A forensic hypothesis for the mystery of al-Hasan’s death in the 7th century: Mercury(I) chloride intoxication

Nicole Burke¹, Mitchell Golas¹, Cyrus L. Raafat² and Aliyar Mousavi¹

Abstract
The puzzle of a mysterious death in the Middle Ages has been hypothesized in terms of contemporary forensic legal and scientific methods. That al-Hasan ibn-‘Ali died in 669 aged just 45 has been forensically analyzed based on written sources that dictate eyewitness accounts of historical events. The report of the contemporaneous poisoning of another individual who resided under the same household as al-Hasan’s and experienced similar, yet non-lethal, symptoms has served as the beginning of the analysis. In light of ancient (medieval) documents and through using mineralogical, medical, and chemical facts, it has been hypothesized that mineral calomel (mercury(I) chloride, Hg₂Cl₂) from a certain region in the Byzantine Empire (present-day western Turkey) was the substance primarily responsible for the murder of al-Hasan.

Keywords
al-Hasan, calomel, intoxication, mercury(I) chloride, mystery

Introduction
A mysterious death in the Middle Ages is that of al-Hasan ibn-‘Ali. Born of the Prophet Muhammad’s favorite daughter, Fatimah, al-Hasan was declared as the legitimate successor of his father, ‘Ali ibn-abi-Talib (the last of the caliphs known to Arab historians as “orthodox”),¹ in 661.² Faced by his rival, Mu’awiyah ibn-abi-Sufyan, who had been proclaimed caliph in 660 in Jerusalem,³ al-Hasan abdicated in the same year and reasoned as follows: “I have deemed it right to make peace with him and have pledged allegiance to him, since I considered whatever spares blood as better than whatever causes it to be shed.”³ After living in retirement in al-Madinah (in present-day Saudi Arabia) for eight years,¹ al-Hasan died in 669,² when he was just 45 years of age.¹ The belief that al-Hasan died peacefully² has not been ruled out by all the experts on Muslim history. However, in general, Muslim theologians commonly believe that his death was caused by a fatal act of poisoning.

With autopsy information unavailable, historical documents are the only available evidence in order to investigate cases such as al-Hasan’s death scientifically. A few traditions,⁴ such as the one containing the following quote, mention that when al-Hasan was about to die, he was asked by his younger brother¹ al-Husayn to identify his poisoner but refused to do so (as he wanted no innocent person to be falsely accused and killed):

If he/she [the poisoner] is not [i.e., not the one whom I suspect], I would like no innocent person to be killed because of me.

Still, the following tradition⁴ addresses two concurrent acts of poisoning, which resulted in two victims: al-Hasan and a survivor.

Ja’dah daughter of al-Ash’ath ibn-Qays al-Kindi poisoned al-Hasan ibn-‘Ali, peace be upon both of them, and poisoned a freedwoman of his; however, the freedwoman of his vomited the poison while al-Hasan kept it in his stomach. Then he was wrecked by it and died.

This tradition says that a freedwoman of al-Hasan who had also been poisoned “vomited the poison”

¹Science and Engineering Technology Department, Nashua Community College, USA
²Golden Gate University School of Law, USA

Corresponding author:
Aliyar Mousavi, Science and Engineering Technology Department, Nashua Community College, 505 Amherst Street, Nashua, NH 03063, USA.
Email: amousavi@ccsnh.edu
and survived, which means that she could have served as forensic evidence for the murder of al-Hasan. But are there any historical reports in which the poison is qualitatively described and can lead to a forensic hypothesis for the murder of al-Hasan?

“Gold filings” or mineral calomel?

