Prevalence of HIV and syphilis among Turkish blood donors

To the Editor: Screening of blood is now mandatory for many diseases and is undertaken routinely in blood banks. Many studies have been done on human immunodeficiency virus (HIV) and syphilis, separately, but knowledge about the interrelationship between these transfusion transmitted diseases is limited.[1,2] This study was undertaken to assess the correlation between positivity for HIV infection and syphilis among blood donors of Eskisehir. Donors who applied to our blood center in a 10-year period (1998-2007) were retrospectively evaluated. No professional or honorary donation was included.

Serum samples were screened for anti-HIV by ELISA (microparticle enzyme immunoassay, Axym, Abbott Corp., IL, USA) and for syphilis by the Veneral Disease Research Laboratory (VDRL) test (nontreponemal test, immunotrep Omega Diagnostic, Scotland, UK). Samples were not screened for VDRL. Of the total of 19630 individuals, 6850 (34%) were females and 12780 (66%) were males. VDRL positivity was found in 33 donors (0.168%) and anti-HIV was positive in 3 cases (0.015%) (Table 1). When the VDRL test was positive, a confirmatory treponemal test was done. Anti-HIV positivity was also confirmed by the western blot test. For the statistical analyses, chi-square and the Fisher exact test were used.

The prevalence of VDRL reactivity varied from 0.03% to 0.3% in blood donors in different regions of Turkey.[3] VDRL reactivty increased from 0.1% in first period to 0.3% in the second period (P=.001). This seems to be an alarming signal in the local blood banks for the probable increase in syphilis and further diagnostic tests should be applied in these cases. According to the results reported from other regions of Turkey, anti-HIV positivity rates ranged between 0% and 0.66%.[4] Anti-HIV positivity was not found in the first period , but in the second period it was 0.03% (P=.105). Two donors with positive HIV serology (66.6%) were also positive for VDRL (P≤.001). The problem of safe blood has become an issue worldwide; there is no available method to reduce the infection risk from transfusion to zero. Thus, it appears to be essential to carefully select appropriate donors and to avoid unnecessary transfusion. In conclusion, blood donors in our region show lower seropositivity rates, although there seems to be a regular increase in the rates of anti-HIV and syphilis. Thus, taking into consideration the rising prevalence of these infections, a routine screening of all the donated blood products for anti-HIV and syphilis should be done, which will assist blood transfusion services in improving transfusion safety.

Table 1. Seropositivity rates in blood donors with VDRL and anti-HIV tests during two 4-year periods.

	1998-2002 (n=10362)		2003-2007 (n=9268)	
	VDRL (+)	Anti-HIV (+)	VDRL (+)	Anti-HIV (+)
Female	1	-	5	-
Male	7	-	20	3
Total	**8**	**-**	**25**	**3**

Omer Coskun,[a] Cem Gul,[a] Hakan Erdem,[a] Orhan Bedir,[b] Can Polat Eyigun[a]

From the [a]Department of Infection Disease and Clinic Microbiology and [b]Department of Microbiology, Gulhane Military Medical Academy, 06018 Ankara, Turkey

Correspondence and reprints:
Omer Coskun
Gulhane Military Medical Academy
Department of Infection Disease and Clinic Microbiology
06018 Ankara, Turkey
T: +90-312-304-4308
F: +90-312-304-4300
coskunomer23@hotmail.com

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Where were Avicenna and Rhazes from?

To the Editor: I read with interest the reply of Dr. Farid S. Haddad to the Letter of Dr. Farrokh Habibzadeh.[1] I found that the composition of the reply is somewhat incongruent insofar as the first part is aggressive and attacks the commentator (Dr. Habibzadeh), while the second part is more pacifistic, advising universalism and preaching the tenets of a "science sans frontier."

I, for one, believe that the best answer is the one that has already been written by Dr. Habibzadeh. I would only add that in the last 22 years, I had three publications on the nationality of Avicenna (Poure-Sina) and Mohammad Zakaria Razi and documented that they were all thor-

oughbred-Iranian[2-4] as Abu-Nowas and Al-Mutanabi were Arabs. Even the fair Arab investigator, Professor Azeem Majeed, clearly admitted the fact that Iran was one of the sources of Islamic science[5] and stated that "as Islam spreads out of the Arabian Peninsula into Syria, Egypt and Iran, it met long established civilizations and centers of learning."

