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Modelling Human Immunodeficiency Virus cum Tuberculosis Co-infection Dynamics in Kwande-Nigeria: An Econometrics Time Series Approach

David Adugh Kuhe[1*] and James Serumun Ivande[1]

[1]Department of Mathematics/Statistics/Computer Science, Federal University of Agriculture, Makurdi, Benue State-Nigeria.

Authors' contributions

This work was carried out in collaboration between both authors. Author DAK designed the entire study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Author JSI contributes in the literature searches. Both authors read and approved the final manuscript.

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ABSTRACT

Aims: To model the dynamics of Human Immunodeficiency Virus (HIV) and Tuberculosis (TB) co-infection in Kwande region of Benue state in Nigeria using econometrics time series techniques.
Place and Duration of Study: The study is conducted in Kwande-Nigeria and utilizes monthly data of serologically confirmed cases of HIV and Tuberculosis infections from patients attending General hospital Adikpo in Kwande region from January 2007 to December 2016.
Methodology: The study employs Phillips-Perron (PP) unit root test, simple correlation and regression analysis, Engle-Granger cointegration test, error correction model (ECM) as well as pairwise Granger causality test as methods of analysis.
Results: Results showed that the series are integrated of order one, HIV and TB are found to be strongly and significantly positively correlated. HIV is found to have long lasting, positive and significant impact on TB and every HIV patient in the study area is found to have equal chance of developing TB. The study found a long-run stable relationship between HIV and TB. HIV is also

**Corresponding author: Email: davidkuhe@gmail.com;*

found to Granger caused TB in the study area.
Conclusion: The study recommends policies that would improve health promotion and education of the people, health awareness campaign of the public on HIV and TB be implemented in the study area and beyond, also the importance of HIV and TB screening prior to marriage contracts should be emphasized.

Keywords: HIV/AIDS; Mycobacterium tuberculosis; seropositivity; econometrics; Kwande; Nigeria.

1. INTRODUCTION

Human Immunodeficiency Virus (HIV) is an etiological agent that causes Acquired Immune Deficiency Syndrome (AIDS). When HIV enters the human body it damages the immune system to such an extent that many infectious diseases begin to occur as a result of the weakened immune system. HIV makes people more vulnerable to invasions by many opportunistic infectious agents including mycobacterium, the etiological agent responsible for tuberculosis [1]. Eventually, an individual acquires various illnesses due to the damage caused by the virus. AIDS is not a specific illness, it is rather a collection of many different conditions that manifest in the body or specific parts of the body because the HIV has so weakened the body's immune system that it can no longer fight the disease-causing agent that are constantly attacking it. We therefore define AIDS as a syndrome of opportunistic diseases, infections and certain cancers; each or all of which has the ability to kill the infected individual in the final stage of the disease [2].

HIV is exclusively found in the human blood, semen and vaginal fluid of an infected person and is transmitted from one person to another through unprotected Sexual intercourse (be it vaginal, anal or oral), blood transfusion, use of sharp unsterilized objects such as needles, syringes, razor/surgical blades, knives, barbing clippers, and from an infected mother to her baby during pregnancy delivery and after delivery during breast feeding [3]. According to [4] the signs and symptoms of HIV/AIDS include severe fever and headache, extreme and unexplained tiredness, rapid weight loss, recurring fever or profuse night sweats, prolonged swelling of the lymph glands in the armpits, groin, or neck, diarrhea that lasts for several weeks, sores of the mouth, anus, or genitals, pneumonia, red, brown, pink, or purplish blotches on or under the skin or inside the mouth, nose, or eyelids, memory loss, depression, and other neurologic disorders.

Tuberculosis on the other hand is an airborne bacterial disease and an opportunistic infection which is caused by tubercle bacilli called *Mycobacterium tuberculosis* and exclusively transmitted from an infected person to an uninfected person via spread of infectious droplets which are produced when someone with sputum smear positive tuberculosis of the lung. A healthy person acquired TB through interactions that involve sharing of a common closed environment with an infectious individual. After one is infected with TB, one stays infected for years and possibly latent-infected for life if not properly treated. *Tuberculosis* is one of the global leading causes of death among people living with HIV/AIDS [5]. More than four billion people of the world's total population are estimated to have infected with TB in the year 2014 [6].

