request the retention of the heart organ and had the discussion with the family about why this might occur as well. The family was quite adamant that the heart was not to be retained and had to be returned when the body was released. The Coroner passed this information along to the Central Dispatch Unit and requested that the Forensic Pathologists be made aware of the family's religious requirements.

The Forensic Pathologist called the Coroner on the next morning and indicated that the family's requests could not be accommodated in terms of an early release as the case was being treated as a criminal investigation and therefore the autopsy might take several days and there would likely be a twenty four hour hold placed on the release of the body after the autopsy was done as occurred routinely with potential homicides. The Forensic Pathologist also indicated to the Coroner that the heart would be retained. She requested that the family be informed of these developments by the Coroner. However, the examination of the heart was able to be completed in time and the organ was not retained.

The Coroner passed this information along to the brother who requested to be allowed to speak with a higher authority within the Coroners system. The Investigating Coroner therefore called the Regional Supervising Coroner and asked the Regional Supervising Coroner to contact the brother. The Regional and Investigating Coroner suggested that the Family Liaison Officer with the Office of the Chief Coroner and Forensic Pathology Service of Ontario become involved. The family was connected with this Family Liaison Officer by the Regional Supervising Coroner and the Family Liaison Officer was able to facilitate a viewing by the family at the Coroners' Complex in Toronto. It is the Coroner's understanding that the Forensic Pathologist also had a meeting with the family around that time as well and this was arranged by the Family Liaison Officer.

The Forensic Pathologist did call the Coroner with the preliminary results of the autopsy and indicated there was "No anatomical cause of death" identified and the cause of death would be pending further investigations of the histology and the toxicology. It was reported that there were bruises seen on the back and the heart was slightly abnormal. None of these findings were felt to be sufficient to assign a cause of death at this time. The Forensic Pathologist requested that the Investigating Coroner attempt to obtain previous medical records for review. The Coroner completed an Authority to Seize the Medical Records from the psychiatrist in Scarborough who had treated the decedent in the past. This Authority to seize document was given to the Investigating Detective Constable at the Kawartha Lakes Police Service.

Medical records from the Central East Corrections Center were reviewed and it was noted that there were several notations made by staff about attempts to secure psychiatric records from the previous treating psychiatrist. The notes indicate that the records were not available due to a lack of consent and consent could not be easily obtained due to the deteriorating mental health of the inmate.

The Coroner was able to connect with the brother a few days later after the call from the Forensic Pathologist and attempted to explain the preliminary findings. The Coroner had left a telephone message with the brother a day or so prior to this conversation. The brother had questions about why no cause of death was available after the autopsy and the Coroner explained that it sometimes takes up to several months before a cause of death can be determined and in some cases even with an autopsy, the cause cannot be determined. The Coroner requested again that the family provide information about previous treating physicians so that a full past medical history could be obtained to assist the Pathologist in her deliberations about cause of death.

The autopsy results were finalized and received on June 15th, 2017. This written report was extremely detailed and quite complex. There

Statement #:	Coroner:	CIS Case #:
2016-2677-B	20332 - DR Ready, Eric	2016-14763

were additional consultation reports from a Forensic and Cardiovascular Pathologist as well as reports from Forensic Toxicology, Cardiology Genetics and a Forensic Odontologist. Despite the in-depth analysis, the conclusions at the end were that a cause of death could not be ascertained. Although there were obvious injuries noted externally, none of these injuries were felt to be sufficient to cause death. Toxicology showed the presence of olanzapine, beta-hydroxybutyrate and acetone but these were not felt to be at levels capable of causing death. The heart did show some mild enlargement, hypertrophy and remodeling. Genetic analysis directed at inheritable cardiac arrhythmic disorders and myopathic disorders showed two genetic variants but the significance of these variants was not known. In particular, the Forensic Pathologist was unable to find a disease or injury process that could explain death. It was not possible to show evidence of an asphyxia cause of death. The drug olanzapine is known to prolong the QT interval of the heart rhythm in some cases which can lead to fatal arrhythmias. It is possible that the documented physical and emotional struggle could combine with the heart changes and the olanzapine to create a pro-arrhythmic state leading to a fatal arrhythmia, but without electrocardiographic documentation at the time, this theory can only remain a theory. So the final conclusion after an in-depth post-mortem analysis is that the cause of death remains unascertained. This would then lead on to the manner of death conclusion being undetermined.

Due to the complexity of these findings, the local Coroner has not informed the family of this final autopsy report. It is planned that the Forensic Pathologist and/or the Regional Supervising Coroner will arrange to meet with the family to explain these very complex findings.

In summary, Soleiman Faqiri, (Date of Birth: 01 January 1986) of 152 Atherton Avenue, Ajax, ON, died at the Central East Correctional Centre at Lindsay while he was an inmate in a segregation cell (B-10) on December 15th, 2016. After a very thorough and complex post mortem analysis, the cause of death is unascertained and the manner of death is undetermined.

It is highly likely that an inquest will be called in this case due to the circumstances and more information may become available through that process. The family will be informed of this through the Regional Coroner's Office.

It should be noted as well that the Forensic Pathologist is advising that due to the genetic variations found relating to cardiac arrhythmias and myopathies, the surviving family members should be directed to seek consultation with a cardiologist who specializes in inherited genetic cardiac traits. This may have important implications for the future health and health care of the family members. Information for this purpose will be shared with family through the Regional Coroner's office.

Coroner's Signature: _____ **Date:** _____

2016-14763

ONTARIO FORENSIC PATHOLOGY SERVICE

Peer Review Form

CASE DATA	
Name of Deceased	Soleiman FAQIRI
Autopsy File Number	A2813-2016
Date of Autopsy	December 16, 2016
Pathologist	Dr. M. Bellis
Coroner	Dr. E. Ready
Regional Supervising Coroner	Dr. P. Dungey
Reviewing Pathologist	Dr. C. Kepron

ITEMS REVIEWED	Yes	No	N/A
Postmortem examination report	✓		
Photographs	✓		
Microscopic slides		✓	
Toxicology report	✓		
Other (specify): cardiac path, molecular genetics, biochemistry (x2), microbiology, odontology	✓		

Part 1: ADMINISTRATIVE AUDIT	Yes	No
Name and autopsy number recorded on report	✓	
Recommended template used	✓	
History provided	✓	
Opinion provided	✓	
Cause of death provided	✓	
Disclosure of retained samples and organs provided	✓	

Part 2: TECHNICAL AUDIT	Yes	No
Descriptions are satisfactory	✓	
Appropriate ancillary testing performed	✓	
Report is free of major language errors	✓	
Report is independently reviewable	✓	
Cause of death is reasonable	✓	
Other opinions are reasonable	✓	

The Chief Forensic Pathologist must be notified by the Reviewing Pathologist, if "no" is recorded in part 1 or 2, or if the turnaround time exceeds 12 months.

The pathologist who performed the postmortem examination is responsible for providing testimony on the autopsy.

A copy of this evaluation is to be submitted to the OFPS (OFPS@ontario.ca)

Received & Scanned by Appeal
JUN 14 2017
East Region - Kingston Office

Signature of Reviewing Pathologist

Date: June 13, 2017

2016-14763

REPORT OF POSTMORTEM EXAMINATION

NAME	Soleiman FAQIRI
AGE (Date of Birth)	30 years (January 1, 1986)
SEX	Male
AUTOPSY NUMBER	A2813 - 2016
CIS NUMBER	2016 - 14763
PLACE OF EXAMINATION	Provincial Forensic Pathology Unit, Toronto, Ontario, Canada
CORONER	Dr. E. Ready
DATE DEATH PRONOUNCED	December 15, 2016
FORENSIC PATHOLOGIST	Dr. M. Bellis
DATE OF AUTOPSY	December 16, 2016
PATHOLOGIST'S ASSISTANTS	T. Gardner, M. Arias
FORENSIC IDENTIFICATION OFFICERS	Constable N. Finn, Kawartha Lakes Police Service, Forensic Identification Services
INVESTIGATORS	Detective J. Rausch, Kawartha Lakes Police Service

CAUSE OF DEATH

Part I: *Immediate cause of death.*	
(a)	UNASCERTAINED

MB

QUALIFICATIONS

DEGREES AND PROFESSIONAL QUALIFICATIONS

Year	Certificate	Institution	Specialty
2006	MB BCh BAO	National University of Ireland, Cork	Medicine
2013	Fellow	Royal College of Physicians and Surgeons of Canada	Anatomical Pathology
2014	Fellow	Royal College of Physicians and Surgeons of Canada	Forensic Pathology

EMPLOYMENT AND APPOINTMENTS

2014 July – Present	Forensic Pathologist	Ontario Forensic Pathology Service
2014 July – Present	Coroner	Office of the Chief Coroner
2016 Jan - Present	Lecturer, part-time academic appointment	University of Toronto, Department of Laboratory Medicine and Pathobiology

I am a Forensic Pathologist at the Provincial Unit of the Ontario Forensic Pathology Service located in Toronto. I am listed as a Category A pathologist on the Ontario Forensic Pathology Service Register. I am also an Investigative Coroner for homicides and criminally suspicious deaths in Toronto. I have a part-time academic appointment as Lecturer with the Department of Laboratory Medicine and Pathobiology, Faculty of Medicine, University of Toronto. My main professional duties are to perform medicolegal autopsies and to supervise and teach pathology residents, medical students, and residents from other specialties. I have performed approximately 650 autopsies.

DECLARATION

This postmortem examination is performed under instruction of a Coroner's Warrant for Postmortem Examination. I have performed this postmortem examination using the *Practice Manual for Pathologists* (2014) produced by the Ontario Forensic Pathology Service.

I understand that my overriding duty is to the court, both in preparing reports and in giving oral evidence. I have complied with and will continue to comply with that duty. I have done my best, in preparing this report, to be accurate and complete. I have mentioned all matters that I regard as relevant to the opinions I have expressed.

SOURCES OF INFORMATION

1. Coroner's warrant for postmortem examination.
2. Scene photographs provided by the police.
3. CCTV video surveillance from Central East Correctional Centre.
4. Interview with the investigating officer attending the postmortem examination.
5. Ambulance call report.
6. Police occurrence and numerous witness statements (Kawartha Lakes Police Service, Occurrence # KL16010348).

7. Select hospital records from The Scarborough Hospital (dates in May, 2016).

HISTORY

1. This 30 year-old man with a history of mental illness was an inmate at Central East Correctional Centre in Lindsay, Ontario.
2. He had been incarcerated for the offence of aggravated assault and other similar charges since December 4th, 2016.
3. For some time during his incarceration he had been displaying behavioural problems including covering himself in his own excrement. He was recently placed in segregation.
4. On the day of death, at approximately 13:15 he was taken to the shower.
5. He remained in the shower for just under two hours. He was continuously resisting exiting the shower and was splashing/spraying the guards with water and throwing shampoo bottles at them.
6. He eventually calmed down and several guards were able to remove him from the shower and escort him to his cell. This was at approximately 3 pm.
7. He was escorted back to his cell with resistance, although his behavior was not described as "excited" or "delirious".
8. His ankles and wrists were cuffed and he was hunched forward as he walked. He was smacked after spitting on a guard.
9. Upon arrival to his cell, he resisted entering, and pepper spray was deployed. The guards were then able to get him into the cell and made several attempts to get him onto the ground again, against resistance. He was pepper sprayed a second time and was repeatedly told to "stop resisting". Despite this, he continued to try and get up. As the guards were not able to restrain him after a few minutes a "code blue" was called. (NOTE: from witness statements, this code blue is not equivalent to a medical code blue but rather indicates staff in trouble or inmates fighting or being aggressive).
10. After the code blue was called, several more staff entered the cell to relieve those who had already been inside struggling with the decedent. Several of the guards describe exhaustion after attempting to restrain Soleiman.
11. Soleiman continued to try to push himself up off the floor. Guards were reportedly holding down his limbs with his arms outstretched and cuffed in front of him while he was prone on the ground. A spit hood was placed over his head and leg irons were placed on his legs. It was explained to him that they needed to cuff his hands behind his back (switch the cuffs from in front to behind). He seemed to understand this and appeared to calm down and co-operate. After his arms were cuffed behind his back the guards began leaving the cell. This second shift of guards in the cell lasted for approximately 5 to 10 minutes.
12. Shortly thereafter, onlookers noted that Soleiman was no longer moving and had stopped breathing. A medical emergency was called.
13. Officers went in, removed the cuffs and placed him in recovery position. Nurses came into the cell very soon after. They began CPR and applied an AED which detected asystole.
14. Paramedics also arrived on scene. CPR continued for approximately 45 minutes in the cell. Death was pronounced at around 15:45 pm.