One point which should be clarified is the wrong statement of Dr. Haddad that "how could *ibn sÿna*, who died 1000 years before 'Iran' was scripted on a map, be Iranian? I have yet to see a work by *ibn sÿna* or by *alrazÿ* written in Persian." If one work is enough, I advise Dr. Haddad to study the excellent book of "*Daneshnameh Aalaie*" by Poure-Sina and "Dar Amadi bar Elmeh Pezeshki" (an introduction to medicine) by Mohammad Zakaria Razi (according to Ibn Abi Usaybi'ah)[6,7] both written originally in Persian.

Concerning the name, "Iran" was firmly put on the map over a thousand years ago by Ferdowsi, one of the greatest world poets, who created the masterpiece of Shahnameh where he mentioned the name of this country as "Iran" several times. Accidentally, Ferdowsi and Poure-Sina were contemporary. To convince Dr. Haddad, I quote Professor Majeed that "Arab scholars translated philosophical and scientific works from Greek, Syriac, Pahlavi (the scholarly language of pre-Islamic Iran) and Sanskrit into Arabic."[5]

Back to the anatomy charts presented by Dr. Haddad, I want to emphasize that the two plates presented so far, are from a treatise written by Mansouri Shirazi. The number of these charts as mentioned in his book is limited to five.[8] The language of that book is Persian and the work has not yet been translated to any other languages.

Dr. Behrooz Broumand

Iran University of Medical Sciences
Iranian Academy of Medical Sciences
T: +98-21-8872-1140
F: +98-21-8872-1141
broumand@ams.ac.ir

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Reply

In response to the query of Professor Doctor Behrooz Broumand: "Where was Avicenna and Rhazes from?" I say: the first was born in the village of Afshana near Bukhara and the second was born in Raÿÿ in the province of Fars. Bukhara is in present-day Uzbekistan, hence a few people maintain that Avicenna was Uzbekÿ and others think he is Russian! In order to economize space and time, and to cut short a polemic that seems to have no end, I refer the reader to a few of my published works on Avicenna and on Rhazes.[1-22]

If we return to our main topic, the anatomy charts, of which there are over seventy scattered around the world in various manuscripts. I have been trying to decipher their enigma for over 20 years without much success. We are planning, and hopefully we can achieve the main purpose of our plan; which is to publish most of these charts so that they reside in the public domain at the disposal of researchers who may be interested to decipher some of the following secrets: who is the author of these charts? What is the relation of one set to the other? What is their medical and historical importance, if any?

It is my hope that other readers might help in the solution of this puzzle rather than bring a dim set of chauvinistic glasses and sidestep the worthy endeavor.

Dr. Farid S. Haddad

6409 Vista Pacifica
Rancho Palos Verdes
CA 90275
T: +310 541 0435
haddadmd@cox.net

Male hypogonadism in a patient with Cogan syndrome

To the Editor: Cogan syndrome is a rare, rheumatic disease characterized by inflammation of the ears and eyes that may also be associated with vasculitis in other areas of the body.[1] Lunardi and colleagues advanced identification of the eye, inner ear, nervous system and endothelium autoantigens of Cogan syndrome with their finding that the isolated 12-residue Cogan peptide is homologous to four autoantigens: laminin, connexin 26, the collagen disease-associated Ssa/Ro, and the receptor-like phosphatase DEP-1/CD148, and one viral protein.[2] The condition can lead to visual difficulty, hearing loss and dizziness. It is fatal in less than 10% of patients. Mild disease may be treated with anti-inflammatory medications, including steroids and nonsteroidal anti-inflammatory drugs.[1]

The age-related decrease in testosterone and potential hypogonadism may result in decreased libido and erectile dysfunction, loss of muscle mass and strength, weight gain, and declining cognitive function.[3-5] Men on long-term corticosteroid therapy often have low testosterone levels.[6] Androgen receptor levels are regulated by androgens, other steroids and non-steroidal hormones.[7] Therefore, we thought it would be interesting to highlight a case of male hypogonadism in a patient with Cogan syndrome.

