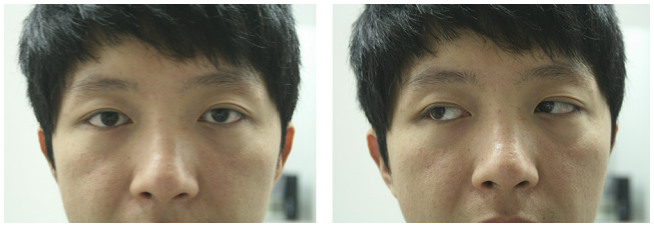

FIGURE 4. After 4 months of follow-up, the patient presented an improvement of ocular movement on the lateral gaze.

CN VI lies medially and is not protected, so that CN VI is often the first cranial nerve to be involved.[1–3] While spontaneous CCFs (indirect shunts) present initial clinical features within months due to low-flow fistulas, the first onset of traumatic CCF, which is formed by a tear in the carotid artery in cavernous sinus and caused high-flow shunts, usually appears within hours.[1,2,4] Untreated CCFs can lead to visual loss, subarachnoid hemorrhage, glaucoma, cataract formation, neurologic deficit, seizure, and fatal epistaxis.[2,3] Fortunately, the overall mortality of CCF is quite low; only 3% of the cases had intracranial hemorrhage.[2]

The gold standard in diagnosis of CCF is 4-vessel digital subtraction cerebral angiography, but CT with contrast is also quite useful, especially in finding engorged superior ophthalmic veins, a common radiographic sign in CCF (Fig. 4).[2]

Treatment of CCF has changed dramatically with the evolution of interventional radiography.[5–7] Fistula is occluded while preserving the patency of internal carotid artery. Definitive treatment of CCF is occlusion of fistula using an endovascular coiling or a detachable balloon to maintain the patency of the internal carotid artery. Successful embolization of CCF will result in immediate resolution of proptosis, chemosis, and bruits, but ophthalmoplegia and optic nerve dysfunction may take 4 months to resolve.[2]

Although traumatic CCF usually manifests symptoms early after trauma, in this case the patient presented clinical signs 8 weeks postinjury, while the longest time that was acknowledged in another previously released article was 6 weeks. After the treatment, the patient experienced good recovery. Recently, the cases of craniofacial trauma are tremendously increasing. To rule out the risk of CCF, according to this case, surgeons should recommend careful follow-up for patients with skull base fracture until 3 months at least.

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Anterior Rhinomanometry and Determination of Nasal Mucociliary Clearance Time With the Saccharin Test in Children With Crimean-Congo Hemorrhagic Fever

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Objectives: Crimean-Congo hemorrhagic fever (CCHF), like other viral infections, may prolong mucociliary clearance time and increase nasal resistance in children. The aim of the present prospective case-control study was to study, using saccharin and anterior rhinomanometry tests, whether CCHF infections caused any change in nasal physiology.

Methods: Overall, 40 subjects, 20 of whom had CCHF (group 1) and 20 of whom were healthy controls (group 2), were enrolled in this study. The definitive diagnosis of CCHF infection was made based on typical clinical and epidemiological findings and detection of CCHF virus-specific IgM by ELISA or of genomic segments of the CCHF virus by reverse transcription-polymerase chain reaction. Anterior rhinomanometry was performed in all participants according to current recommendations of the Committee Report on Standardization of Rhinomanometry. A saccharin test was used to evaluate mucociliary clearance, and nasal mucociliary clearance time was assessed with the saccharin test as described previously.

Results: In our patients, the mean time from the application of saccharin crystals to the first feeling of a sweet taste was 6.77 ± 3.25 minutes (range 2–16 min). In terms of the mean time from the application of saccharin crystals to the first feeling of a sweet taste, there was no difference between two groups. The mean total air flow was 637.60 ± 76.18 mL/s (range 490–760 mL/s). The mean total nasal airway resistance was 0.24 ± 0.03 Pa/mL s (range 0.20–0.31 Pa/mL s). In terms of the degree of nasal air flow and nasal airway resistance and the total air flow and total nasal airway resistance of each nostril, there was no difference between the 2 groups.