15. From witness statements there is no description of use of excessive force or chest/neck compression. Witnesses deny "hog-tying" the decedent. Many of the witnesses describe the ordeal as exhausting.
16. CCTV footage of the incident shows Mr. Faqiri being escorted back to his cell from the shower by 5 or 6 guards. It shows the guards having difficulty getting him into his cell. When they succeed at getting him in, they all go in with him. There is no footage from inside the cell. After a minute or so, several items are seen to be thrown out of the cell. A few minutes later numerous guards are seen running towards the cell. A few go into the cell and a few come out. Several remain outside of the cell. Guards continue to come and go. Several minutes pass and then several guards exit the cell. Many remain outside and are looking into the cell. Several more minutes pass and more guards exit the cell; many walk away while others remain outside the cell. After another short time all the guards go into the cell many other staff (most appear to be nursing staff) come running down the hallway and enter the cell. Soon after, a crash cart is wheeled over. People continue to come and go. The paramedics arrive after several minutes and leave after some time with all of their equipment; eventually all of the prison staff leave the vicinity of the cell.
17. Mr. Faqiri had a past medical history of schizoaffective disorder, cluster B personality disorder, sleep apnea (on CPAP) and possible hypertension. He was a smoker. He had been followed by psychiatry for a number of years. His medications included Seroquel, lithium and Epival. He was known to be non-compliant with his medications and became confrontational and agitated when not appropriately medicated.
18. He was admitted to hospital involuntarily in May of 2016 for 11 days for worsening symptoms. He appeared to be improving during his stay, however signed himself out against medical advice.

SCENE

1. After the autopsy I reviewed digital images of the scene provided to me by Constable Neil Finn, Kawartha Lakes Police Service, Forensic Identification Services.
2. The scene is that of a prison cell block.
3. There are several discarded items outside of the decedent's prison cell (mattress, book, white sheets, oranges sheets, garbage).
4. The body is lying supine on the floor of a small prison cell.
5. There is evidence of therapeutic intervention and medical paraphernalia around the body.
6. There are obvious injuries on the body.
7. Photos of the shower stall are also included; these are unremarkable.

PROCEDURES

1. A pre-autopsy CT scan has been obtained. The results are summarized in the ancillary test section below.
2. The Forensic Identification Officer has obtained photographs of the postmortem examination.

3. The PFPU staff photographer has also obtained photographs. These are stored on a secure server.
4. A standard external examination is performed.
5. Physical exhibits from the external and internal surfaces of the body are collected and transferred into the custody of Forensic Identification Officer (see exhibit list below).
6. A standard internal examination is performed. Special dissections are also performed as follows: layered musculocutaneous dissection of the anterior torso; layered dissection of the neck and face in an avascular field; posterior neck dissection; layered musculocutaneous dissection of posterior torso; musculocutaneous dissection of the extremities; dissection of the testes.
7. Consultations have been requested as follows: Cardiovascular Pathology and Forensic Odontology. These are documented in separate attached reports.

CONTINUITY AND IDENTIFICATION

1. The body is received in a body bag with police seal number 0031787 (red plastic tag securing zipper).
2. The body is positively identified to me by a Coroner's tag on the right wrist.
3. The hands are wrapped in brown paper bags secured with orange ribbons.

EXTERNAL EXAMINATION

GENERAL DESCRIPTION

1. The body is that of an obese (class II, BMI = 38) and appropriately developed 5'9" and 255 lbs. man.
2. The body is clothed in green shorts. A white folded towel is resting on the upper right thigh.
3. There is no jewelry. There are no personal effects.
4. Rigor mortis is present and full.
5. Postmortem hypostasis is present on the dorsal surfaces of the body.
6. There are no decompositional changes of the body.

HEAD AND NECK

1. See Signs of Recent Injury.
2. There is blood staining of the face.
3. There is facial acne.
4. The head hair is short black and wavy with male pattern baldness. Facial hair is worn as a black beard and moustache.
5. The corneas are clouded.
6. The conjunctivae are mildly congested with scattered bilateral petechial hemorrhages.
7. The philtrum and ears are unremarkable and uninjured.
8. The teeth are natural and uninjured.

9. The frenula are unremarkable and uninjured.
10. The labial, vestibular, gingival and buccal mucosae are uninjured.

TORSO

1. See Signs of Recent Injury.
2. Striae of the upper chest, abdomen and back.
3. Oblique scar, right lower abdominal quadrant, 7.4 cm.
4. Numerous punctate erythematous lesions associated with hair follicles of the abdomen and buttocks.

EXTREMITIES

1. See Signs of Recent Injury.
2. Healing ulcers:
    a. Ovoid, shallow, 3 x 1.5 cm, lateral upper left arm.
    b. Round, 0.5 cm, posterior left little finger.
    c. Round, 0.8 x 0.8 cm, dorsal left great toe.
    d. Bilateral inner thighs, with associated scarring.
2. Healing vertical laceration of right lower lip, 0.5 cm.
3. Scars:
    a. Vertical, 4.2 cm, dorsal right wrist.
    b. Curvilinear, 1 cm, beneath left elbow.
4. Crusted abrasions:
    a. Four, punctate, right inguinal crease.
    b. Vertical, intermittent, antero-lateral upper right thigh, 2.5 cm.
    c. Two, left knee, horizontal, 1.5 cm and vertical, 1.2 cm.
    d. Three, right knee, 1 to 3.6 cm.
    e. Several of the right knee and shin, punctate to 3.6 cm.
    f. Horizontal, intermittent, posteromedial left thigh, 3 cm.
    g. Vertical intermittent, lateral right thigh, 6 x 0.7 cm.
5. Numerous punctate erythematous lesions associated with hair follicles of the lower limbs.

EXTERNAL GENITALIA AND PERINEUM

1. The external genitalia, perineum, and anus are unremarkable and uninjured. Normal circumcised adult male.

SIGNS OF THERAPY/MEDICAL INTERVENTION

TUBES & CANNULA	Oropharyngeal airway (with blood in lumen) Intravenous cannula, dorsal right hand (attached to a bag of NaCl)
LEADS & MONITORS	Defibrillator pads
RESUCITATION-RELATED ARTIFACTS	Chest abrasions associated with defibrillation/ chest compressions

SIGNS OF RECENT (EXTERNAL AND INTERNAL) INJURY

i. Injuries are described in continuity with the body in anatomical position (upright, frontal with palms facing forward).
ii. The order in which the injuries are described is arbitrary and does not necessarily represent the order of occurrence.
iii. All injuries show a vital reaction and are recent unless stated otherwise.

HEAD AND NECK

1. Horizontal bruised laceration of the central forehead, 2.5 cm, associated with two foci of frontal subscalp hemorrhage 1 x 0.5 cm and 1.5 x 1 cm.
2. Abraded red-purple bruise, bridge of nose, 3 x 2.5 cm.
3. Subscalp occipital bruises, 6 x 3.5 right and 5 x 5 cm left.
4. Ill-defined blue-purple bruise of superior right neck, just inferior to ear, 2 x 1 cm, (associated with deep bruise over parotid gland).
5. Black-purple bruise, beneath left ear, 1.5 x 0.5 cm.
6. Abrasion, supero-lateral left neck, 0.6 x 0.3 cm.
7. Abraded purple bruise, infero-lateral left neck, 3 x 1.1 cm.
8. Intramuscular haemorrhages of the strap muscles of neck (inferior left and right sternothyroid).
9. Intramuscular hemorrhage, posterior lower neck, 6 x 3.5.

TORSO

1. Patchy petechial and blue bruising, upper left chest, 9 x 5.5 cm.
2. Pink bruise, upper left chest, 2.5 x 2 cm.
3. Linear vertical abrasion, posterior right shoulder, 3.5 x 0.5 cm.
4. Red bruise, left mid back, 4.5 x 8 cm, associated with intramuscular hemorrhage.
5. Horizontal petechial bruise, lateral left back, just beneath #4, 5 x 1 cm.
6. Intermittent horizontal abrasion, upper left buttock, 4 cm.
7. Oblique intermittent vertical abrasion, lateral left buttock, 5 cm.
8. Foci of intramuscular hemorrhage, posterior right and left shoulders.

HANDS AND FEET

1. There are horizontal ligature marks that partially encircle the ankles and wrists. They have a general 'tram-track' appearance. There are multiple impressions at each site. Most are associated with subcutaneous hemorrhage.
    a. Right wrist:

i. The anterior aspect is spared. Postero-medially there are four to five pink bruised marks, 8.5 x 4 cm in area. The postero-lateral aspect contains five mostly horizontal bruised abrasions, 0.7 to 2.8 cm each, 4.5 x 3.3 cm in area.

b. Left wrist:
   i. Anteriorly there is a punctate abrasion on the medial aspect and purple bruising of the inferior thenar eminence by the wrist, 8 x 2.3 cm. The medial wrist contains three short abrasions 0.6 to 1.4 cm each. Posteriorly there are at least three overlapping bruised horizontal tram-track marks, 9.5 x 2.6 cm in area.

c. Right ankle:
   i. Anteriorly there is a single faint horizontal pink bruises, 10 x 4 cm. Posteriorly there are three faint horizontal pink bruises, 7 x 4.5 cm area, (2 to 4 cm each in length).

d. Left ankle:
   i. Anteriorly there are two marks, 2 x 6.5 cm in area, (6.5 cm and 4.7 cm each in horizontal length). Posteriorly there are two pink bruised marks, 2.3 x 9.4 cm in area, (8.5 and 9.4 cm each in length).

2. Curvilinear laceration, posterior right little finger, 0.8 cm.
3. Punctate abrasions on posterior right fingers: three on the ring, one each on middle and index.
4. Faint pink bruise, dorsal right foot, 2 x 2 cm.
5. Brown-yellow bruise, inferior to lateral left ankle, 2.5 x 2.5 cm.
6. Purple bruise, base of left great toe, 1 x 0.5 cm.

RIGHT ARM

1. Oblique intermittent petechial bruise, lateral shoulder, 4.5 cm.
2. Ill-defined red bruise, antero-superior upper arm, 3 x 1.2 cm.
3. Oblique abrasion, anterior upper arm, 0.5 cm.
4. Round blue bruise, anterior-mid upper arm, 1.5 x 1.2 cm.
5. Red bruise, distal anterior upper arm, 5 x 3.5 cm.
6. Red bruise, antecubital fossa, 0.8 x 0.1 cm.
7. Blue bruise, antero-superior forearm, 0.6 x 0.6 cm.
8. Round red bruise, distal anterior forearm, 2.5 x 2.5 cm.
9. Black-purple bruise, postero-medial upper arm (near axilla), 1.3 x 1.1 cm.
10. Petechial bruise, postero-superior upper arm, 2.2 x 1.8 cm.
11. Two horizontal red bruises, postero-medial upper arm, 7 x 2 cm and 9 x 1.5 cm.
12. Blue bruise, postero-distal upper arm, 14 x 6 cm.
13. Annular intermittent abrasions (~ 5), elbow, 6 x 4.5 cm area (~ 0.3 cm each). NOTE: *a photo of this lesion is sent for Forensic Odontology interpretation. See attached report.*
14. Two punctate abrasions, posterior forearm.

LEFT ARM

1. Group of abrasions, anterior shoulder, 5.5 x 2 cm.
2. Patchy blue-purple bruise, supero-medial upper arm, 8.5 x 7 cm.
3. Patchy pink and petechial bruise, medial upper arm, 9 x 13 cm.
4. Punctate abrasions (~ 4-5), posterior upper arm.
5. Red-purple bruise, posterior upper arm, 15 x 14.5 cm.

6. Red bruise, medial elbow, 6.7 x 3 cm.
7. Annular intermittent abrasions, elbow, 6.2 x 4.5 cm area, (0.1 to 1 x 0.3 cm each). NOTE: *a photo of this lesion is sent for Forensic Odontology interpretation. See attached report.*
8. Oblique abrasion beneath left elbow, 2.2 cm.
9. Abrasion, postero-superior forearm, 1.5 x 0.8 cm.
10. Punctate abrasions (~ 4-5), posterior forearm.