A 47-year-old male with increasing loss of sexual drive, general lack of enthusiasm, and frequent exhaustion presented to our department because of loss of libido and erectile dysfunction for one year. He was divorced without children. Four years previously, a diagnosis of Cogan syndrome was made based on a bilateral progressive hearing loss, tinnitus and ataxia, as well as a bilateral interstitial keratitis following a viral infection of the upper airways. He had previously been treated with methylprednisolone for approximately 4 years with a maintenance dose of 4 mg daily. Physical examination revealed sparse secondary hair, muscle atrophy, a normal prostate and testis with a volume of 8 mL on both sides. All baseline hematological, biochemical and additional hormone parameters where within normal values except for total cholesterol 9.3 mmol/L (normal range, 0.0.-5.0 mmol/L), HDL cholesterol 1.2 mmol/L (normal range, 1.0.-5.0 mmol/L), LDL cholesterol 6.7 mmol/L (normal range, 0.0.-3.0 mmol/L) and triglyceride 3.1 mmol/L (normal range, 0.0.-1.7 mmol/L). His total testosterone levels were 8.4 and 13.5 nmol/L (normal range, 10.5-49 nmol/L), free testosterone 170 pmol/L (normal range, 174-900 pmol/L), luteinizing hormone 15.7 U/L (normal range, 1.5-5.0 U/L), follicle-stimulating hormone 38.9 U/L (normal range, 1-10.5 U/L). A semen analysis showed azoospermia. Chromosome analysis excluded abnormalities, so the karyotype was 46,XY. Testicular biopsy confirmed marked hyalinization and thickening of the basement membrane of the spermatic tubules. Color duplex sonography of the aorta and intraabdominal vessels excluded severe vascular manifestations of Cogan syndrome. Osteodensitometry revealed a normal bone density for age. Therapy with intramuscular injections of testosterone enanthate 250 mg was started every 3 weeks and atorvastatin 20 mg daily was added. At last review, lipids were well controlled. Corticosteroids have a catabolic effect, often intensified by the androgen deficiency. Contrary to the fact that long-term therapy with corticosteroids can induce secondary hypogonadism our patient developed primary hypogonadism. Testicular biopsy excluded vasculitis as an underlying pathological mechanism. Our case is a clear example of a rare systemic autoimmune disease that requires a broad multidisciplinary approach to optimize its treatment and improve the patient's well-being.

Maja Radman,[a] Ognjen Kraus,[b] Miljenko Solter[c]

From the [a]Department of Endocrinology, University Hospital Centre Split, Soltanska 1, Split 21000, Croatia, [b]Department of Urology, and the [c]Department of Endocrinology, University Hospital Sestre Milosrdnice, Vinogradska c. 29, Zagreb 10000, Croatia

Correspondence and reprints:
Maja Radman, MD
Department of Endocrinology
University Hospital Centre Split
Soltanska 1, Split 21000, Croatia
T: +385-2-155-7400
maja.radman1@st.htnet.hr

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Anovestibular fistula with imperforate anus in two adults

To the Editor: Imperforate anus occurs in one of 2500 to 5000 live births.[1] It is reported that the majority of girls with imperforate anus will have a fistula to the perineum, fourchette, or vestibule.[1] Anovestibular fistula (AVF) is the most common form of anorectal anomaly in female infants.[2] Treatment of these lesions can be by a variety of techniques including anal transposition, or posterior or anterior sagittal anorectoplasty in the neonatal period.[2-4] These procedures can be safely performed with or without a diverting colostomy. To our knowledge, only two cases of AVF presenting in adulthood have been described in the literature.[5] Posterior and anterior sagittal anorectoplasty were performed in these cases.[5] We report anal transposition for AVF for the first time in two adults.

An 18-year-old female patient was referred to our institution with the chief complaint of defecating from the vulvovaginal

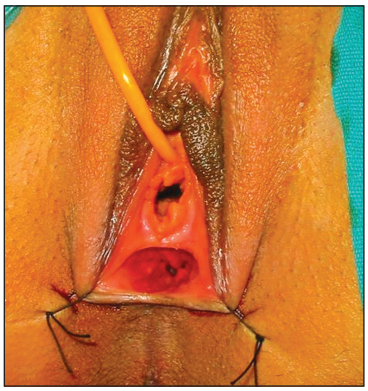

Figure 1. Preoperative image of patient.

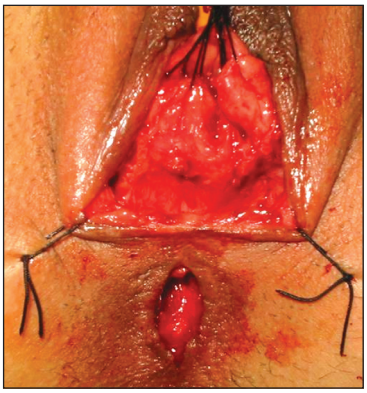

Figure 2. Dissection step for anal transposition procedure.

