Specialist Hospital.[1] High prevalence rates of rotavirus infection have also been reported in other Saudi cities, 41.3% in Al-Taif and Jeddah.[2] Rotavirus infection also appears to be a major health problem all over the world. Prevalence rate of 15.3% in Iran,[3] 13.9% in Bahrain,[4] 42.3% in Indonesia,[5] 17.3% in France[6] and 6.8% in United State of America[7]. Since viral GE is not a disease of obligatory notification, the above variation in the prevalence rates of rotavirus infection appears to be dependent on the study design. In which this discrepancy may be explained by the difference in the duration of the stool samples collection for each study, whether the number of samples collected during year round or during the peak season. In addition to age dependence, the possibility of technical variation from laboratory to another, as well as the difference in the sensitivity of the methods used for the detection of rotavirus in each study may also contribute to the wide prevalence rates and should be carefully taken into consideration. In this study, the prevalence rate of rotavirus infection between male and female children was not significant. Similar results were reported in Iran[3] and Indonesia[5]. Also, we found a significant relationship between age and rotavirus infection. The highest incidence was in infants aged between 6-12 months, and the lowest was in the 5-12 year age group. This data clearly indicated that rotavirus infection acquired early in life. Therefore, older children become resistant to infection due to previous exposures.

The minimal number of rotavirus diarrhea cases observed in infants younger than 6 months might be attributed to maternal immunity, breast feeding or age-dependent physiological factors. When the seasonal distribution of rotavirus diarrhea was investigated, we found no significant seasonal variation exists. Rotavirus infection occurred all year round. This observation has been supported by other studies carried out in Riyadh.[1] However, our finding did not agree with that reported in Jeddah and AI-Taif,[2] which reported higher incidence of rotavirus infection in cooler and warmer months, respectively. Since the climate in Riyadh city is characterized by high temperature for most of the year, dryness and low amount of rain falls, the effect of these environmental factors on the prevalence of rotavirus infection appears to be minimal. In contrast to the high prevalence rate of rotavirus infection (25.4%), the incidence of enteric adenoviruses was 1.1% and 0.8% astroviruses. Several studies have similarly shown that enteric adenoviruses and astroviruses came second to rotaviruses as a cause of viral gastroenteritis.[6,7] Low prevalence rates of 5.2% and 6.8% have been reported with astroviruses infection in USA[7] and France[6]. Enteric adenoviruses were found in acute diarrheal cases in Bahrain (0.6%)[4], Indonesia (4%)[5] and KSA (5.3%). Although, the prevalence rates of adenoviruses (1.1%) and astroviruses (0.8%) reported in the above mentioned studies were higher, it is clearly supported our finding that adenoviruses and astroviruses are the secondary causes of viral gastroenteritis in children.

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Biochemical parameters and *Helicobacter pylori* seropositivity of patients with cerebral infarct

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H*elicobacter pylorus* (Hp) is strongly associated with duodenal ulceration, gastric ulceration, and gastric cancer. Although some small case-control studies have reported an association between Hp seropositivity and ischemic heart disease, there are only a few data on Hp seropositivity and stroke.[1] In

a pilot study, it was claimed that a significant relation existed between Hp infection and stroke.[2] The mechanism underlying an association between Hp and vascular disease is uncertain. Inflammatory processes have fundamental roles in stroke in both the etiology of ischemic cerebrovascular disease and the pathophysiology of cerebral ischemia.[3] We aimed to determine the prevalence of Hp seropositivity in patients with acute ischemic cerebrovascular disease.