RIGHT LEG

1. Oblique intermittent abrasion, supero-lateral thigh, 2 cm.
2. Faint ill-defined blue bruise, lateral thigh, 4 x 5 cm.
3. Intermittent abrasion, disto-lateral thigh, 7 x 2 cm.
4. Abrasion, knee, 0.8 x 0.5 cm.
5. Vertical abraded petechial bruise, shin, 2 .3 cm.
6. Faint intermittent oblique abrasion, popliteal fossa, 8.6 cm.
7. Curvilinear abrasion, superior calf, 2 x 0.6 cm.

LEFT LEG

1. Petechial bruise, disto-medial thigh, 1.7 x 1.7 cm.
2. Approximately six abrasions, knee, punctate to 1.8 cm.
3. Two abrasions shin, 0.9 x 0.7 cm and 0.6 x 0.4 cm (proximal and distal respectively).
4. Vertical abrasion, posterior thigh, 1.2 cm.
5. Cluster of punctate abrasions, 1.5 x 1 cm area, postero-distal left thigh.
6. Punctate abrasions, posterior right calf (~ 5-6).

INTERNAL EXAMINATION

BODY CAVITIES

PERICARDIUM & CAVITY	No blood or effusion.
PLEURA & CAVITIES	No blood or effusion.
PERITONEUM & CAVITY	No blood or effusion.
DIAPHRAGM	Unremarkable and intact.
RETROPERITONEUM	No hemorrhage.

CARDIOVASCULAR SYSTEM

NOTE: *The heart is submitted for same day Cardiovascular Pathology examination. See attached report.*

HEART (WEIGHT)	450 g.
AORTA	Fatty streaks.
INFERIOR VENA CAVA	Unremarkable.
PLMONARY VESSELS	Unremarkable.

NECK AND RESPIRATORY SYSTEM

TONGUE & SOFT TISSUES	See Signs of Recent Injury for soft tissues. The tongue is unremarkable.
HYOID BONE	Not fractured.
LARYNX	The laryngeal cartilages are uninjured. Erythema of laryngeal mucosa and epiglottis.
TRACHEA, BRONCHI & CARINA	Patent. Blood stained froth in airway
LUNGS	Right: 830 g; Left: 710 g. Terminal congestion and edema. No emboli, tumours, or areas of consolidation.

DIGESTIVE SYSTEM

ESOPHAGUS	Unremarkable.
STOMACH CONTENTS	Scant brown-red liquid.
GASTRIC MUCOSA	Very focal hemorrhagic gastropathy.
INTESTINES	Not obstructed or perforated; no evidence of segmental or global ischemia/infarction. There is inflammation of the duodenum. The small and large intestines have been opened.
VERMIFORM APPENDIX	Present; unremarkable.
LIVER	2470 g; Unremarkable
GALL BLADDER	Present. Cholesterolosis.
PANCREAS	Unremarkable.

GENITOURINARY SYSTEM

KIDNEYS	Right: 260 g; Left: 240g. Unremarkable.
URINARY BLADDER	Focally hemorrhagic. The urinary bladder contains approximately 40 cc of straw coloured urine.
UTERUS, CERVIX, VAGINA, OVARIES, & FALLOPIAN TUBES	Unremarkable.
PROSTATE GLAND	Unremarkable.

OTHER ORGANS AND TISSUES

SPLEEN	460 g. enlarged.
LYMPH NODES	Subcarinal enlargement.
BONES	The bones directly visualized within the body cavities including the ribs, sternum, vertebrae, and pelvis are free of fractures, callous, or degenerative changes. No long bone fractures are identified.
SKELETAL MUSCLES	See Signs of Recent Injury.
PITUITARY GLAND	Not examined.
THYROID GLAND	Unremarkable.
ADRENAL GLANDS	Unremarkable.

HEAD AND CENTRAL NERVOUS SYSTEM

SKULL	No fractures.
SCALP	Unremarkable.
BRAIN (WEIGHT)	1560 g.
DURA & ARACHNOID	Free of hemorrhage, exudate or discolouration. Patent venous sinuses.
CIRCLE OF WILLIS	Usual anatomic configuration without evidence of aneurysm. No atherosclerosis.
BRAIN (EXTERNAL)	Unremarkable.
BRAIN (INTERNAL)	Unremarkable. No localized lesions, hemorrhages or ventriculomegaly.
SPINAL CORD	Not examined.

ANCILLARY TESTS

TISSUE RETENTION	Small representative tissue samples are retained in formalin with selected tissues processed for histology. No whole organs are retained. *Stored tissue samples will undergo disposition two years after completion of the autopsy.*
TOXICOLOGY	Samples are collected and sent to the Centre of Forensic Sciences for toxicological analysis. Please see attached report.

FINDINGS (Femoral Blood):

SUBSTANCE	CONCENTRATION/ANALYSIS
Olanzapine	0.13 mg/L
Beta-hydroxybutyrate	141 mg/L
Acetone	Traces
Lithium	Not detected

FINDINGS (Urine):

SUBSTANCE	CONCENTRATION/ANALYSIS
Acetone	Detected

STAT URINE DRUG SCREEN	A qualitative tableside analysis for the presence of drugs in urine is performed: FINDINGS: negative for tested substances.
BIOCHEMISTRY	Vitreous fluid is collected and sent for biochemical analysis. Urine is collected for myoglobin testing. Please see attached reports.
	FINDINGS (in brief): Vitreous - unable to perform testing. Urine – negative for myoglobin.
MICROBIOLOGY	Blood is submitted for microbiological culture assessment. Please see attached report.
	FINDINGS (IN BRIEF): no growth.
RADIOLOGY	A pre-autopsy CT scan was obtained
	FINDINGS: No significant additional findings.
GENETICS	A sample of DNA is extracted for genetic testing. See attached report.

EXHIBITS

SEAL NUMBER	EXHIBIT
2V87658	TUBE OF RIGHT HEART BLOOD IN PRESERVATIVE
2V87655	TUBE OF FEMORAL BLOOD IN PRESERVATIVE
2V87656	TUBE OF FEMORAL BLOOD IN PRESERVATIVE
2V87657	TUBE OF URINE IN PRESERVATIVE
2V87659	TUBE OF BLOOD - EDTA
2V87643	PULLED HEAD HAIR
2V87641	LEFT HAND FINGERNAIL CLIPPINGS
2V87642	RIGHT HAND FINGERNAIL CLIPPINGS
2V87653	ORAL SWAB #1
2V87651	ORAL SWAB #2
2V87648	RECTAL SWAB #1
2V87649	RECTAL SWAB #2
2V87647	RECTAL SWAB #3
2V87646	PERI-ANAL SWAB #1
2V87645	PERI-ANAL SWAB #2
2V87654	PENILE SWAB #1
2V87652	PENILE SWAB #2
2V87644	? BITEMARK L. ELBOW
2V87650	? BITEMARK R. ELBOW

HISTOLOGY (in brief)

TISSUE	BLOCK	MICROSCOPIC FINDINGS (IN BRIEF)
LUNGS – SAMPLES FROM ALL LOBES	1-5	Intra-alveolar blood, emphysematous changes and very focal bronchopneumonia.
LEFT BACK	6	Acute perimuscular and interstitial hemorrhage.
RIGHT WRIST LIGATURE MARK	7	Acute soft tissue hemorrhage.
STRAP MUSCLE	8	Acute intramuscular hemorrhage.
PSOAS MUSCLE	9	Unremarkable.
RIGHT ANKLE LIGATURE MARK	10	Acute soft tissue hemorrhage.
SUBCARINAL LYMPH NODE	11	Sinusoidal congestion.
LIVER	12	Moderate steatosis and zone three congestion.
SPLEEN	13	Congested.
KIDNEYS	14, 15	Mild arteriosclerosis; otherwise unremarkable.

CONSULTATIONS

Cardiovascular Pathology and Forensic Odontology. See attached reports.

SUMMARY AND OPINION

1. This 30 year-old man was a prison inmate. He had a past medical history of schizoaffective and personality disorders and during his incarceration displayed behavioural problems. According to witness statements, on the day of death he required guarded escort from the shower to his cell, as he was displaying defiant and resistant behaviour. There were no reports of him acting "excited" or "delirious". He continued to resist guards as they attempted to get him into his cell and onto the floor, including after a code blue was called for additional officer assistance. At some point officers were able to restrain his outstretched arms and legs. Later a spit hood and leg shackles were placed on him. The guards were attempting to get his hands cuffed behind his back (they were currently cuffed in front of him). Eventually he calmed down and was co-operative; guards were able to cuff his hands behind him and leave the cell. Around this time, someone noticed he was no longer moving or breathing. Death was pronounced after a prolonged period of CPR.
2. At postmortem examination, the body was that of an obese adult man with obvious injuries. Findings included:
    a. Several external and internal bruises of the upper and lower extremities, posterior shoulders, posterior head and neck and left back. There were also a few hemorrhages of the lower strap muscles of the neck.
    b. Numerous abrasions on the body.
    c. Ligature marks around the ankles and wrists.
    d. Mild enlargement, hypertrophy and remodeling of the heart with no significant microscopic pathology.
    e. Focal incidental bronchopneumonia and duodenitis.
3. Toxicological analysis of postmortem blood revealed the presence of olanzapine, the ketoacid beta-hydroxybutyrate and acetone, none of which were in concentrations high enough to cause death.

    The results of blood cultures and urine myoglobin testing were negative.

    The Forensic Odontologist found that lesions identified on the decedent's elbows were not in keeping with bite-marks.

    Genetic testing for the presence of heritable cardiac arrhythmic and myopathic disorders revealed two variants of uncertain significance.
4. After review of the scene, history and circumstances, findings from the postmortem examination, results of ancillary tests and consultation reports, the cause of death is unascertained. All of the injuries identified were caused by blunt impact trauma. They were insufficient to explain death. Many of the injuries would be in keeping with the story of attempts to restrain this man, but falls, or blows or other impacts to these regions cannot be excluded. In addition, the possibility of an asphyxial mechanism of death cannot be excluded, although florid petechiae were not present.

    There were no disease processes to explain death. The heart showed only mild pathological changes. These findings may be due to his overall size/body habitus or may be secondary to sleep apnea and hypertension, or a combination.

    It is unclear what role, if any, the atypical antipsychotic medication olanzapine played in this death. This drug can increase the QT interval in some patients and contribute to sudden

cardiac death. The two cardiac variants of uncertain significance that were detected by genetic testing are not definitive disease-causing mutations. As such, without a full medical history/cardiac workup during life, their contribution to death remains unknown. However, given these findings, it is recommended that first degree relatives (parents, siblings and children, if any) be assessed by a cardiologist specializing in heart rhythm disorders.

Finally, it is clear from the history that this man was involved in a physical struggle with probable emotional agitation and pain prior to death which, in the setting of the genetic and toxicological findings, may have promoted a pro-arrhythmic state.