Conclusions: The results obtained in anterior rhinomanometry and saccharin test showed that there was no statistically significant difference between CCHF (+) patients and controls. These results suggest us that CCHF virus infection does not affect nasal physiology. However, this is the first study performed on this issue and further studies on larger series need to be performed.

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Key Words: Crimean-Congo hemorrhagic fever, anterior rhinomanometry, saccharin test, nasal mucociliary clearance time, nasal physiology

Crimean-Congo hemorrhagic fever (CCHF), caused by Crimean-Congo hemorrhagic fever virus (CCHFV), is a viral zoonotic disease with a high mortality rate in humans. In CCHF patients, there are clinical findings frequently mimicking influenza during the 1–7 days term before hemorrhage. Although the pathogenesis of the disease has not been clearly identified yet, the most important step has been thought to be endothelial infection. Through proliferation in endothelial cells or through the effects of cytokine, chemokine, and other mediators released from mononuclear cells, endothelial activation and a disturbance in barrier function occur.[1]

Nasal congestion or obstruction is one of the most frequent symptoms encountered in primary care and specialist clinics, and it is often the predominant symptom in upper respiratory tract disorders, such as allergic rhinitis, rhinosinusitis, nonallergic rhinitis, and nasal polyposis.[2] Rhinomanometry can be used for clinical evaluation of the symptom of nasal obstruction, objective assessment of nasal airflow, research in nasal physiology, allergy challenge testing, preoperative and post-treatment assessments of surgical or medical therapy, and for evaluation of patients with sleep apnea.[3,4]

Nasal mucociliary activity is both a good indicator of nasal mucosal functions and one of the important defense mechanisms of the body. Although many methods have been described to evaluate nasal mucociliary clearance, saccharin test is frequently preferred as it is the easiest, cheapest, and reliable method.[5]

CCHF, like other viral infections, may prolong mucociliary clearance time and increase nasal resistance in children. In the present study, the aim was to study, through nasal mucociliary clearance time (NMCC) with saccharin test and anterior rhinomanometry, whether similar changes in nasal physiology occur in children with CCHF.

METHODS

Study Design

This prospective study was carried out at the Department of Otorhinolaryngology and Pediatrics at Cumhuriyet University Hospital in the city of Sivas located in central Anatolia, Turkey, between April 2010 and 2011. The study was performed in patients hospitalized in the Pediatric Department. Twenty subjects having laboratory-confirmed diagnosis of CCHF (group 1) and 20 healthy control subjects (group 2) were included in the present study.

The Human Ethics Committee of Cumhuriyet University approved this study in accordance with the Helsinki declaration. Patients were included in the study only after having given their informed written consent.

Patients

Forty subjects, 20 [8 girls (40%) and 12 boys (60%), aged between 7 and 16 years] of whom had CCHF (group 1) and 20 [12 girls (60%) and 8 boys (40%), aged between 7 and 16 years] of whom were healthy controls (group 2), were enrolled in this prospective case-control study. Acute and convalescent phase serum samples were sent to the virology laboratory of Refik Saydam Hygiene Center in Ankara, Turkey for serological and virological analyses. The definitive diagnosis of CCHF infection was made based on typical clinical and epidemiological findings and the detection of CCHF virus-specific IgM by ELISA or of genomic segments of the CCHF virus by reverse transcription-polymerase chain reaction (RT-PCR) either in the acute and convalescent phase of the disease.[1]

The rhinoscopic examination was performed by the same physician (EEA) at baseline. Exclusion criteria of the study were having any of the following: (1) vasomotor rhinitis, (2) allergic rhinitis, (3) previous septoplasty, (4) chronic sinusitis, (5) sinonasal malignancy, (6) nasal valve collapse, and (7) adenoid hypertrophy.