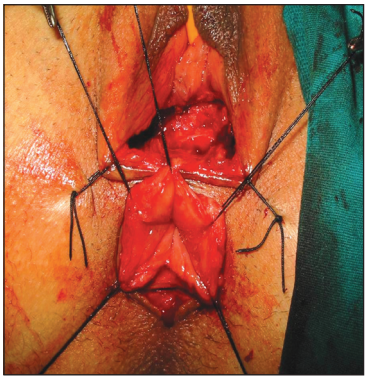

Figure 3. Pull-through step of fistula in to neoanus.

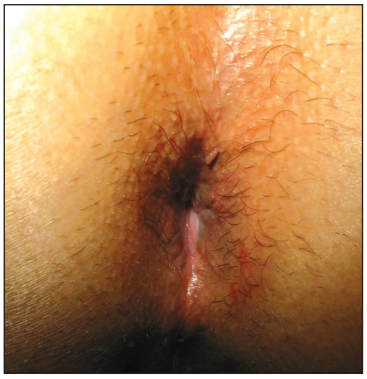

Figure 4. Postoperative images of patient 2 years after operation.

vestibule. The family recognized the abnormal place of defecation at birth, but no attempt was done for the management of this pathology. There was also a history of intermittent constipation but no history suggestive of intestinal obstruction. However, the patient denied a history of frequent urogenital infections. On physical examination, the patient was found to have imperforate anus and AVF (Figure 1). No additional urogynecologic abnormality was found on detailed physical examination. An abdominopelvic ultrasound did not reveal any abnormality of the urogenital system. Anal transposition without colostomy was performed in the gynecologic position under general anesthesia (Figure 2,3). She had an uneventful postoperative recovery and was discharged on the seventh postoperative day. She was well doing on the 33-month follow-up without any complication. The Cleveland Clinic Incontinence Score (CCIS) was 5 (good continence). She had no complaints about her social life after marriage.

The second case was a 19-year-old female patient referred to our institution with the complaint of defecating from a vulvovaginal vestibule and constipation. Admission for treatment was due to the decision of marriage. The medical history and complaints of the patient were similar to the first case. On physical examination, she was diagnosed with an imperforate anus and AVF. No additional urogynecologic abnormality was found on a detailed physical examination or on abdominopelvic ultrasound. Laboratory and physical investigations showed no other systemic pathologies. An anal transposition procedure without colostomy was performed in the gynecologic position under general anesthesia. She had an uneventful postoperative period and was discharged on the sixth postoperative day. She was doing well on 31-month follow-up with satisfactory continence and perineal cosmesis without any complication. Postoperative infection dehiscence and fistula recurrence did not occur, hence no secondary surgery was necessary. The CCIS scale was 7 (good continence). The patient did not consent to providing information about her social life after marriage.

The number of untreated adult patients with anorectal malformations is so few that only case reports appear in the literature. For this reason, there is no consensus on the surgical management of these adult cases. On the contrary, various operative techniques have been reported for the treatment of patients in the neonatal period with this abnormality. Large case series with various operative techniques have been published in infants. Currently, posterior sagittal anorectoplasty (PSARP) defined by Alberto Pena in 1982, is the most popular and frequently preferred surgical technique for neonatal period. Vijay et al. reported two adult cases with AVF. They performed limited ASARP in one patient and PSARP in the other.5 We performed anal transposition without colostomy in the treatment of both cases. Colostomy may not be necessary in adult patients in the case of optimal fecal passage. Colostomy is a poorly tolerated procedure in adult

patients compared with neonatal patients. For this reason, surgical interventions without colostomy is probably a better surgical option in adults with AVF. Single staged anal transposition without colostomy as a minor, safe operation may be the technique of choice for treatment of adults with imperforate anus and anovestibular fistula with satisfactory continence and perineal cosmesis (Figure 4).

Alpaslan Terzi,[a] Ali Coskun,[b] Fahrettin Yildiz,[a] Sacit Coban,[a] Omer Faruk Akinci[c]

From [a]Harran University, Faculty of Medicine, Department of General Surgery, Sanliurfa, Turke, [b]Kecioren Training and Research Hospital, Department of General Surgery, Ankara, and [c]Umraniye Training and Research Hospital, Department of General Surgery, Istanbul
Correspondence and reprints:
Assist. Prof. Dr. Alpaslan Terzi
Harran University School of Medicine
Department of General Surgery
63300 Sanliurfa -Turkey
T: +90-414-314-1170
F: +90-414-315-1181
aterzi@harran.edu.tr

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erratum

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Comparison of the 2005 growth charts for Saudi children and adolescents to the 2000 CDC growth charts

On page 339, the following acknowledgement should have appeared:

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