CAUSE OF DEATH

Part I: *Immediate cause of death and antecedent causes, if any, giving rise to the immediate cause (a).*

(a)	**UNASCERTAINED**

June 15, 2017
Date

MAGDALENI BELLIS MB BCh BAO FRCPC (AP & FP)
Forensic Pathologist
Ontario Forensic Pathology Service
25 Morton Shulman Avenue, Toronto, ON

ATTACHED REPORTS:
Toxicology Report
Vitreous Biochemistry Report
Urine myoglobin report
Microbiology Report
Cardiovascular Pathology Report
Cardiology Genetics Report
Forensic Odontology Report

THIS CASE HAS BEEN PEER REVIEWED ACCORDING TO PROTOCOL

MB

Cardiovascular Pathology Consultation
CVP101-16

Soleiman FAQIRI

Ministry of Community Safety and Correctional Services

Ontario Forensic Pathology Service

Forensic Services and
Coroners' Complex
25 Morton Shulman Avenue
Toronto ON M3M 0B1

Telephone: (416) 314-4040
Facsimile: (416) 314-4060

Ministère de la Sécurité communautaire et des Services correctionnels

Service de médecine légale de l'Ontario

Complexe des sciences judiciaires
et du coroner
25, Avenue Morton Shulman
Toronto ON M3M 0B1

Téléphone: (416) 314-4040
Télécopieur: (416) 314-4060

May 1, 2017

Dear Dr. Bellis:

Re: Soleiman FAQIRI
Cardiovascular Examination for Postmortem Examination A2813-16 (Dr. Bellis)

MAJOR DIAGNOSTIC FINDINGS:

a. **Heart [450 g], with:**
   i. **Cardiomegaly with remodelling***
   ii. **Mild right atrial enlargement**
   iii. **Mild to moderate right ventricular enlargement**
   iv. **Borderline right ventricular hypertrophy**
   v. **Mild enlargement of the left atrial chamber**
   vi. **Mild to moderate enlargement of the left ventricle**
   vii. **Eccentric pattern of left ventricular hypertrophy (mild)**
      1. **Cardiomyocyte hypertrophy with non-specific, mild, patchy, interstitial fibrous tissue deposition**

* The expected weight of the heart in an otherwise healthy adult male with a normal body mass index is 233-383 g. For individuals with body mass index >30, the range that is observed is greater and at least in part, likely reflects early cardiovascular disease from chronically increased body weight. Furthermore, depending on the circumstances, additional pathological factors within the myocardium may also contribute to the increased weight observed. (*Am J Forensic Med Pathol* 2012; 33: 363-367)

OPINION

Information provided indicated that the decedent was a 30-year-old who died in a segregation cell within a correctional centre. The decedent had been psychiatrically unwell in the days prior to his death and had been placed in segregation a number of hours prior to his death. He required a shower and had been in there for nearly two hours and was resisting leaving the shower. Ultimately, the decedent had his wrists and ankles restrained with cuffs and he was escorted by multiple correctional officers to his segregation cell. There may have been one or more altercations between the decedent and a number of correctional officers at some point, although the precise nature of this interaction and its timing relative to his death is not available. Within a few minutes of entering his segregation cell a 'code blue' was called, where it is inferred the decedent had a cardiac arrest. A nurse arrived some time later as well as paramedics later still.

Only limited medical history was available (none of which included his time while incarcerated), which included a prior diagnosis of schizoaffective disorder with manic episodes and a mixed cluster B personality disorder. The decedent had had multiple prior hospital admissions for his psychiatric disease and was noted to be variably compliant with his medications. At least at some point prior to his incarceration his medications included seroquel, lithium and epival. The decedent's remaining medical history included sleep apnea (for which he required a CPAP machine) and possible hypertension.

The relevant autopsy findings that were provided indicated that the decedent had a length of 5'9" and a weight of 255 lbs. (Body mass index: 37.7). The heart had a mass of 450 g. No pericardial effusion was identified. No anatomical cause for death was found at the time of the postmortem examination. Multiple blunt injuries were noted to his left flank and back. Postmortem toxicological assessment revealed the presence of olanzapine (0.13 mg/L) as well as the ketones beta-hydroxybutyrate (141 mg/L) and acetone (traces).

Following review of the materials submitted, multiple relevant opinions may be proffered following completion of the cardiovascular examination:

a. The heart exhibited evidence of mild enlargement, hypertrophy and remodeling. The decedent had a body mass index of 37.7 and this alone could have accounted for the relatively mild increase in the size of the heart, although the sleep apnea and possible history of hypertension may also have contributed to some degree.

b. Aside from mild cardiomyocyte hypertrophy and interstitial fibrous tissue deposition, no significant pathological process was identified microscopically. Specifically, no evidence of a clinically significant structural disease process, such as acute ischemia / infarction, valvular disease, acquired myocardial disease or cardiomyopathic process was identified.

c. The precise circumstances surrounding this man's death are not available. However, given the information accessible it is not unreasonable to infer that this man's death was sudden and unexpected. Thus, considering his young age, the absence of an anatomical cause for death at the time of the autopsy and the apparent sudden and unexpected nature of his death, genetic testing was undertaken to ascertain if an underlying primary arrhythmia syndrome could have contributed to his death. The results of this testing are found in the attached report. Two variants of uncertain significance were identified. One was in the *ANK2* gene (c.1135 C>T / p.R379C), which has been previously described in an individual from the United Arab Emirates, who had a family history where two family members reportedly died following cardiac arrest. This variant has also been described independently and in conjunction with other cardiogenic variants in individuals referred for Long QT Syndrome (LQTS) genetic testing. The R379C variant is a non-conservative amino acid substitution, which is likely to affect secondary protein structure and occurs at a position that is conserved across species. *In silico* analysis predicts that it is likely to damage protein structure and function. Nevertheless, the R379C variant has been observed in up to 0.5% of alleles from individuals of south Asian background indicating that it may be a rare, benign variant under some circumstances. Other heterozygous mutations in the *ANK2* gene have been reported in association with LQTS and other forms of cardiac arrhythmia.

   The *TTN* gene variant (c.33199_33219del21 / p.V11067_E11073del) represents an in-frame deletion, resulting in the loss of seven amino acid residues. This particular variant has been reported along with other cardiogenic variants in multiple individuals referred for genetic testing, although has not been deemed a pathological or benign variant. This variant is not expected to result in

truncation or loss of protein product through nonsense mediated decay of the mRNA. Mutations in the *TTN* gene have been associated with various forms of myopathy and cardiomyopathy.

The *ANK2* variant (and possibly the *TTN* variant) may be pathologically significant given cardiac phenotype present and the circumstances of this man's death (as far as they are understood). However, the ultimate determination of their significance requires interpretation of the decedent's full medical history (including any ECG's that may be available) in conjunction with assessment of first degree family members by a cardiac electrophysiologist and genetic counsellor.

d. Given the potential heritability of such conditions, it is recommended that first degree family members (such as siblings or children) consider cardiological assessment by a specialist with expertise in the diagnosis and management of heart rhythm disorders (e.g. an arrhythmia clinic / hereditary heart disease clinic) to help exclude a similar disorder in other family members. A sample of DNA will be banked at the Provincial Forensic Pathology Unit should it ever be needed for further genetic assessment by those evaluating family members.

e. Atypical anti-psychotic medications (such as olanzapine) are thought to increase the risk of QT interval prolongation in some patients and have, under some circumstances, been felt to contribute to sudden cardiac death[1,2]. This effect however is thought to be uncommon. It is unclear under the current circumstances if the atypical antipsychotic medication present (olanzapine) had contributed to a possible arrhythmic mechanism for death.

f. Finally, based on the history available, the decedent may have been psychologically agitated, he may have been physically active (struggling against his restraints and possibly correctional officers) and possibly in pain at the time of his presumed cardiac arrest. These factors may have promoted a pro-arrhythmic state when considered in the context of each of the other issues noted above.

g. Ultimately, interpretation of the relevance of these issues presented is dependent on an adequate understanding of events preceding this man's death as well as within the context of the entire death investigation.

[1]Salvo *et al.* Sudden cardiac and sudden unexplained death related to antipsychotics: A meta-analysis of observational studies. *Clinical Pharmacology and Therapeutics* (2016) 99(3); pp306-314
[2]Ray *et al.* Atypical antipsychotic drugs and the risk of sudden cardiac death. *New England Journal of Medicine* (2009) 360; pp225-235

I have reviewed the following items in preparation for this consultation report:

1. Draft Report of Postmortem Examination
2. Preliminary Coroner's Investigation Statement
3. Selected medical records
4. Postmortem toxicology report

MATERIALS SUBMITTED

The specimen container is labelled with the decedent's identification and as "A2813-16" and contains an adult heart.

MACROSCOPIC DESCRIPTION

An adult heart submitted for assessment. The heart has been in formalin for approximately one hour prior to the cardiovascular examination. Overall the heart shows evidence of cardiomegaly as well as mild hypertrophy involving the left ventricular myocardium. There is a moderate degree of epicardial fat noted on the external surface that is not only present within the atrioventricular and interventricular sulci but also extends significantly onto the ventral surface of the right ventricular myocardium, over the acute margin, the lateral wall of the left ventricle including the apex and the crux of the heart. There is also focal epicardial fat noted on the dorsal surface of the left atrial myocardium. Fine fibrous tissue is present over the posterior-inferior surface of the right ventricular myocardium as well as over the right atrial myocardium that is somewhat patchy and plaque-like in character. There is also very fine fibrous tissue noted on the ventral surface of the right ventricular myocardium as well as the pulmonary trunk. Ostia for the posterior and inferior caval veins are appropriately placed relative to the right atrial chamber. In addition, four pulmonary venous ostia were evident. The pulmonary trunk is identified ventral and to the left, of the dorsal and rightward oriented ascending aorta.

Serial sectioning of the epicardial coronary arteries demonstrates a right dominant circulation. The left main coronary artery trifurcates into the left anterior descending, the ramus intermedius and the circumflex branches. The ramus intermedius has a naturally small calibre. The right coronary artery not only leads to the posterior interventricular branch but also sends two small posterior basal branches on the basal aspect of the posterior-inferior surface of the left ventricular myocardium. No macroscopic evidence of significant atherosclerosis is identified. No congenital coronary artery anomalies are identified.

The right atrial chamber is mildly enlarged. This is associated with an appendage that is overall triangular in shape with a large orifice relative to the chamber and pectinate muscles that extend from the endoluminal aspect of this auricle out onto the free wall. Extending from the inferior caval orifice down towards the membranous interventricular septum is a relatively broad fibromuscular ridge that is a remnant of the fetal circulation and also extends across the edge of the coronary sinus ostium where there is a second fibromuscular ridge along its orifice. At its widest this ridge measures approximately 1.2 cm across and then tapers down to nothing as it reaches the membranous interventricular septum. There is mild fibrous tissue deposition noted in the vicinity of the interatrial septal overlying the limbus and the septum secundum. There is no patent foramen ovale identified. No evidence of endoluminal thrombus is identified.

The annular circumference of the trileaflet, tricuspid valve is 12.6 cm. Each of the leaflets is soft and pliable, however they exhibit a mild degree of fibrous tissue deposition noted over the rough zones of each leaflet. There is also perhaps a very mild degree of redundancy identified.

The thickness of the right ventricular myocardium along the acute margin is 0.4 cm and measures 0.4-0.5 cm along the right ventricular outflow tract. The right ventricular myocardium tapers as it approaches the apex of the heart. The inflow and outflow portions of the right ventricular chamber are mild to moderately enlarged. No evidence of fibrofatty tissue deposition is identified along the cut surface of the right ventricular myocardium. The right ventricular chamber extends down to the apex of the heart. The

septomarginal trabeculation and the crista supraventricularis are without significant abnormality. Aside from the aforementioned enlargement of the right ventricular outflow tract there is no macroscopic abnormality with the infundibulum. The trabeculae carneae are coarse relative to those identified in the left ventricular chamber. No evidence of an intramuscular, infundibular or perimembranous ventricular septal defect is identified.

The annular circumference of the tricuspid, pulmonary valve is 6.4 cm. Each of the cusps is soft, pliable and semi-translucent. A short segment of the pulmonary trunk is available for assessment and is without significant abnormality.

The left atrial chamber is mildly enlarged. It is associated with an appendage that likewise shows mild enlargement and is overall comma shaped with a narrow orifice relative to the chamber and pectinate muscles that are limited to the endoluminal aspect of this auricle. No endoluminal thrombus is identified. The septum secundum is without significant abnormality.

The annular circumference of the bileaflet, mitral valve is 10.5 cm. Each of the anterior and posterior leaflets is soft and pliable and exhibits mild fibromyxomatous thickening along the rough zone. Multiple fine primary and secondary chordae tendinae extend down to appropriately placed anterolateral and posteromedial papillary muscles.

The thickness of the left ventricular myocardium along its lateral aspect is 1.4 cm. The left ventricular chamber is mild to moderately enlarged. Within the left ventricle, although the trabeculae carneae are fine relative to those identified in the right ventricular chamber, they are still somewhat hypertrophied relative to what is normal. The left ventricular outflow tract demonstrates mild fibrous tissue deposition in its basal aspect, just inferior to the aortic annulus.

The annular circumference of the tricuspid, aortic valve is 6.4 cm. Each of the valve cusps is soft, pliable and semi-translucent although very small nodules of Arantius are identified on each one of the lunulae.

The right and left coronary artery ostia are identified within their respective aortic sinuses just below the level of the sinotubular junction. The left coronary artery ostium is able to accommodate a 4 mm vascular probe and the right coronary artery ostium able to accommodate a 3.5 cm vascular probe. A short segment of the ascending aorta is submitted which shows very focal evidence of atheroma within the intima but otherwise is unremarkable.

Serial sectioning of the right and left ventricular myocardium does not show macroscopic evidence of acute ischemic injury or fibrous tissue deposition.