Rhinomanometry

Anterior rhinomanometry was performed in all participants according to current recommendations of the Committee Report on Standardization of Rhinomanometry.[6] To analyze the degree of nasal airway obstruction, nasal air flow (mL/s) and nasal airway resistance (expressed in Pa/mL s) were measured by anterior rhinomanometry (2006, ZAN Messgerate GmbH, Germany) of each nostril, calculated at a pressure of 150 Pa, with merging of values from the left and right nostril.

All tests were made between 16:00 and the 17:30. Because physical exercise is known to exert a vasoconstrictive effect, the children were kept at rest in the sitting position for approximately 15 minutes before starting the test.[6–8]

Determination of Nasal Mucociliary Clearance Time (NMCC) With the Saccharin Test

A saccharin test was applied for estimation of mucociliary clearance and NMCC time was assessed with the saccharin test as described previously.[9] The results were expressed as NMCC time, which negatively reflects the efficiency of NMCC and is normally between 20 and 60 minutes.[9] An hour before the saccharin test, patients were asked to clean their nasal secretions (mechanically using water) and not to consume anything. Patients were instructed not to sniff, attempt to clear their nose, or sneeze during the test.

A 5-mg particle of saccharin measuring approximately 0.5 mm in diameter was placed on the medial aspect of the inferior turbinate of 1 nasal cavity under direct visualization using a headlamp and a nasal speculum. After correct particle placement was visually confirmed, a stopwatch was started and the total time was recorded. The total time (in minutes) from application of saccharin crystals on the anterior edge of inferior nasal conchae to first feeling of sweet taste was a measure of the mucociliary clearance. Thirty minutes was taken as a higher limit of normal values. If the subject was unable to taste any sweetness after 30 minutes, an additional particle of saccharin was placed on the anterior aspect of the subject's tongue to exclude taste loss. In all determinations, the subjects had intact taste.

Statistical Methods

Statistical Package for Social Science (SPSS) 14.0 software program (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Demographic data were stated as numbers and percentages while the quantitative data were presented as mean ± standard deviation. The chi-square test and Mann-Whitney U test were used to examine the association between variables. A P value <0.05 was considered to be statistically significant.

RESULTS

Overall, 40 subjects, 20 [8 girls (40%) and 12 boys (60%), aged between 7 and 16 years] of whom had CCHF (group 1) and 20 [12 girls (60%) and 8 boys (40%), aged between 7 and 16 years] of whom were healthy controls (group 2), were enrolled in this prospective case-control study. Both groups were similar in terms of gender ($P = 0.206$, $P > 0.05$).

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TABLE 1. Selected Demographic, Clinical, and Laboratory Data of 20 Patients With Crimean-Congo Hemorrhagic Fever

Mean Age (Range)	n (%)
Sex, n (%)	
Girl	12 (60%)
Boy	8 (40%)
Most common symptoms, n (%)	
Myalgia	5 (25%)
Headache	8 (40%)
Fever	19 (95%)
Sore throat	18 (90%)
Physical finding, n (%)	
Fever[a]	15 (75%)
Conjunctival hyperemia	7 (35%)
Rash	0 (0%)
Bleeding	2 (10%)
Laboratory features	
Thrombocytopenia[b]	18 (90%)
Leucopenia[c]	17 (85%)
Elevated AST, IU/L	11 (55%)
Elevated ALT, IU/L	11 (55%)
Long PTT, s	6 (30%)

AST indicates aspartate aminotransferase; ALT, alanine aminotransferase; aPTT, activated partial thromboplastin time.
[a]Arm pit, ≥38°C.
[b]Thrombocytopenia, platelet count $<150 \times 10^9$.
[c]Leukopenia, leukocyte count $<4 \times 10^3$.

Some of the demographic, clinical, and laboratory data of these 20 patients with CCHF are presented in Table 1. Fifteen (75%) of the 20 patientshad positive CCHF virus-specific IgM antibodies, 11/20 (55%) had positive RT-PCR test for CCHF virus, and 9/20 (45%) were positive in both tests during the acute and/or convalescent phase of the disease. In all patients, serological markers for viral hepatitis A, B, and C; human immunodeficiency virus; cytomegalovirus; toxoplasma; and Epstein-Barr virus were all negative. Tube agglutination assays for *Brucella* spp. were also negative. In our patients, the most common symptom was fever (95%) while the most common laboratory finding was thrombocytopenia (90%).