That al-Hasan was offered a poisoned drink by his wife Ja’dah is reported in both Shiite\(^4\) and Sunni\(^5\) sources. Since intoxicating drinks, such as wine, are not allowed in Islamic law, the drink itself must have been a nonalcoholic drink. According to one tradition,\(^4\) a yogurt drink was used. One tradition\(^4\) describes the poison that was given to al-Hasan as follows: “It has been said that he was given gold filings to drink.” However, how likely is that from a toxicological perspective? Elemental gold is relatively inert, and an important use for it is in dentistry. It dissolves in concentrated hydrochloric acid if a strong oxidizing agent is present (e.g., in a 3:1 mixture of concentrated hydrochloric acid and concentrated nitric acid).\(^6\) The hydrochloric acid in the human stomach is neither concentrated nor in the presence of a strong oxidizing agent. What could the true identity of the toxic substance that al-Hasan had ingested in the drink have been if the powdered solid phase of the substance only looked like gold filings? In order to answer this question accurately, it is useful to know the geographic source of the poison.

The tradition referred to in the Introduction, which says that a freedwoman of al-Hasan had also been poisoned, suggests that the plot to poison al-Hasan was because of some harem jealousy. However, Madelung\(^3\) says that al-Hasan’s “pursuit of women was not more covetous than that of most of his class.” It is logically appropriate to ask if the murder of al-Hasan could have had a political motive force behind it. Although al-Hasan abdicated, in the process of surrendering the reign, he stipulated that his rival “should not be entitled to appoint his successor but that there should be an electoral council.”\(^3\) Still, when the caliph died in 680,\(^2\) he had already “nominated his own son Yazid as his successor”\(^1\) and caused homage to be paid to him.\(^2\) There are reports, accepted by both Shiite sources and several major Sunni historians, stating that the poisoning of al-Hasan by Ja’dah was at the instigation of the caliph.\(^3\) A very specific report\(^4\) says that in order to eliminate al-Hasan, the caliph, whose empire’s capital was Damascus,\(^1\) wrote to the Byzantine emperor and asked him for a poisoned drink, which the emperor, despite refusing at the beginning, sent conditionally. The mention of the conditionality of the emperor’s agreement in this report is consistent with the hostility of Arab–Byzantine relations in 669 (the year al-Hasan died), when Byzantium had an energetic emperor, Constantine IV.\(^1\)

The mainland of the Byzantine Empire in 668 (about one year before al-Hasan’s death) was approximately present-day Turkey (Figure 1). In present-day western Turkey, there are more than 50 mines that contain minerals with deposits of mercury.\(^7\) Mercury is isolated from its main ore, cinnabar (mercury(II) sulfide, HgS), and was used in the...
Mediterranean world for extracting metals by amalgamation as early as 500 BC. The element does not have any known biological functions and has a long history of toxic effects. Mercury(II) chloride (HgCl₂), for instance, which was probably first made by Arabic alchemists in the 10th century, was widely used as a violent poison in the Middle Ages. Do the mercury mines in western Turkey contain any mercury species that look like gold?

Although abandoned since the 1990s, Türkönü and Haliköy are two important mercury mine locations in Turkey. The Haliköy mine exists in an area made up of metamorphic rocks, including gneiss and schist. Cinnabar and metacinnabar contain mercury and are found in the Haliköy fault. Mine locations also contain deposits of pyrite, marcasite, chalcopyrite, arsenopyrite, quartz, and calcite. The mineral calomel (mercury(I) chloride, Hg₂Cl₂) is found as a secondary mineral in oxidized zones along with cinnabar, calcite, and limonite. Calomel can present as a yellow gold crust (Figure 2) and forms as tetragonal crystals presenting in a variety of formations, including tabular, prismatic, and pyramidal.

The environmental assessments performed at the mine locations in Turkey have utilized computer programs to aid in analysis of the soil and water samples. The software programs Aquachem and PHREEQC determined that water samples taken near the Haliköy mine presented with oversaturation with calomel, as well as quartz and cinnabar.

The symptoms in historical reports compared to those of mercury intoxication caused by ingesting calomel

Without an autopsy or any diagnostic testing, it is impossible to diagnose with certainty what poisoned al-Hasan. However, we can hypothesize the type of poison used by the symptoms described in the traditions. Some clinical symptoms described by traditions of al-Hasan’s death are a green coloring of the skin and vomiting of blood. These are also symptoms that can be caused by the ingestion of calomel and the resulting mercury intoxication.