Representative sections will be submitted as follows:

Block 1	Left main and proximal LAD
Block 2	Circumflex and proximal ramus intermedius
Block 3	Right coronary artery
Blocks 4-6	Anterior wall, RV
Blocks 7-9	Anterior wall, LV
Blocks 10-12	Lateral wall, LV
Blocks 13-15	Posterior-inferior wall, LV
Blocks 16-18	Muscular interventricular septum

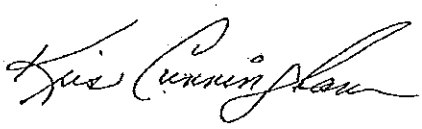
Block 19	Anterior wall, LV-apical third
Block 20	Posterior-inferior wall, LV-apical third
Blocks 21-25	AV nodal region
Blocks 26-27	Posterior-inferior wall, RV

PHOTOGRAPHY	Representative digital images of the specimen(s) were obtained and are stored at the Provincial Forensic Pathology Unit.
DISPOSITION OF TISSUES	Representative samples of the heart and all associated tissues are processed for histology. Any paraffin blocks and glass slides generated in the course of preparing this consultation report are retained and stored at the Provincial Forensic Pathology Unit.

MICROSCOPIC DESCRIPTION

Epicardial coronary arteries	Mild fibrocellular intimal thickening with early atheroma formation. No evidence of clinically significant luminal stenosis or luminal thrombus is identified.
Right and left ventricular myocardium	The right ventricular myocardium exhibits mild cardiomyocyte hypertrophy. The left ventricular myocardium exhibits cardiomyocyte hypertrophy with non-specific, mild, patchy, interstitial fibrous tissue deposition that is found throughout the thickness of the left ventricular myocardium. Otherwise, there is no evidence of acute ischemic injury, myofibre disarray, infiltrative disease or storage disease. The intramural coronary arteries are without pathological abnormality. Two incidental, minute foci of lymphohistiocytic inflammatory cells are observed within the interstitium of the posterior-medial papillary muscle, with no evidence of myonecrosis.
AV nodal region	Without pathological abnormality.

Please contact me if you have any questions



Kristopher S. Cunningham MD, PhD, FRCPC
Forensic and Cardiovascular Pathologist
Ontario Forensic Pathology Serivce

Report attached:

1. Molecular genetics report

GeneDx
an OMICS Health Company

Cardiology Genetics Report

Patient Name:	**FAQIRI, Soleiman**	**GeneDx Accession No:**	**1692195**
Date of Birth:	**01-JAN-1986**	**Date Specimen Obtained:**	**15-DEC-2016**
Specimen Type:	**DNA**	**Date Specimen Received:**	**03-MAR-2017**
Submitters ID No:	**A2813-16 MB**	**Date Test(s) Started:**	**06-MAR-2017**
Ordered By:	**Dr. Kris Cunningham**	**Date of Report:**	**05-APR-2017**

Test(s) Requested: Combined Cardiac Sequencing and Deletion/Duplication Panel

Genes Evaluated: ABCC9, ACTC (ACTC1), ACTN2, AKAP9, ALMS1, ALPK3, ANK2, ANKRD1, BAG3, BRAF, CACNA1C, CACNA2D1, CACNB2, CALM1, CALM2, CALM3, CASQ2, CAV3, CHRM2, CRYAB, CSRP3, DES, DMD, DOLK, DSC2, DSG2, DSP, DTNA, EMD, FHL1, FKRP, FKTN, GATAD1, GLA, GPD1L, HCN4, HRAS, ILK, JPH2, JUP, KCND3, KCNE1, KCNE2, KCNE3, KCNE1L (KCNE5), KCNH2, KCNJ2, KCNJ5, KCNJ8, KCNQ1, KRAS, LAMA4, LAMP2, LDB3, LMNA, MAP2K1, MAP2K2, MIB1, MTND1, MTND5, MTND6, MTTD, MTTG, MTTH, MTTI, MTTK, MTTL1, MTTL2, MTTM, MTTQ, MTTS1, MTTS2, MURC, MYBPC3, MYH6, MYH7, MYL2, MYL3, MYLK2, MYOZ2, MYPN, NEBL, NEXN, NKX2-5, NRAS, PDLIM3, PKP2, PLN, PRDM16, PRKAG2, PTPN11, RAF1, RANGRF, RBM20, RIT1, RYR2, SCN10A, SCN1B, SCN2B, SCN3B, SCN4B, SCN5A, SGCD, SNTA1, SOS1, TAZ, TCAP, TGFB3, TMEM43, TMPO, TNNC1, TNNI3, TNNT2, TPM1, TRDN, TRPM4, TTN, TTR, TXNRD2, VCL

Result: UNCERTAIN CLINICAL SIGNIFICANCE

Gene	Coding DNA	Variant	Zygosity	Classification
ANK2	c.1135 C>T	p.Arg379Cys (R379C)	Heterozygous	Variant of Uncertain Significance
TTN	c.33199_33219del21	p.Val11067_Glu11073del (V11067_E11073del)	Heterozygous	Variant of Uncertain Significance

No definitive pathogenic variants known to be associated with arrhythmia or cardiomyopathy were identified by sequence analysis of the genes on this panel. No deletion or duplication involving any of the nuclear genes analyzed was detected by concurrent targeted array CGH analysis with ExonArrayDx. The following genes are not included on the Combined Cardiac panel ExonArrayDx: CALM1, FKRP, HRAS, and the mitochondrial genes.

Interpretation: **This individual is heterozygous for variants of uncertain significance in the ANK2 and TTN genes.**

Cardiac arrhythmia and cardiomyopathy are genetically heterogeneous conditions. The genes associated with cardiac arrhythmia encode myocardial ion channel proteins that mediate the movement of sodium, potassium, and calcium ions through membranes, as well as channel-associated regulatory factors and interaction partners. The genes associated with cardiomyopathy encode cytoskeletal, contractile, or other proteins in the myocardial cells (Hershberger and Morales, 2015; Cirino and Ho, 2014; McNally et al., 2014). Many of the genes associated with cardiomyopathy encode sarcomeric proteins of the heart muscle, their regulatory factors, and their interaction partners. This panel also includes non-sarcomeric genes associated with a variety of myopathies such as those caused by storage or metabolic defects as well as genes that may also cause electrophysiological disturbances.

ANK2 Gene Summary: The ANK2 gene is a member of the ankyrin protein family, which play a role in cell motility, activation, proliferation, contact, and maintenance of specialized membrane domains (MIM: 106410). Heterozygous pathogenic variants in ANK2 have been reported in association with Long QT syndrome (LQTS) and other forms of cardiac arrhythmia, with variable expressivity and reduced penetrance (Mohler et al., 2007).

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Cardiology Genetics Report

Patient Name:	**FAQIRI, Soleiman**	**GeneDx Accession No:**	**1692195**
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Specimen Type:	**DNA**	**Date Specimen Received:**	**03-MAR-2017**
Submitters ID No:	**A2813-16 MB**	**Date Test(s) Started:**	**06-MAR-2017**
Ordered By:	**Dr. Kris Cunningham**	**Date of Report:**	**05-APR-2017**

ANK2 p.R379C: p.Arg379Cys (R379C) (CGT>TGT): c.1135 C>T in exon 11 of the ANK2 gene (NM_001148.4)

The R379C variant of uncertain significance in the ANK2 gene has been previously reported in the heterozygous state in one proband from the United Arab Emirates who underwent whole exome sequencing during an evaluation in a metabolic clinic (Al-Shamsi et al., 2016). This individual's family history is significant for two siblings who reportedly died of cardiac arrest. R379C was also reportedly identified in both parents; however, neither parent had undergone a cardiac evaluation (Al-Shamsi et al., 2016). In addition, R379C has been identified both independently and in conjunction with additional cardiogenetic variants in individuals referred for LQTS genetic testing at GeneDx; however, thus far, segregation data is absent for these individuals due to the lack of clinical information provided and insufficient participation by informative family members.

The R379C variant is a non-conservative amino acid substitution, which is likely to impact secondary protein structure as these residues differ in polarity, charge, size and/or other properties. This substitution also occurs at a position that is conserved across species. In silico analysis predicts R379C is probably damaging to the protein structure/function. Nevertheless, R379C has been observed in up to 0.5% of alleles from individuals of South Asian background, indicating it may be a rare benign variant in this population (Lek et al., 2016; 1000 Genomes Consortium et al., 2015).

Therefore, based on the currently available information, it is unclear whether this variant is pathogenic or rare benign. This result cannot be interpreted for diagnosis or used for family member screening at this time.

TTN Gene Summary: The TTN gene encodes titin, a large sarcomeric filament that provides elasticity to cardiac and skeletal muscle cells (Tskhovrebova et al., 2010). Pathogenic variants in TTN cause several forms of skeletal muscle myopathy and/or cardiomyopathy. Missense mutations in the 119th fibronectin domain cause hereditary myopathy with early respiratory failure (HMERF). HMERF is an adult onset, autosomal dominant, slowly progressive myopathy. The phenotype is variable, but patients often present with an abnormal gait or nocturnal breathing difficulties, although some individuals never develop symptoms, indicating incomplete penetrance (Pfeffer and Chinnery, 2014). Pathogenic variants within the sarcomere M-line, in the C-terminal domain (encoded by the last 6 exons), cause tibial muscular dystrophy (TMD), limb-girdle muscular dystrophy type 2J (LGMD2J), and Salih myopathy. TMD is an autosomal dominant, fully penetrant, adult-onset distal myopathy characterized by distal muscle weakness in the leg (Suominen et al., 2013). A founder mutation accounts for the majority of cases of TMD and homozygosity for this mutation causes LGMD2J, which is a progressive proximal myopathy that has an earlier age of onset than TMD (Pegoraro et al., 2012). Autosomal recessive Salih myopathy results from frameshift variants, and is characterized by muscle weakness and motor developmental delay followed by cardiac dysfunction and early death (Carmignac et al., 2012). Truncating variants in the A-band of TTN account for approximately 25% of familial and 18% of sporadic idiopathic dilated cardiomyopathy. However, truncating variants in the TTN gene have been reported in approximately 3% of control alleles (Herman et al., 2012). Lastly, variants in TTN have also been associated with arrhythmogenic right ventricular cardiomyopathy, and these variants may correlate to a distinct presentation compared to individuals with rare desmosomal gene variants, including supraventricular arrhythmias and conduction disease (Taylor et al., 2011; Brun et al., 2014).

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an OPKO Health Company

Cardiology Genetics Report

Patient Name:	**FAQIRI, Soleiman**	**GeneDx Accession No:**	**1692195**
Date of Birth:	**01-JAN-1986**	**Date Specimen Obtained:**	**15-DEC-2016**
Specimen Type:	**DNA**	**Date Specimen Received:**	**03-MAR-2017**
Submitters ID No:	**A2813-16 MB**	**Date Test(s) Started:**	**06-MAR-2017**
Ordered By:	**Dr. Kris Cunningham**	**Date of Report:**	**05-APR-2017**

TTN
c.33199_33219del21:

c.33199_33219del21:p.Val11067_Glu11073del (V11067_E11073del) in exon 144 of the TTN gene (NM_001256850.1)

The c.33199_33219del21 variant of uncertain significance has been identified in the TTN gene. This in-frame deletion results in a loss of seven amino acid residues, beginning with valine 11067 and ending with glutamic acid 11073. Although this variant has not been published as a pathogenic or benign variant to our knowledge, c.33199_33219del21 has been identified in conjunction with additional cardiogenetic variants in multiple individuals referred for cardiac genetic testing at GeneDx. Of note, varied cardiac phenotypes were reported, and informative segregation data are absent for these individuals. Furthermore, some of these individuals harbored another variant that corroborated their clinical presentation.

This variant is not expected to result in truncation or loss of protein product through nonsense-mediated mRNA decay. Furthermore, c.33199_33219del21 is not located in the A-band region of titin, where the majority of truncating pathogenic variants associated with DCM have been reported (Herman et al., 2012). Nevertheless, c.33199_33219del21 is not observed in large population cohorts (Lek et al., 2016; 1000 Genomes Consortium et al., 2015; Exome Variant Server).

Therefore, based on the currently available information, it is unclear whether this variant is pathogenic or benign. This result cannot be interpreted for diagnosis or used for family member screening at this time.