There seem to be a long-run latent period relating to HIV infection and the onset of HIV-related diseases in adults. As the infection of HIV progresses, immunity weakens and patients begin to be more susceptible to common opportunistic infections. In many regions of the world today, HIV and TB treatments are more common and the use of drugs has drastically changed the co-infection dynamics of TB and HIV [1]. According [7] more than one-third of the 39.5 million people living with HIV worldwide are said to be co-infected with TB and more than 50% of the people infected with HIV are expected to develop TB. Many of the TB carriers who are living with HIV are more prone to develop active TB than those living without HIV. HIV replication rate tend to increase the presence of other opportunistic infections including TB for people who are already infected with HIV. This accelerates rapid progression of HIV to AIDS, the final stage of the disease [8].

In this study, we explore the potential implications on the co-infection dynamics of HIV and TB using applied econometric time series approach. The role of statistical and mathematical models in exploring epidemic interactions and disease co-infections in human population cannot be over emphasized. Long et al. [9] emphasizes the role of mathematical

modelling in guiding policymakers for resources allocation in the prevention and control of infectious diseases. [1] stressed that modelling TB and HIV co-infection dynamics pose serious mathematical challenges owing to their distinct models of transmission. Porco, Small and Blower [10] used a discrete event simulation model in predicting the effect of HIV on TB outbreaks. West and Thompson (1997) developed numerical simulations model and used it to estimate parameters of HIV and TB co-infection dynamics and predicted the future transmission of TB in the United States. Langat [11] used time series approach to model HIV and TB co-dynamics in Kenya. ARIMA(0,1,2) model was found to provide the best fit for HIV prevalence rate while ARIMA (1,2,0) model provided the best fit for TB case notification rate. The study found a long run equilibrium relationship between HIV prevalence and TB notification rates. HIV was also found to Granger caused TB. Lawna *et al.* [12] conducted a study to investigate the long term impact of TB on HIV among HIV-infected patients receiving HAART in South Africa; the study found that long-term HAART reduces risk in TB considerably. Several single-diseased dynamic models also exist in literature. A good number of them focus on TB transmission see for example [13,14,15,16,17,18,19,20] or on HIV/AIDS transmission dynamics see also [21, 22,23,24]. For more HIV and TB co-infection dynamic models see [25,26,10,27-30] for more surveys.

1.1 Background of the Study Area

This study is conducted in Kwande Local Government Area of Benue State in Nigeria. Kwande is one of the 23 local government areas of Benue State headquartered at Adikpo. It has an area of 2,891km^2 with a population of 248,697 people at the 2006 population census. It has an estimated population of 308,045 people in 2016. More than 75% of the Kwande population are farmers. As a result of its mountainous nature and proximity to the Cameroonian range of mountains, Kwande LGA usually has cold weather which makes it very conducive for farming, trading and investment. The local government has big rivers for domestic, agricultural and industrial needs. The crops grown in the local government include rice, yam, cassava, beniseed, beans, African yam beans, ground-nut, Barbara-nut, mangoes, oranges, pears, and palm-oil among others.

HIV/AIDS affects the most active and productive segment of the Kwande population, thereby threatening agricultural productivity and food security in the study area. Many children and elderly people now become head of households as a result of the epidemics. In addition, family members spend time, which could have otherwise been invested in agriculture to care for the sick and to attend funerals and mourn the dead. This diminishes the family's food availability, nutrition and well-being. Given their traditional responsibilities for agriculture and caring for the sick and dying, women and girls face the greatest burden of work. In many hard-hit households, girls are being withdrawn from school to help lighten the family burden. As earlier stated HIV damages the body's immune system and renders it defenceless against opportunistic infections including *tuberculosis*.

The increasing rates of tuberculosis infection in Kwande LGA of Benue state are posing serious challenges to the local government, individuals and international donor agencies and raise critical questions as to whether HIV is a significant contributor to the growth of the disease. In this study, our approach is slightly different from those found in the literature. However, we focus on the co-infection dynamics of HIV and TB in a common population and the technique does not exclude the possibility of joint infections. The study investigates whether HIV and TB have the same order of integration and whether the study variables share a common stochastic drift. A model is developed to explore the impact of HIV on the prevalence of TB among co-infected individuals in the study area. An error correction model which integrates the short-run and long-run co-infection dynamics is developed to estimate the speed of adjustment for disequilibrium correction. Finally the study employs pairwise Granger Causality test to investigate the causal relationship between HIV and TB in the study area.