Al-Hasan was noted to have had a green hue to his skin. This could occur due to kidney damage caused by calomel intoxication. Calomel acts on the kidneys and causes renal tube necrosis and can lead to acute kidney failure. The green coloring is due to a type of anemia called hypochromic anemia, also called chlorosis or green sickness. This presents with pallor, a known side effect of calomel poisoning. This type of anemia is caused by a lack of hemoglobin, the oxygen binding protein responsible for nourishing our body tissues with oxygen. Hemoglobin is found within the erythrocytes or red blood cells, and a low erythrocyte count is the most common cause of hypochromic anemia. If acute renal failure occurred, erythropoietin, the hormone that signals hemiopoetic stem cells in the bone marrow to have a cell fate of becoming erythrocytes, would not be present due to damage of the proximal renal tubule. The promixal renal tubule is the area in which the red blood cell mass or hematocrit is determined by the kidneys. It stands to reason that if the hematocrit level could not be correctly sensed due to renal tube necrosis, then the signal that is normally sent to the cortical labyrinth to make erythropoietin would be unlikely to occur. The lack of this hormone would prevent new erythrocytes from being made, which in turn would result in a lower hemoglobin level, and a person affected by this would present with an ashen appearance, sometimes said to appear green.

Al-Hasan was said to have vomited blood. The corrosive effects of calomel on the alimentary or gastrointestinal tract are known to cause damage and irritation of the esophagus. The traditions also mention that he was vomiting up his “kabid,” an Arabic word that means “liver” and also means “interior.” If ulceration were to occur in the esophagus, a certain extent of bleeding should be expected. When human blood reaches the stomach, it is broken down and can coagulate, very often resulting in vomiting a substance that has the appearance of coffee grounds. Fresh red blood could also be present with bleeding in the esophagus. This occurrence of vomiting blood is referred to as hematemesis and generally occurs due to esophagitis, bleeding ulcers, and certain cancers. Considering that the understanding of the human body in the 7th century was less extensive, the “commoner’s Arabic expression” to refer to vomited blood clots would have been “kabid.” In fact, in some rural areas in the Arab World, it is still said, “My liver is torn and thrust away (kabidi mutaqatti’ mat‘un).” In several traditions, al-Hasan says that he is vomiting pieces of his “kabid,” which

Figure 2. Calomel from Mariposa Mine, Texas, USA. (Rob Lavinsky, The Arkenstone, www.iRocks.com, is credited for the image.)
could have been coagulated blood in the emesis, resembling pieces of his liver and/or internal organs due to the coffee-ground color and texture. Liver damage is another noted effect of mercury intoxication by calomel, which is due to the accumulation of deposits of mercury via the hepatic portal vein system. The liver is a known filtering organ that has capillaries that are sinusoid. This capillary type has the largest pores and is the only type that allows exchange of red blood cells through their pores. Red blood cells have susceptibility of the attachment of “corrosive sublimate” (mercury(II) chloride, \( \text{HgCl}_2 \)) by way of a sulfhydryl attachment. “Corrosive sublimate” can also be transported by the blood. The liver is one of the areas where red blood cells are destroyed when they are damaged, which would allow for more mercury accumulation than the filtering of blood alone.

Aqueous calomel ingested by the human body is converted into “corrosive sublimate,” which is known to have much more harmful effects than calomel alone. After ingestion, aqueous calomel (\( \text{Hg}_2\text{Cl}_2 \)) is converted to “corrosive sublimate” (\( \text{HgCl}_2 \)), by oxidation in the hydrochloric acid (\( \text{HCl} \)) within the stomach. This reaction, which is shown by Equation (1), goes to completion, as the calculated equilibrium constant for it, at the normal human body temperature (37°C), is \( K_{eq} = 2.4 \times 10^{19} \).