Follow-up Testing:

DNA not required for this completed test may be available for banking. DNA banking is not a service provided by GeneDx, however sending DNA to a banking facility may be arranged. Please visit the GeneTests website for more information: www.genetests.org or contact GeneDx at 301-519-2100 and ask to speak with a cardiology counselor for details.

Recommendation:

1. It is recommended that any first-degree relatives receive continued clinical evaluation and follow-up.

2. Targeted testing of affected relatives could be considered to determine if these variants segregate independently with disease in this family. If there are no affected relatives, targeted testing of this individual's parents could be considered to determine if either of these variants occurred de novo. Cumulative data about these variants, including multiple instances of segregation with disease and/or de novo occurrence, may assist in further variant interpretation and classification.

3. Genetic counseling is recommended to discuss the implications of this test report, specifically the risk of recurrence for this family.

Resources:

GenomeConnect is an NIH initiative created to enable individuals and families with the same genetic variant or medical history to connect and share de-identified information. If you are interested in participating, please visit www.genomeconnect.org.

GeneDx
an EXAGEN Health Company

Cardiology Genetics Report

Patient Name:	**FAQIRI, Soleiman**	**GeneDx Accession No:**	**1692195**
Date of Birth:	**01-JAN-1986**	**Date Specimen Obtained:**	**15-DEC-2016**
Specimen Type:	**DNA**	**Date Specimen Received:**	**03-MAR-2017**
Submitters ID No:	**A2813-16 MB**	**Date Test(s) Started:**	**06-MAR-2017**
Ordered By:	**Dr. Kris Cunningham**	**Date of Report:**	**05-APR-2017**

Methods: Using genomic DNA from the submitted specimen, the coding regions and splice junctions of the 120 genes (Only exons 1-44 for CACNA1C, only the KCNQ1-binding domains including Ser1570 residue for AKAP9, excluding exon 6 of the PKP2 gene and the following genomic regions of the TTN gene: chr2:179527692-179527782, 179523898-179523982, 179523731-179523815) are enriched using a proprietary targeted capture system developed by GeneDx. These targeted regions are sequenced simultaneously by massively parallel (NextGen) sequencing on an Illumina platform with paired-end reads. Bi-directional sequence is assembled, aligned to reference gene sequences based on human genome build GRCh37/UCSC hg19, and analyzed for sequence variants. Capillary sequencing is used to confirm all potentially pathogenic variants and to obtain sequence for regions where fewer than 15 reads are achieved by NextGen sequencing. Concurrent deletion/duplication testing was performed using exon-level oligo array CGH (ExonArrayDx) for most of the coding exons of the requested genes, except for CALM1, FKRP, HRAS, and the 14 mitochondrial genes. EMD, KCNE1L, SCN1B and TAZ have gene level resolution; exon level events may not be detected. Data analysis is performed using gene-specific filtering. Probe sequences and locations are based on human genome build GRCh37/UCSC hg19. The array is designed to detect most intragenic deletions and duplications. Confirmation of copy number changes is performed by MLPA, qPCR, or repeat array CGH analysis. Sequence and array CGH alterations are reported according to the Human Genome Variation Society (HGVS) or International System for Human Cytogenetic Nomenclature (ISCN) guidelines, respectively. Benign and likely benign variants, if present, are not included in this report but are available upon request.

Supplemental Variant Information:

Gene: Coding DNA Variant (Protein)	ANK2: c.1136C>T p.Arg379Cys (R379C)	TTN: c.33199_33219del21 p.Val11067_Gln11073del (V11067_Q11073del)
Classification	Variant of Uncertain Significance	Variant of Uncertain Significance
Zygosity	Heterozygous	Heterozygous
Chr:Position	4: 114177035	2: 179542469
dbSNP	rs143043717	rs587780487
1000G	4/5008 (0.0008)	
1000G_Highest	0.0104	
1000G_Highest_Sub	"Punjabi from Lahore, Pakistan"	
ExAC_Freq	0.0006839	
ExAC_Latino		
ExAC_NFE	0.00002999	
ExAC_AFR	0.00000000	
ExAC_EAS	0.00011555	
ExAC_FIN	0.00000000	
ExAC_Other	0.00220264	
ExAC_SAS	0.00454270	
ExAC_Hom	1	
PROVEAN		
MutTaster_Score	1 (D)	
Interpro_Domain	"Ankyrin repeat-containing domain"	
ClinVar	Uncertain significance	Uncertain significance

b) The suspect dentition cannot be excluded

I examined the image sets from the material provided:

Color photographs A 2813-16AAF and A 2813-16AAG

and

Infrared photograph A 2813-16ABE-IR

And

Ultraviolet photograph A 2813-16ABF-UV

Description

1. A 2813-16AAF

   The dermal injury on this photograph shows eight discrete injuries roughly in a horseshoe pattern. It is tempting for the eye to join these injuries together however they may not all necessarily be related – physically or temporally. Starting at the lower left of the frame there is an angular marking that is a tissue tear. Following in a clockwise direction there is a an abrasion that is irregular in shape but not aligned with the angular tear above. Following along clockwise there is a yellowish indented area that does not possess the dimensions of teeth as it is oblong. Proceeding clockwise (and medially) there are three irregular tissue injuries approximately 4 mm in greatest diameter – smaller than humn teeth. Finally passing "around" the "arch" clockwise and anatomically inferiorly a yellowish purple scabbed area that appears to be indented into skin but not through it. None of these injuries independently are "tooth-like" in appearance and taken as a collective arch shape the "set" is too large for a human arch. Furthermore there is no opposing arch visible anywhere – which one would expect. Finally if these are all of one arch one has to think about how these markings, if they were a bite mark could occur. In order for this to be a single arch marking with no opposing dentition the biter would have to put the entire structure in their mouth. This is not possible with such a large area.

2. A 2813-16AAG

   This injury is comprised of numerous small lacerations ovrer an area too large to be a bite mark. Additionally the pattern of hemorrhage seen here – both interior to and external to the arch is not typical for a bite mark. This alone rules this out a s a bite mark. A bite mark is a pinch injury not an impact injury and generally the

greatest amount of contusion is demarcated central to the perimeter of the teeth that caused it. In this case the hemorrhage is not coherently related to the small lacerations.

3. Infrared photograph A 2813-16ABE-IR

   This infrared image very nicely depicts what the observer could readily believe to be a dental arch however this "arch" is a fortuitous arrangement of shadows and at least two of the "deeper" injury elements would be at the posterior of a dental arch. Since the mouth opens partly like a hinge – it is not a hinge after relatively small amount of opening – it would be highly unlikely that these indented markings seen at the bottom of the viewers field could be teeth.

4. Ultraviolet photograph A 2813-16ABF-UV

   This is as nice an ultraviolet image as I have ever seen. It is particularly useful in this case because it shows that each of the elements the observer's eyes see are very different with respect to the elemental properties. The mark to the lower right is clearly not through the skin although it is the largest. The mark at the lower left is through the skin yet the one directly above it is not all the way through the skin. The medial markings all appear to extend through the skin. The varying depths of the markings as seen here on this UV view are not typical of a bite mark.

Conclusions:

I conclude that this injury or constellation of injuries is not a bite mark for the reasons stated above.

If you require further information please do not hesitate to contact me.

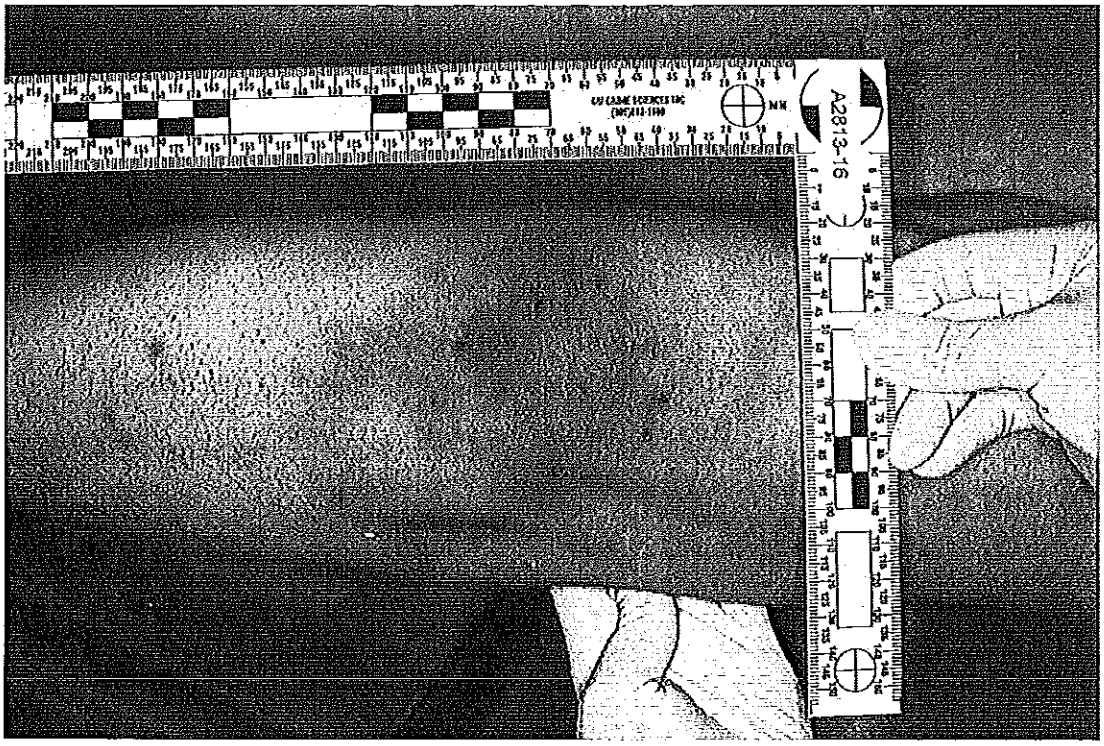
Sincerely and with respect,

R.E. Wood DDS MSc PhD FRCD (C) Dip. A.B.F.O.
Consultant in Forensic Odontology, Ontario Forensic Pathology Service,
Office of the Chief Coroner,
Toronto Ontario

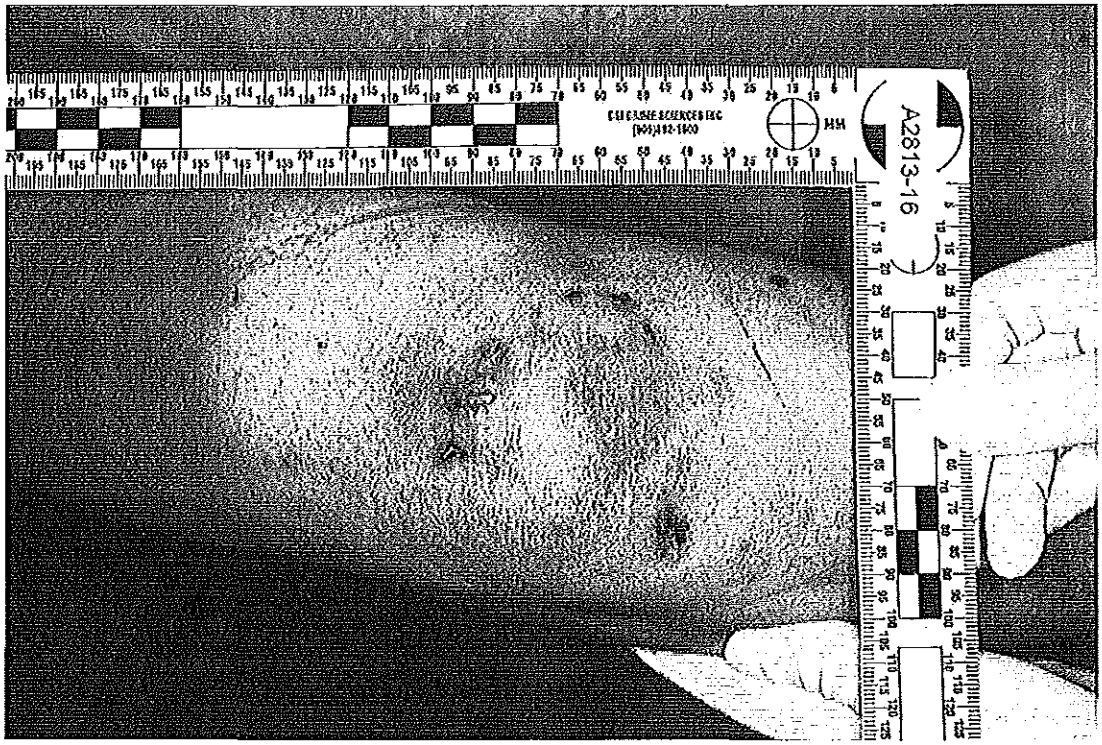
This report is based on the materials provided to me at the time of the comparison. If new evidence is made available the author of this report reserves the right to amend of change this report.