The overall mean age was 12.33 ± 2.76 years (range 7–16 years). The mean age was 12.45 ± 2.52 (range 7–16 years) in group 1 and 12.20 ± 3.03 (range 7–16 years) in group 2. There was no difference between the 2 groups ($P = 0.838$, $P > 0.05$).

The mean anterior rhinomanometry and saccharin test time after hospitalization was 5.75 ± 1.83 days (range 3–10 days) in group 1.

Among a total of 40 patients, the mean time from the application of saccharin crystals to the first feeling of a sweet taste was 6.77 ± 3.25 minutes (range 2–16 min). The mean time from the application of saccharin crystals to the first feeling of a sweet taste by the patients was 7.39 ± 4.30 minutes (range 2-16 min) in group 1 and 6.14 ± 1.54 minutes (range 3.30–8 min) in group 2. There was no difference between the 2 groups ($P = 0.414$, $P > 0.05$).

The mean nasal air flow was 314.18 ± 32.93 mL/s (range 251–368 mL/s) in the left nasal cavity, 319.38 ± 42.77 mL/s (range 238–391 mL/s) in the right nasal cavity, and mean total air flow was 637.60 ± 76.18 mL/s (range 490–760 mL/s). The mean nasal airway resistance was 0.48 ± 0.54 Pa/mL s (range 0.41–0.60 Pa/mL s) in the left nasal cavity, 0.48 ± 0.07 Pa/mL s (range 0.38–0.63 Pa/mL s) in the right nasal cavity, and mean total nasal airway resistance was 0.24 ± 0.03 Pa/mL s (range 0.20–0.31 Pa/mL s). Anterior rhinomanometry results according to the groups are presented in Table 2.

DISCUSSION

We studied nasal mucociliary clearance by means of the saccharin test, and clinical evaluation of the symptom of nasal obstruction was made by rhinomanometry in patients with CCHF in pediatric age. The results were compared with clearance measurements in healthy control patients. This is the first report investigating the effect of CCHF and nasal physiology. We could not find any study investigating CCHF and change in nasal physiology in the literature. Thus, we were unable to compare our results with another study. However, the results of the present study showed that there were no statistically significant differences between the CCHF (+) patients and controls. It may be related with the fact that our study group was relatively small in terms of numbers of patients and controls.

Mucociliary clearance is a primary defense mechanism of the upper and lower airways, and disruption of this process, whether acquired or inherited, predisposes an individual to acute and chronic nasal, paranasal sinus, and airway infections.[10] In light of this data, it is possible to hypothesize that the factors responsible for reduced mucociliary functions include decreased ciliated epithelium.[11] Impaired mucociliary clearance may increase susceptibility to upper and lower respiratory tract infections due to prolonged swelling of infectious agents on the respiratory mucosa and due to the presence of factors that hinder ability to prevent microbial colonization and in invasion. A wide range of normal mucociliary clearance times have been reported in the literature.[12] Passali and Bianchini Ciampoli found values up to 9.96 minutes in healthy children whereas the mean time discovered by other studies ranged between 4.4 minutes and 7.5 minutes.[13]

Pedersen et al[14] investigated nasal mucociliary clearance transport rate. They measured (saccharin test) 26 subjects with naturally acquired common colds and found that the transport rate was markedly reduced during the disease. Sakakura[15] investigated, using chicken and Newcastle disease virus, the relationship between the mucociliary function and acute upper respiratory infection, and found that the average transit times were significantly longer than those of controls.

Using a nasal saccharin transport test, Milgrim et al[16] investigated a mechanism underlying the increased incidence of sinusitis, that of prolonged mucociliary transport time in 30 HIV-infected patients and 30 matched, non-HIV controls. Their results supported an inherent delay of mucociliary clearance in HIV-infected patients which is chronic, possibly irreversible, and, in association with nasal obstruction, represents a major mechanism of both the high acute and recurrent sinusitis rate in their population.