2. MATERIALS AND METHODS

2.1 Source of Data

The data used in this study comprises of serologically confirmed cases of HIV and TB infection from patients attending General hospital Adikpo, Kwande Local Government area of Benue State-Nigeria from January 2007 to December 2016. The data consists of 120 monthly observations of persons believed to be indigenes of Kwande local government area.

2.2 Methods of Data Analysis

The following statistical tools are employed in the analysis of data in this research work using Eviews version 8.0.

2.2.1 Engle-granger cointegration test

Testing for cointegration using Engle-Granger approach involves three basic steps:

1. We pre-test the study variables for the presence of unit roots and check if the study variables have the same order of integration
2. We estimate the long run equilibrium regression model and obtain the residuals
3. We test to see whether the residuals are I(0).

2.2.2 The phillips-perron (PP) unit root test

Unit root test is conducted to check the stationarity or otherwise of a series. For this study [31] unit root testing procedure is employed. Phillips-Perron unit root test is a non-parametric method of controlling for serial correlation when testing for a unit root. The PP method estimates the non-augmented DF test equation

$$\Delta Y_t = \alpha Y_{t-1} + X_t^{'}\delta + \varepsilon_t \quad (1)$$

and modifies the $t-$ratio of the α coefficient so that serial correlation does not affect the asymptotic distribution of the test statistic. The PP test is based on the statistic:

$$\tilde{t}_\alpha = t_\alpha \left(\frac{\psi_0}{\phi_0}\right)^{1/2} - \frac{T(\phi_0 - \psi_0)(se(\hat{\alpha}))}{2\phi_0^{1/2} s} \quad (2)$$

Where $\hat{\alpha}$ is the estimate of α, and t_α the $t-$ratio of $\alpha, se(\hat{\alpha})$ is coefficient standard error, and s is the standard error of the test regression, ψ_0 is a consistent estimate of the error variance in (1) which is calculated as $(T-k)s^2/T$, where k is the number of regressors and ϕ_0 is an estimator of the residual spectrum at frequency zero.

2.2.3 Testing for cointegration

Here we conduct Augmented Dickey-Fuller (ADF) unit root test on the residuals of the series. The residuals used here are not the actual error terms, but estimated values from the long run equilibrium equation expressed as:

$$\Delta Y_t = \beta_1 \Delta X_t + \beta_2 \hat{\varepsilon}_{t-1} + \varepsilon_t \quad (3)$$

where $\hat{\varepsilon}_{t-1} = (Y_{t-1} - \varphi X_{t-1})$, ε_t should be I(0) if the variables Y_t and X_t are cointegrated. The residuals of equation (3) are then tested to see if they are stationary or non-stationary. Thus we estimate the following regression equation:

$$\hat{\varepsilon}_t = Y_t + \hat{\beta} X_t \quad (4)$$

Equation (4) simply checks for stationarity of residuals. The ADF test is given by

$$\Delta\hat{\varepsilon}_t = \rho\hat{\varepsilon}_{t-1} + u_t,\ u_t \sim \text{iid} \quad (5)$$

Engle and Granger [32] tabulated sets of critical values which are used to test for cointegration. The ADF test checks the following pairs of hypothesis: $H_0 = \rho = 1$ (no cointegration) versus $H_1 = \rho < 1$ (there is cointegration). A large ADF test statistic rejects the null hypothesis of no cointegration whereas a small ADF test statistic fails to reject the null hypothesis of no cointegration.

2.2.4 Pearson product moment correlation coefficient (r)

Pearson Correlation Coefficient (r) measures the correlation between two variables x and y. It is a numerical measure of the linear relationship between x and y and is defined as the ratio of the covariance between x and y, written as $Cov(x,y)$, to the product of the standard deviation of x and y. Symbolically

$$r = \frac{Cov(x,y)}{\sigma_x \sigma_y} = \frac{n\sum xy - (\sum x)(\sum y)}{\sqrt{[n\sum x^2 - (\sum x)^2]\left[n\sum y^2 - (\sum y)^2\right]}} \quad (6)$$

$$\sigma_x = \sqrt{\frac{\sum(x-\bar{x})^2}{n}} \text{ and } \sigma_y = \sqrt{\frac{\sum(y-\bar{y})^2}{n}} \quad (7)$$

where $\bar{x}$ is the Sample Mean of x, $\bar{y}$ is the Sample mean of y, σ_x is the Sample standard deviation of x and σ_y is the Sample Standard Deviation of y. Pearsonian correlation coefficient lies between -1 and $+1$, (i.e., $-1 \leq r \leq 1$).