Images appended...

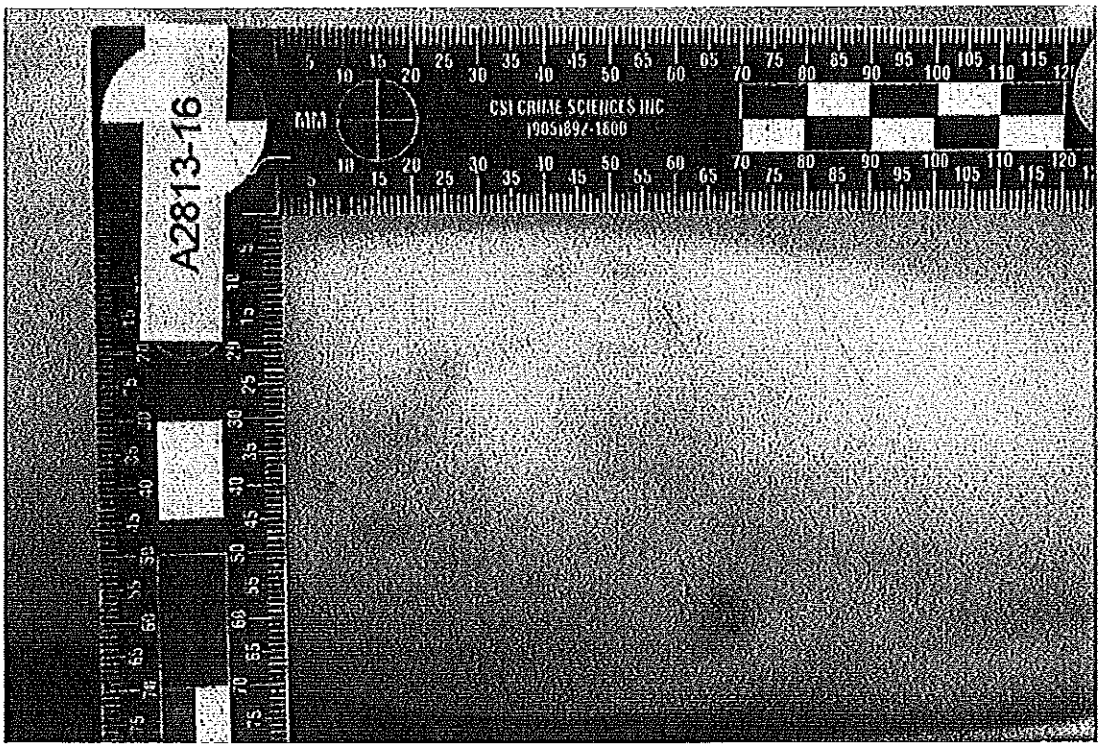
A 2813-16AAF



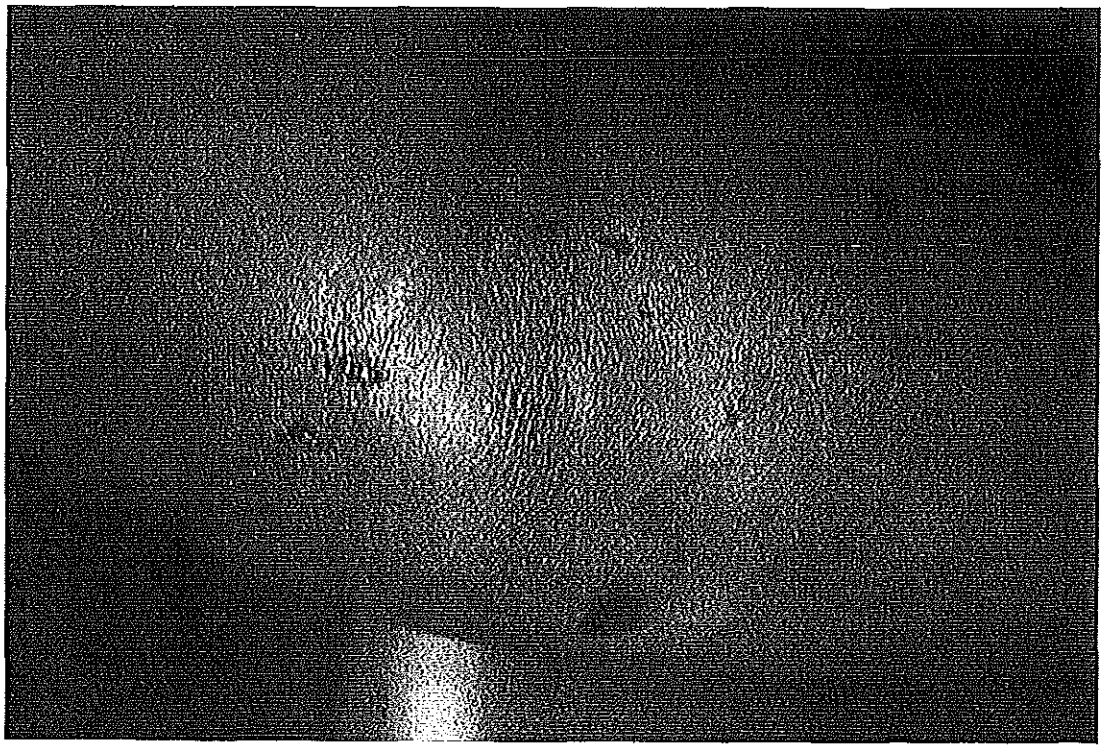
A 2813-16AAG



A2813-16ABE-IR



A2813-16ABF - UV



Ministry of Community Safety and Correctional Services

Centre of Forensic Sciences

25 Morton Shulman Avenue
Toronto ON M3M 0B1
Telephone: 647 329-1320
Facsimile: 647 329-1361

Ministère de la Sécurité communautaire et des Services correctionnels

Centre des sciences judiciaires

25, avenue Morton Shulman
Toronto ON M3M 0B1
Téléphone : 647 329-1320
Télécopieur : 647 329-1361

Ontario

TOXICOLOGY REPORT

Date: February 22, 2017

CFS File No.: 2016-014665
CFS Request No.: 0004
Incident No.: A2813-16
CIS#2016-14763
KL16010348
R2016-02587

E-mail: amanda.lowe@ontario.ca
Telephone: 647-329-1402

Reference: FAQIRI, Soleiman

For: Dr. M. Bellis, Pathologist, Toronto

Investigators: Dr. E. Ready, Coroner, Lindsay
PC. N. Finn #51, City of Kawartha Lakes Police
D/Cst. J. Burke #58, City of Kawartha Lakes Police
Office of the Chief Coroner, Toronto

Reported by: Amanda Lowe, M.Sc., Forensic Scientist, Toxicology

PURPOSE

To examine the submitted item(s) for the presence/absence of drugs and/or poisons.

ITEM		RESULTS		MU
1	Femoral Blood 2V87655	Olanzapine:	0.13 mg/L	± 0.01 mg/L
		Beta-hydroxybutyrate:	141 mg/L	± 12 mg/L
		Acetone:	traces	
		Barbiturates:	not detected	
		Cannabinoid metabolites:	not detected	
		Cocaine:	not detected	
		Codeine:	not detected	
		Ethanol:	not detected	
		Fentanyl:	not detected	
		Hydromorphone:	not detected	
		Methadone:	not detected	
		Morphine:	not detected	
		Oxycodone:	not detected	
4	Urine 2V87657	Acetone:	detected	
		Ethanol:	not detected	

CONCLUSIONS

1. Therapeutic olanzapine concentrations vary widely. Daily oral administration of 10 – 20 mg of olanzapine to patients resulted in plasma concentrations ranging from 0.008 – 0.032 mg/L (Aravagiri *et al.* 1997. Ther Drug Mon 19:307-313). However, plasma concentrations up to 0.3 mg/L have been tolerated without adverse effects (Jenkins *et al.* 1998. J Anal Toxicol 22:605-609). Adverse effects may include slurred speech, disorientation, and/or sedation. In a fatality attributed to olanzapine overdose, a post-mortem femoral blood concentration of 1.2 mg/L was detected (Stephens *et al.* 1998. J Forensic Sci 43:1252-1253). Olanzapine is susceptible to degradation in storage, so the detected concentration may have been higher at the time of death.
2. Beta-hydroxybutyrate is a ketone which arises naturally in the body and concentrations less than 50 mg/L are considered incidental. Elevated concentrations have been associated with ketosis due to a variety of factors including periods of fasting, uncontrolled diabetes, and alcoholic ketoacidosis. In a review of more than 350 fatalities, the authors concluded that post-mortem blood beta-hydroxybutyrate concentrations greater than 250 mg/L are pathologically significant (Elliot *et al.* 2010. Forensic Sci Int 198:53-57). Acetone can arise as a result of ketosis.
3. **In item 1, no other significant findings were detected by the following methods: GC and GC/MS screen, Headspace GC-FID, GHB/BHB method, and LC-MS/MS mix 3.**
4. **In item 4, no other significant findings were detected by the following method: Headspace GC-FID.**
5. Based on the findings to date, and the information associated with this case, further examination is not warranted at this time. If additional relevant information becomes available, further analysis may be undertaken upon request.

NOTES/REMARKS

Also received but not examined:	Item 2, Femoral Blood	2V87656
	Item 3, Heart Blood	2V87658

To obtain information about sample availability for re-testing or additional testing, clarification, or a copy of the documentation underlying this report, please contact the writer of this report.

Technical assistance has been provided to the writer of this report, in accordance with the policies and procedures of the Centre of Forensic Sciences, in the examination and analysis of items discussed in this report.

Numerical values are reported ± measurement uncertainty (MU), which represents the variability of the analytical method with 95.45% confidence.

This report contains interpretations and opinions based on scientific data.

Items were also submitted to the Biology and Chemistry Sections of the Centre of Forensic Sciences.

CONTINUITY

The "Evidence List Report" attached to this report relates the laboratory item number to the submitter item number and description.

Item Receipt

Refer to the "CFS submission receipt" previously provided to the submitter for information regarding item receipt and acceptance.

All item transfers occurring within CFS are recorded in the Laboratory Information Management System (LIMS). A full continuity report from this system is available on request.

Item Disposition

ITEMS 2 AND 3 WILL BE RETAINED FOLLOWING THE PROVISIONS OUTLINED IN THE CORONER'S ACT. ITEMS 1 AND 4 WILL BE RETAINED FOR THREE MONTHS FROM THE DATE OF THIS REPORT AND THEN DESTROYED.

ATTRIBUTION

Attribution of items to a source is based on information provided to the Centre of Forensic Sciences.

ADDITIONAL DOCUMENTS

Statements of qualifications for CFS scientists (including the writer of this report) are available to the Ministry of the Attorney General, Criminal Law Division on CLD.net in the expert evidence section in the legal research and law area. Access is restricted to legal staff, including crown attorneys.

Technical information sheets and glossaries that define the technical terms used in this report are available for download from the CFS section of the Ministry of Community Safety and Correctional Services (MCSCS) website.

Technical Information Sheet(s) can be accessed using the following hyperlink:
Technical Information Sheets – Toxicology

ALI-087-T

The Centre of Forensic Sciences is an ASCLD/LAB-*International* accredited testing laboratory and conforms with ISO/IEC 17025:2005.

Ministry of Community Safety and Correctional Services

Centre of Forensic Sciences

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Télécopieur : 647 329-1361

Ontario

TOXICOLOGY REPORT

Please refer to previous toxicology report dated February 22, 2017

Date: May 25, 2017

CFS File No.: **2016-014665**
CFS Request No.: 0036
Incident No.: A2813-16
CIS#2016-14763
KL16010348
R2016-02587

E-mail: amanda.lowe@ontario.ca
Telephone: 647-329-1402

Reference: FAQIRI, Soleiman

For: Dr. M. Bellis, Pathologist, Toronto

Investigators: Dr. E. Ready, Coroner, Lindsay
PC. N. Finn #51, City of Kawartha Lakes Police
D/Cst. J. Burke #58, City of Kawartha Lakes Police
Office of the Chief Coroner, Toronto

Reported by: Amanda Lowe, M.Sc., Forensic Scientist, Toxicology

PURPOSE

To examine the submitted item(s) for the presence/absence of drugs and/or poisons.