\[
2\text{Hg}_2\text{Cl}_2(aq) + 4\text{HCl}(aq) + \text{O}_2(g) \rightarrow 4\text{HgCl}_2(aq) + 2\text{H}_2\text{O}(l) \quad (1)
\]

Calomel absorption into the body tissues is not seen in large quantities. However, it is enough to cause the anemia mentioned previously. Effects of “corrosive sublimate,” such as intestinal ulceration, are thought to increase the amount absorbed due to the breakdown of protective barriers such as mucous membranes, which prevent absorption before damage. Noting that in the traditions al-Hasan refers to being poisoned at least three times, if the initial poisoning had started this damage, the subsequent poisonings would have allowed a much greater amount of absorption of calomel or “corrosive sublimate.” Therefore, subsequent poisonings will allow for greater accumulation of mercury within the tissues.

In later centuries, for example, when there was an outbreak of syphilis in Europe at the end of the 15th century, calomel was routinely ingested as a medicine. Why could the calomel in al-Hasan’s drink have been lethal?

1. The exact number of times al-Hasan was poisoned is not historically known. After the final poisoning, according to a couple of traditions, he says that he has been poisoned three times, and according to a few other traditions, he conveys the information that he has been poisoned at least four times. If the latter is the case, the number of times he was given calomel could have been too many to survive.
2. According to one tradition, the final poisoning was when al-Hasan was breaking his fast (Islamic fast, which includes refraining from eating and drinking from dawn to dusk) on a hot day. If that is the case, it will naturally lead to the conclusion that he ingested a large quantity of the drink containing aqueous calomel, which is “completely” converted to “corrosive sublimate” in the human stomach according to Equation (1).
3. The same net ionic equation as that of Equation (1) (Equation (2)) may be written for the reaction between any acids in the “to-be-ingested” nonalcoholic drink and the aqueous calomel in the drink, leading to the formation of mercury(II), which is the mercury in “corrosive sublimate.” A couple of examples of common Arabic nonalcoholic drinks in the 7th century are honey water and yogurt drink, which both contain acids.

\[
2\text{Hg}_2^{2+}(aq) + 4\text{H}^+(aq) + \text{O}_2(g) \rightarrow 4\text{Hg}^{2+}(aq) + 2\text{H}_2\text{O}(l) \quad (2)
\]

With the reaction shown by Equation (2) in the “to-be-ingested” nonalcoholic drink, al-Hasan has ingested some aqueous mercury(II) cation, in addition to the ingested aqueous calomel, which is converted to “corrosive sublimate” later (in the human stomach, according to Equation (1)).
4. It is possible that the mineral calomel that was mixed with the drink was naturally accompanied by some other toxic substance(s).

**Conclusions**

Records of a green hue to al-Hasan’s skin, which could occur due to acute renal failure, concurrent with his vomiting of blood and, apparently, coagulated blood in the emesis are decisively consistent with those of calomel intoxication. Mineral calomel can present as a yellow gold crust (Figure 2), which can, in the powdered solid phase, be mistaken for “gold filings” (the identity of the poison according to a certain tradition). The effect of al-Hasan’s poisoned freedwoman’s recorded experience seems to indicate that contemporaneously to al-Hasan’s death, both people suffered the same affliction while residing under a common household. This, in light of several historical sources, points at al-Hasan’s wife Ja’dah as the prime suspect.

A considerable number of calomel-containing mercury mines exist in present-day western Turkey, which was in the heart of the Byzantine Empire around the time of al-Hasan’s death. The caliph sought to
nominate his own son as his successor (when the caliph died, he had done so) and had caused homage to be paid to the son, who actually succeeded him). Al-Hasan was obtrusively an obstruction to that goal, because that was despite the caliph’s official guarantee of al-Hasan’s stipulation that he (the caliph) “should not be entitled to appoint his successor.” As often happens in political dynasties, assassination becomes expedient. The report of the caliph’s solicitation of the Byzantine emperor by writing a letter asking the emperor for a poisoned drink is a clear example of his intent to act in furtherance of the plot, and, according to the report, the emperor’s final response was a positive and deliberate agreement to that end.