2.2.5 Model specification for long-term relationship

To investigate the impact of HIV on TB, we employ a simple linear regression model using ordinary least squares (OLS). The model is specified as follows:

$$\ln TB = f[\ln HIV] \quad (8)$$

The natural log of TB is a function of the natural log of HIV. Our linear model is thus given by:

$$\ln TB_t = \beta_0 + \beta_1 \ln HIV_t + \varepsilon_t \quad (9)$$

where $\ln TB_t$ represents the natural log of tuberculosis at time t used as proxy for TB infection, $\ln HIV_{tt}$ represents natural log of Human Immunodeficiency Virus at time t and ε_t is the error term assumed to be normally and independently distributed with zero mean and constant variance, which captures all other explanatory variables that influence TB but are not captured in the model. β_0 is the intercept of the regression model which represents the predictive value of the dependent variable when the independent variable is kept constant. β_1 is the slope coefficient of the independent variable (HIV) that measure the impact of the explanatory variable on TB. For the independent variable (HIV) to have positive impact on TB the slope coefficient β_1 must be positive and significant.

2.2.6 Pairwise granger causality test

HIV is said to Granger-Cause TB if TB can be better predicted using the histories of both HIV and TB than it can by using the history of TB alone. We can test for the absence of Granger Causality by estimating the following vector Autoregressive (VAR) equation:

$$TB_t = a_0 + a_1 TB_{t-1} + \cdots + a_p TB_{t-p} + b_1 HIV_{t-1} + \cdots + b_p HIV_{t-p} + \varepsilon_t \quad (10)$$

$$HIV_t = c_0 + c_1 HIV_{t-1} + \cdots + c_p HIV_{t-p} + d_1 TB_{t-1} + \cdots + d_p TB_{t-p} + v_t \quad (11)$$

Then testing $H_0: b_1 = b_2 = \cdots = b_p = 0$ against $H_1: b_1 \neq b_2 \neq \cdots \neq b_p \neq 0$ is the test that HIV does not Granger-cause TB. Similarly $H_0: d_1 = d_2 = \cdots = d_p = 0$ against $H_1: d_1 \neq d_2 \neq \cdots \neq d_p \neq 0$ is a test that TB does not Granger cause HIV. In each case, a rejection of H_0 implies Granger causality.

3. RESULTS AND DISCUSSION

3.1 Phillips-Perron Unit Root Test Result

To investigate the unit root and stationarity characteristics of the study variables, we apply Phillips-Perron unit root test. The results of the test is presented in Table 1.

The PP unit root test results of Table 1 reveals that both HIV and TB infections are non-stationary in level. This is indicated by the PP test statistics been greater than the critical values of the test. The PP unit root test result is supported by the corresponding p-values. We can see that all the PP test values are insignificant at 1%, 5% and 10% significance levels. However, the PP test results of the first differenced series show evidence of stationarity as the PP test statistics for both HIV and TB infections are far less than the corresponding critical values of the tests and the p-values are highly statistical significance at all the conventional test sizes. The p-values of the test show how strong the null hypotheses of unit root in the series are rejected. We therefore conclude that the series are integrated of order one, I(1).

Table 1. Phillips-perron unit root test results

Variable	Option	PP test statistic	P-value	PP test critical value		
				1%	5%	10%
hiv	Intercept only	-1.9673	0.9876	-3.4866	-2.8861	-2.5799
	Intercept & trend	-2.6578	0.9543	-4.0377	-3.4483	-3.1493
Δhiv	Intercept only	-9.0831	0.0000	-3.4866	-2.8861	-2.5799
	Intercept & trend	-9.1921	0.0000	-4.0377	-3.4483	-3.1493
tb	Intercept only	-0.1287	0.7514	-3.4866	-2.8861	-2.5799
	Intercept & trend	-1.3126	0.5489	-4.0377	-3.4483	-3.1493
Δtb	Intercept only	-6.4332	0.0000	-3.4866	-2.8861	-2.5799
	Intercept & trend	-8.0190	0.0000	-4.0377	-3.4483	-3.1493

3.2 Pearson Correlation Coefficient Result

To investigate the degree of association between HIV and TB, we employ Pearson Correlation Coefficient. The result is presented in Table 2.