Rosen et al[17] investigated the effects of HIV infection on mucociliary clearance time, and they found prolonged mucociliary clearance

TABLE 2. Patients' Anterior Rhinomanometry Results

	Group, n	Mean ± SD	*P*
Nasal air flow left	Group 1 (n = 20)	318.00 ± 27.60	0.616
	Group 2 (n = 20)	310.35 ± 37.85	
Nasal air flow right	Group 1 (n = 20)	324.20 ± 36.36	0.620
	Group 2 (n = 20)	314.55 ± 48.83	
Total air flow	Group 1 (n = 20)	645.50 ± 63.99	0.704
	Group 2 (n = 20)	629.70 ± 87.68	
Nasal airway resistance left	Group 1 (n = 20)	0.48 ± 0.04	0.673
	Group 2 (n = 20)	0.49 ± 0.06	
Nasal airway resistance right	Group 1 (n = 20)	0.47 ± 0.06	0.664
	Group 2 (n = 20)	0.49 ± 0.08	
Total nasal airway resistance	Group 1 (n = 20)	0.23 ± 0.02	0.632
	Group 2 (n = 20)	0.24 ± 0.04	

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time and more sinonasal symptoms in HIV-positive patients.

In the present study, we were thinking that NMCC time could be prolonged in CCHF cases as in HIV-positive patients and as in those having common cold. However, the results we obtained showed that NMCC time was 7.39 ± 4.30 minutes in CCHF cases while it was 6.14 ± 1.54 minutes in healthy cases in the control group. There was no statistically significant difference between the 2 groups. NMCC time of our CCHF cases were within the NMCC time interval stated by Passali and Bianchini Ciampoli[12] in a study on healthy children.

Our hypothesis is that CCHF, like other viral infections, may increase nasal symptoms and nasal resistance in children. Respiratory viral infections may worsen bronchial hyperreactivity. However, there is no data on the possible role of recurrent infectious rhinitis in nose hyperreactivity. The mechanisms that underlie an acute increase in nasal resistance are not well known.[18]

Inspiratory nasal airway resistance is 0.39 Pa/cm^3/s in nondecongested nose of a normal person (0.34–0.40 in average) while it is 0.26 Pa/cm^3/s after decongestion (0.25–0.30 in average).[19] Kobayashi et al[20] aimed at assessing the validity nasal resistance measurements produced using anterior active rhinomanometry by comparing the results with those of their previous study and determining normal value of the nasal resistance in Japanese elementary schoolchildren. They found that nasal resistance was 0.35 ± 0.16 Pa/cm^3/s in the normal group.

What is valuable in rhinomanometric measurements is the total nasal airway resistance and normal values that are between 0.12 and 0.33 Pa/mL s.[21] In our study, total nasal airway resistance was 0.23 ± 0.02 Pa/mL s in CCHF cases while it was 0.24 ± 0.04 Pa/mL s in healthy controls in the control group. There was no statistically significant difference between the 2 groups. Moreover, total nasal airway resistance of CCHF cases were within the normal values stated in the studies performed by Huizing and de Groot.[19,21]

CONCLUSIONS

In conclusion, the results we obtained by rhinomanometry and saccharin tests suggest that CCHFV infection does not affect nasal physiology. However, our study is the first one conducted on this subject and thus further studies on larger series need to be performed.

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Coil Embolization and Surgical Removal of Carotid Body Paraganglioma

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Abstract: Carotid body paraganglioma has considerable malignant potential and locally aggressive behavior, so it should be treated as soon as it is discovered.

We report the case of 60-year-old male patient with a carotid body paraganglioma (Shamblin group II) that was causing the carotid arteries to spread. Angiography showed 1 dominant feeding artery arising from the right external carotid artery. Selective angiography was performed 2 days before surgical removal of the tumor, and the feeding artery was successfully embolized with coils.

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