ITEM		RESULTS	
1	Femoral Blood 2V87655	Lithium:	not detected

CONCLUSIONS

1. Based on the findings to date, and the information associated with this case, further examination is not warranted at this time. If additional relevant information becomes available, further analysis may be undertaken upon request.

NOTES/REMARKS

Also received but not examined:	Item 2, Femoral Blood	2V87656
	Item 3, Heart Blood	2V87658
	Item 4, Urine	2V87657

To obtain information about sample availability for re-testing or additional testing, clarification, or a copy of the documentation underlying this report, please contact the writer of this report.

Technical assistance has been provided to the writer of this report, in accordance with the policies and procedures of the Centre of Forensic Sciences, in the examination and analysis of items discussed in this report.

This report contains interpretations and opinions based on scientific data.

Items were also submitted to the Chemistry Section of the Centre of Forensic Sciences.

CONTINUITY

The "Evidence List Report" attached to this report relates the laboratory item number to the submitter item number and description.

Item Receipt

Refer to the "CFS submission receipt" previously provided to the submitter for information regarding item receipt and acceptance.

All item transfers occurring within CFS are recorded in the Laboratory Information Management System (LIMS). A full continuity report from this system is available on request.

Item Disposition

ITEMS 1, 2 AND 3 WILL BE RETAINED FOLLOWING THE PROVISIONS OUTLINED IN THE CORONER'S ACT. ITEM 4 WILL BE RETAINED FOR THREE MONTHS FROM FEBRUARY 22, 2017 AND THEN DESTROYED.

ATTRIBUTION

Attribution of items to a source is based on information provided to the Centre of Forensic Sciences.

ADDITIONAL DOCUMENTS

Statements of qualifications for CFS scientists (including the writer of this report) are available to the Ministry of the Attorney General, Criminal Law Division on CLD.net in the expert evidence section in the legal research and law area. Access is restricted to legal staff, including crown attorneys.

Technical information sheets and glossaries that define the technical terms used in this report are available for download from the CFS section of the Ministry of Community Safety and Correctional Services (MCSCS) website.

Technical Information Sheet(s) can be accessed using the following hyperlink:
Technical Information Sheets – Toxicology

ALI-087-T

The Centre of Forensic Sciences is an ASCLD/LAB-*International* accredited testing laboratory and conforms with ISO/IEC 17025:2005.

UNIVERSITY HEALTH NETWORK
MOUNT SINAI HOSPITAL
JOSEPH & WOLF LEBOVIC HEALTH COMPLEX
Department of Microbiology
600 University Avenue
Toronto, ON M5G 1X5
T: 416-586-4432
F: 416-586-3138

Medical Record#:	FAQIRI, SO
Case Number:	R2016-02587
Last Name:	FAQIRI
First Name:	SOLEIMAN
Date of Birth:	Sex: N
Health Card#:	
Facility:	ONT
Location:	FORENSIC PATHOLOGY
Room:	
Physician:	BELLIS, MAGGIE
Copy to:	
Order#:	R6191304
D&T Ordered:	2016.12.19 11:09
D&T Received:	2016.12.19 11:09

Source: Blood Culture
Site: A2813-16/R2016-02587

Test Results — Status Reported

Culture and Sensitivity: — FINAL 2016.12.24 12:00

No growth after 5 days incubation.

---

* FIRST PRINTING OF THIS RESULT
PRINTED: 2016.12.24 17:04 by SCC to MS2

Order#: R6191304
D&T Ordered: 2016.12.19 11:09

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North York, Ontario M3C 1Z2
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www.ICLabs.ca

Ontario Forensic Pathology Services, Forensic Service and Coroner's Complex, 25 Morton Shulman Ave.
Toronto, ON M3M 0B1
Canada

Patient Name	Sex	Date of Birth (mm/dd/yyyy)
FAQIRY, SOLEIMAN	M	01/01/1986

Client's File No.
R2016-02587/A2813-16

Order ID
39416358

Health Number

ICL Login Date (mm/dd/yyyy)
12/23/2016 1:12PM

Report Printed
12/29/2016 7:13AM

Authorized Requester
Bellis, Maggie, Dr.

Myoglobin, Random Urine

Final - Received 12/29/2016 7:13AM

Sample ID: UHN161223015
Collection Date (mm/dd/yyyy)
12/16/2016 12:00PM

TEST	RESULT	FLAG	NORMAL/THERAPEUTIC RANGE	UNITS	TEST SITE
Urine myoglobin (Qualitative)	Negative		Negative		UHN
Tech Comment (urine)	NOT REPORTED				UHN

Reporting Laboratories:
(1) UHN, University Health Network, 200 Elizabeth St, 11E-444, Toronto, ON M5G 2C4,

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Toronto, ON M3M 0B1
Canada

Patient Name	Sex	Date of Birth (mm/dd/yyyy)
FAQIRI, SOLEIMAN	M	01/01/1986

Client's File No.
R2016-02587/A281316

Order ID
42716358

Health Number

ICL Login Date (mm/dd/yyyy)
12/23/2016 1:38PM

Report Printed
12/23/2016 6:53PM

Authorized Requester
Bellis, Maggie, Dr.

Order Comments: Fluid Type = : VH

Chloride, Fluid

Sample ID: UHN161223027
Collection Date (mm/dd/yyyy)
12/16/2016 12:00PM

Final - Received 12/23/2016 6:53PM

Order Choice Comments: Unable to perform testing, viscosity too high

TEST	RESULT	FLAG	NORMAL/THERAPEUTIC RANGE	UNITS	TEST SITE
FLUID TYPE	Not provided				UHN
CHLORIDE-FLUID	NOT REPORTED			mmol/L	UHN
Tech Comment (fluid)	The reference range and other method performance specifications have not been established for this body fluid. The test result must be integrated into the clinical context for interpretation. Comparison to the concentration in blood is strongly recommended.				UHN

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Patient Name	Sex	Date of Birth (mm/dd/yyyy)
FAQIRI, SOLEIMAN	M	01/01/1986

Order ID
42716358

Client's File No.
R2016-02587/A281316

Health Number

ICL Login Date (mm/dd/yyyy)
12/23/2016 1:38PM

Report Printed
12/23/2016 6:53PM

Authorized Requester
Bellis, Maggie, Dr.

Creatinine, Fluid

Final - Received 12/23/2016 6:53PM

Sample ID: UHN161223027
Collection Date (mm/dd/yyyy)
12/16/2016 12:00PM

Order Choice Unable to perform testing, viscosity too high
Comments:

TEST	RESULT	FLAG	NORMAL/THERAPEUTIC RANGE	UNITS	TEST SITE
FLUID TYPE	Not provided				UHN
CREATININE-FLUID	NOT REPORTED			umol/L	UHN
Tech Comment (fluid)	The reference range and other method performance specifications have not been established for this body fluid. The test result must be integrated into the clinical context for interpretation. Comparison to the concentration in blood is strongly recommended.				UHN

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Canada

Patient Name	Sex	Date of Birth (mm/dd/yyyy)
FAQIRI, SOLEIMAN	M	01/01/1986

Client's File No.
R2016-02587/A281316

Order ID
42716358

Health Number

ICL Login Date (mm/dd/yyyy)
12/23/2016 1:38PM

Report Printed
12/23/2016 6:53PM

Authorized Requester
Bellis, Maggie, Dr.

Glucose, Fluid

Final - Received 12/23/2016 6:53PM

Sample ID: UHN161223027
Collection Date (mm/dd/yyyy)
12/16/2016 12:00PM

Order Choice Unable to perform testing, viscosity too high
Comments:

TEST	RESULT	FLAG	NORMAL/THERAPEUTIC RANGE	UNITS	TEST SITE
FLUID TYPE	Not provided				UHN
GLUCOSE-FLUID	NOT REPORTED			mmol/L	UHN
Tech Comment (fluid)	The reference range and other method performance specifications have not been established for this body fluid. The test result must be integrated into the clinical context for interpretation. Comparison to the concentration in blood is strongly recommended.				UHN

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Canada

Patient Name	Sex	Date of Birth (mm/dd/yyyy)
FAQIRI, SOLEIMAN	M	01/01/1986

Client's File No.
R2016-02587/A281316

Order ID
42716358

Health Number

ICL Login Date (mm/dd/yyyy)
12/23/2016 1:38PM

Report Printed
12/23/2016 6:53PM

Authorized Requester
Bellis, Maggie, Dr.

Potassium, Fluid

Sample ID: UHN161223027
Collection Date (mm/dd/yyyy)
12/16/2016 12:00PM

Final - Received 12/23/2016 6:53PM

Order Choice Unable to perform testing, viscosity too high
Comments:

TEST	RESULT	FLAG	NORMAL/THERAPEUTIC RANGE	UNITS	TEST SITE
FLUID TYPE	Not provided				UHN
POTASSIUM-FLUID	NOT REPORTED			mmol/L	UHN
Tech Comment (fluid)	The reference range and other method performance specifications have not been established for this body fluid. The test result must be integrated into the clinical context for interpretation. Comparison to the concentration in blood is strongly recommended.				UHN

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Canada

Patient Name	Sex	Date of Birth (mm/dd/yyyy)
FAQIRI, SOLEIMAN	M	01/01/1986

Order ID
42716358

Client's File No.
R2016-02587/A281316

Health Number

ICL Login Date (mm/dd/yyyy)
12/23/2016 1:38PM

Report Printed
12/23/2016 6:53PM

Authorized Requester
Bellis, Maggie, Dr.

Ketones, Fluid

Sample ID: UHN161223027
Collection Date (mm/dd/yyyy)
12/16/2016 12:00PM

Final - Received 12/23/2016 6:53PM

Order Choice Unable to perform testing, viscosity too high
Comments:

TEST	RESULT	FLAG	NORMAL/THERAPEUTIC RANGE	UNITS	TEST SITE
FLUID TYPE	Not provided				UHN
fluid ketones				mmol/L	UHN
Negative					
Tech Comment (fluid)	The reference range and other method performance specifications have not been established for this body fluid. The test result must be integrated into the clinical context for interpretation. Comparison to the concentration in blood is strongly recommended.				UHN

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Canada

Patient Name	Sex	Date of Birth (mm/dd/yyyy)
FAQIRI, SOLEIMAN	M	01/01/1986

Order ID
42716358

Client's File No.
R2016-02587/A281316

Health Number

ICL Login Date (mm/dd/yyyy)
12/23/2016 1:38PM

Report Printed
12/23/2016 6:53PM

Authorized Requester
Bellis, Maggie, Dr.

Sodium, Fluid

Sample ID: UHN161223027
Collection Date (mm/dd/yyyy)
12/16/2016 12:00PM

Final - Received 12/23/2016 6:53PM

Order Choice Unable to perform testing, viscosity too high
Comments:

TEST	RESULT	FLAG	NORMAL/THERAPEUTIC RANGE	UNITS	TEST SITE
FLUID TYPE	Not provided				UHN
SODIUM-FLUID	NOT REPORTED			mmol/L	UHN
Tech Comment (fluid)	The reference range and other method performance specifications have not been established for this body fluid. The test result must be integrated into the clinical context for interpretation. Comparison to the concentration in blood is strongly recommended.				UHN

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Patient Name	Sex	Date of Birth (mm/dd/yyyy)
FAQIRI, SOLEIMAN	M	01/01/1986

Client's File No.
R2016-02587/A281316

Order ID
42716358

Health Number

ICL Login Date (mm/dd/yyyy)
12/23/2016 1:38PM

Report Printed
12/23/2016 6:53PM

Authorized Requester
Bellis, Maggie, Dr.

Urea, Fluid

Sample ID: UHN161223027
Collection Date (mm/dd/yyyy)
12/16/2016 12:00PM

Final - Received 12/23/2016 6:53PM

Order Choice Unable to perform testing, viscosity too high
Comments:

TEST	RESULT	FLAG	NORMAL/THERAPEUTIC RANGE	UNITS	TEST SITE
FLUID TYPE	Not provided				UHN
UREA-FLUID	NOT REPORTED			mmol/L	UHN
Tech Comment (fluid)	The reference range and other method performance specifications have not been established for this body fluid. The test result must be integrated into the clinical context for interpretation. Comparison to the concentration in blood is strongly recommended.				UHN

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