Fifty consecutive patients with acute ischemic cerebrovascular disease (31 males, 19 females, aged 32-67 years, mean 60.02 ± 10.90 years), and as control group, 50 age and gender matched healthy hospital staff (31 male, 19 female, aged 30-65, mean 56.66 ± 5.66 years) were included in the study. Stroke was defined according to the World Health Organization criteria. During hospitalization, neurological evaluation was performed by the same neurologist. Patients with hypertension (reported systolic blood pressure ≥160 mm Hg, reported diastolic blood pressure ≥95 mm Hg,), diabetes mellitus (fasting serum glucose level ≥120 mg/dL), hepatic, renal or clotting disorders, patients with cardiac valve disease and arrhythmia, and current smokers were not included in the study. All blood samples were obtained between 07:30-08:30 am after a 12 hour fasting period. Blood samples were immediately centrifuged and separated after obtaining. Blood samples were collected in tubes containing citric acid and stored at -40ºC until assayed. Immunoglobulin G (IgG) antibodies to Hp were detected using a rapid enzyme linked immunosorbent assay (ELISA; PyloriStat, BioWhittacker, Walkersville, MD). Blood samples were assessed for hematocrit (Htc), hemoglobin (Hb), white blood cell (WBC), platelet, serum glucose, blood urea nitrogen, serum glutamate oxaloacetate transaminase, serum glutamate pyruvate transaminase, total cholesterol, high density lipoprotein (HDL), low density lipoprotein (LDL), very low density lipoprotein cholesterol (VLDL), fibrinogen, anti-thrombin 3 (AT 3) levels. Serology was performed blinded to case-control status. Tecan minilyser system that used an antibody concentration of 10 units/ml was taken as an indication of positive serology; the sensitivity when validated against diagnosis on samples taken at gastroscopy was 94% while specificity was 93%.

Brain computed tomography (CT) with Toshiba Xvision/GX spiral CT and, when necessary, magnetic resonance imaging (MRI), with Siemens Magnetom Expert 1.0. Tesla of brain were performed within 72 hours after onset of stroke in all patients. Cerebral infarction was diagnosed on the basis of results of the first CT scan, and when brain CT failed to demonstrate cerebral infarction (CT scans negative), MRI was used for diagnosis of cerebral infarction (n = 12). Statistical analysis was performed using Epi Info 200. The comparison of risk factors of cerebrovascular disease was determined using student-t test or 2 tests where appropriate, and subsequently with logistic regression analysis to control for other cerebrovascular risk factors. The relation between Hp positivity and biochemical and serologic parameters were evaluated using student's t test and subsequently multiple regression to control for other risk factors. Results are expressed as mean ± standard deviation (SD). A value of $p<0.05$ was accepted to be statistically significant. The Hp seropositivity was significantly higher in patients with acute ischemic cerebrovascular disease than control subject (90% versus 56.6%, odds ratio 7.07, 95% confidence interval 2.4-20.8, $p<0.05$). Total cholesterol, HDL-cholesterol, LDL-cholesterol, and VLDL- cholesterol, WBC, fibrinogen and AT3 levels of the patients were also higher than healthy control subjects ($p<0.05$). Biochemical results of patients and healthy controls and all serological results for Hp are shown in **Table 1** .We found that patients with acute cerebral ischemia had higher antibody titers against Hp than control subjects. Therefore, there may be an interaction between Hp infection and acute cerebral stroke. The main cause of acute cerebral stroke is atherosclerosis, and various mechanisms related with increasing effect of Hp on risk of arterial plague formation have been proposed.[4] One of the pathogenetic mechanisms

Table 1 - All biochemical results of patients and healthy controls.

Parameters	Patients (n=50)	Controls (n=50)	*p*
Age (year)	60.02 ± 10.90	56.66 ± 5.64	ns
Hb (g/dl)	13.032 ± 1.66	12.872 ± 0.85	ns
Htc (%)	39.8 ± 3.7	42.8 ± 3.8	ns
WBC (K/UL)	10.742 ± 4229	7551 ± 1687	.05
Plt (K/UL)	272 ± 81	290 ± 63	ns
Glucose (mg/dl)	94 ± 19	89 ± 9	ns
BUN (mg/dl)	38.4 ± 14.1	30.2 ± 7	ns
SGOT (u/L)	23.6 ± 11.1	27.7 ± 9.1	ns
SGPT (u/L)	27.7 ± 17.4	32.8 ± 12.1	ns
Total cholesterol (mg/dl)	183.6 ± 49.7	188.6 ± 35.8	ns
HDL-cholesterol (mg/dl)	36.6 ± 8.9	44.1 ± 5.5	.05
LDL-cholesterol (mg/dl)	121.8 ± 46.2	83.3 ± 14.6	.05
VLDL-cholesterol (mg/dl)	26.9 ± 12.3	48.9 ± 14.4	.05
Fibrinogen (mg/dl)	474 ± 106	397 ± 81	.03
AT 3 (%)	94.6 ± 14.7	87.3 ± 8.6	.03