The fact that neither an autopsy nor a judicial investigation was performed by the authorities at the time should not deter the use in evidence of eyewitness accounts. Mineralogical, medical, and chemical facts support the hypothesis that al-Hasan’s death was caused by calomel (mercury(I) chloride) intoxication. This forensic hypothesis is consistent with the historical position, reflected in ancient (medieval) documents, that al-Hasan was poisoned by Ja’dah, at the instigation of the caliph, and with the Byzantine emperor’s involvement.

Acknowledgments
Aliyar Mousavi would like to thank the following individuals, for the following contributions:

1. Ms. Nada Kherbik, teacher of Arabic at Albuquerque Academy, for her assistance in the comprehension of certain Arabic texts used for this article.
2. Ms. Wafaa Alhutmany, project manager at the Arab World for Medical Services, for her linguistic research and input.
3. Dr. Ramezan P. Dowlati, professor of psychology at Northern Virginia Community College, for his constructive comments.

Conflict of interest
The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding
This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

References
1. Hitti PK. History of the Arabs, 10th ed. New York: St. Martin’s Press, 1974.
2. Spuler B. The age of the caliphs, 1st ed. Princeton, NJ: Markus Wiener, 1999.
3. Madelung W. The succession to Muhammad, 1st ed. New York: Cambridge University Press, 1997.
4. Al-Majlisi MB. Bihar al-Anwar, Volume 44, Chapter 22. 1st ed. Beirut: Al-Wafaa’ Foundation [in Arabic], 1983.
5. Bursali MN. Peygamber Çiçekleri Hz. Hasan ve Hz. Hüseyin, 1st ed. Istanbul: Sultan Yayınevi [in Turkish], 2000.
6. Greenwood NN and Earnshaw A. Chemistry of the elements, 1st ed. Tarrytown, NY: Pergamon Press, 1994.
7. Karahalil B, Ulukaya M and Alp O. Pilot study of environmental monitoring of Konya region near abandoned mercury mine in Turkey. Bull Environ Contam Toxicol 2012; 88: 150–153.
8. Petrisor IG. Mercury—hazards and forensic perspectives. Environ Forensics 2006; 7: 289–292.
9. McAuliffe CA (ed). The chemistry of mercury. 1st ed. London: Macmillan, 1977.
10. Gemici U and Tarcan G. Assessment of the pollutants in farming soils and waters around untreated abandoned Türkönu mercury mine (Turkey). Bull Environ Contam Toxicol 2007; 79: 20–24.
11. Gemici U, Tarcan G, Somay M, et al. Factors controlling the element distribution in farming soils and water around the abandoned Halıköy mercury mine (Beydağ, Turkey). Appl Geochem 2009; 24: 1908–1917.
12. Bonewitz RL. Rock and gem, 1st ed. New York: DK, 2008.
13. Bernhoft RA. Mercury toxicity and treatment: a review of the literature. J Environ Public Health 2012; 2012: Article ID 460508.
14. Material Safety Data Sheet: Mercury(I) Chloride. www.lewisu.edu/academics/biology/pdf/ Mercurous_chloride.pdf (accessed 7 March 2015).
15. Ali MAM. The hypochromic anemias. Can Fam Physician 1976; 22: 42–46.
16. Donelley S. Why is erythropoietin made in the kidney? The kidney functions as a critimeter. Am J Kidney Dis 2001; 38: 415–425.
17. Louden I. The diseases called chlorosis. Psychol Med 1984; 14: 27–36.
18. Davis LE. Unregulated poisons still cause mercury poisoning. West J Med 2000; 173: 19.
19. Mayo Clinic. Vomiting blood, www.mayoclinic.org/symptoms/vomiting-blood/basics/definition/sym-20050732 (accessed 25 March 2015).
20. Mousavi A. Mercury(I) chloride in vivo oxidation: A thermodynamic study. Main Group Met Chem 2015; 38(3–4): 121–124.