ns - non-significant, Hb - hemoglobin, Htc - hematocrit, WBC - white blood cell, Plt - platelet, BUN - blood urea nitrogen, SGOT - serum glutamate oxaloacetate transaminase, SGPT - serum glutamate pyruvate transaminase, HDL - high density liprotein, LDL - low density lipoprotein, VLDL - very low density lipoprotein, AT 3 - anti-thrombin 3.

potentially linking chronic infection to ischemic stroke is a continuous low-grade inflammation that may stimulate procoagulant pathways and promote atherogenesis, for example, by activated mononuclear leukocytes. Chronic infection as a vascular risk factor could explain the association of leukocyte count, and fibrinogen with stroke and myocardial infarction and constituents of Hp were shown to activate leukocytes. Result of our study may support existence of direct and indirect effect of Hp on atherogenesis. We also found that WBC, fibrinogen, AT 3, HDL-cholesterol, LDL-cholesterol, and VLDL-cholesterol levels of the stroke patients were higher than healthy control subjects. Anthropological measurements are important and should be taken in this case. However, we could not determine anthropological measurements due to coma in the patient group. Some authors suggested that activation of leucocytes, promotion of leukocyte-endothelium interaction and conversion of local endothelium be a prothrombotic state.[5] Fibrinogen is an acute phase protein and its level strongly corresponds to the process of atherogenesis. Chronic Hp infection may enhance plasma fibrinogen concentration and, therefore, may increase blood viscosity and promote clot formation in Hp positive persons. There may be a possible interaction between Hp infection and acute cerebrovascular stroke. An Hp infection is a well-treatable condition, and its identification and eradication may be important on stroke prevention.

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Open bankart repair using suture anchors and capsular shift for recurrent anterior shoulder dislocation

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Instability of the shoulder is one of the most common disorders affecting the shoulder particularly in young active patients. The glenohumeral joint is more frequently dislocated than any other major joint in the body. The exact nature of the essential lesion of recurrent anterior instability is still not well understood. A common cause of the recurrent dislocation of the shoulder is the avulsion of the anterior capsular labrum complex (Bankart lesion), which is the most important stabilizer for the glenohumeral joint.[1] Various procedures have been described for the repair of the traumatic anterior dislocation of the shoulder, but the most popular surgical management is the Bankart repair (described by Bankart[2] in 1939), which is a reattachment of the capsule to the glenoid rim by placing drill holes through the bone. In 1991, Richmond et al[3] described their modification of the Bankart procedure using suture anchors. The open Bankart repair using suture anchors, and capsular shift for recurrent anterior dislocation of the shoulder is a predictable method of restoring shoulder stability in recurrent dislocation. The suture anchors facilitated the Bankart repair, decreased operative time, spared the articular surface, and allowed secure and quick fixation of the Bankart lesion to the anterior glenoid cavity, in comparison to the trans glenoid traditional sutures. The capsular shift technique adds to the stability without significant reduction of joint motion, mainly in external rotation. Open surgical stabilization of the shoulder has been the gold standard against which all other techniques have been compared. This is attributable to a success rate of 94-97%.[4] The success is defined as the absence of further complications of subluxation or dislocation, presence of stability, pain relief and a near-normal range of motion which will allow overhead function. The goal of surgical repair of anterior shoulder dislocation is to restore stability without compromising shoulder motion. Motion and function need not to be sacrificed in exchange for stability. The purpose of this study was to determine the efficacy and the results of open Bankart repair using suture anchors and capsular shift for the treatment of recurrent anterior dislocation of the shoulder.