The Pearson moment correlation coefficient result shows that HIV and TB in Kwande LGA of Benue state are strongly and significantly positively correlated. This means that both variables move in tandem. That is, as the HIV infection increases TB infection also increases and vice versa.

3.3 Estimates of OLS Regression Model

To investigate the impact of HIV on TB in the study area, we employ OLS regression model. The result is presented in Table 3.

The result of Table 3 indicates that HIV is positively related to TB and statistically significant at 5% level. The coefficient of HIV indicates how much the dependent variable (TB) Varies with the independent variable (HIV). Thus for every one person increase in HIV infection, TB infection is predicted to increases by 0.799013 person (approximately 1 person) in Kwande LGA of Benue state. This result indicates that every HIV patient has an equal chance of developing TB in the study area. The value of the constant term is 99.59678 persons. This indicates that TB infection is predicted to be approximately 100 persons per month in the study area if HIV is completely eliminated in the LGA. This also means that HIV is not the only cause of TB in the study area.

The coefficient of determination (R^2) value of 0.799013 implies that 79.90% of the total variations in the dependent variable (TB) have been explained by the independent variable (HIV) while the remaining 20.10% unexplained variations are being accounted for by the error term or by factors not included in this model. The goodness of fit of the regression remained high after adjusting for degree of freedom as indicated by the adjusted R^2 (adj. R^2=0.544213 or 54.42%).

The F-statistic value 11.651082, which is the measure of the joint significance of the regression parameters, is found to be statistically significant at 5% level as indicated by the corresponding p-value of 0.0001328 indicating that the model is a good fit. Durbin Watson statistic value of 1.033118 is found to be greater than R^2 value and adjusted R^2 indicating that the regression model is non-spurious.

3.4 Engle-Granger Cointegration Test Result

To investigate the long-run stable equilibrium relationship between HIV and TB, we employ Engle-Granger cointegration testing procedure, the result is presented in Table 4.

Table 2. Pearson correlation coefficient of HIV and TB

Pearson correlation coefficient	Value	P-value
r	0.8704	0.0082**

*Note: **Correlation is significant at the 0.01 level (2-tailed).*

Table 3. OLS parameter estimates of regression model

Dependent variable: TB					
Variable	**Coefficient**	**Std. error**	**t-Statistic**		**P-value**
C	99.59678	10.85587	9.174461		0.0000
HIV	0.790504	0.061873	1.284944		0.0132
R-squared	0.799013	F-statistic	11.651082		
Adjusted R-squared	0.544213	Prob(F-statistic)	0.0001328	DW	1.033118

Table 4. ADF unit root test for cointegration

Variable	Option	ADF Test statistic	P-value*	ADF test critical value		
				1%	5%	10%
Residual (ε_t)	Intercept only	-6.3793	0.0000	-3.4861	-2.8859	-2.5798
	Intercept & trend	-7.8189	0.0000	-4.0370	-3.4480	-3.1491

*Note: *denotes [33] one-sided p-values.*

The ADF unit root test for cointegration result shown in Table 4 shows that the statistical hypothesis of no cointegration is rejected at 1% marginal level of significance. This means that there exist a long-run stable equilibrium relationship between HIV and TB co-infection. This is indicated by the ADF test statistics both with intercept only and with intercept and linear trend being less than the ADF test critical values and the significant p-values of the corresponding ADF test statistics. This means that HIV and TB are cointegrated and hence share a common stochastic drift. This shows that HIV and TB will not wander away from each other in the long-run but are bound to vary in sympathy with one another in the study area.

3.5 The Result of Error Correction Model

Using the residuals obtained from OLS regression equation in Table 3, we estimate the error correction model (ECM) which adjusts the speed of disequilibrium in the system. The result is presented in Table 5.

From the result of Error Correction Model presented in Table 5, the short run equilibrium coefficients are ΔTB(-1), ΔTB(-2), ΔHIV(-1) and Δ HIV(-2) whereas the long run equilibrium coefficient is EC(-1) which is called the error correction coefficient. The short run equilibrium coefficients are all statistically significant at lag one and lag two indicating that the impact of HIV on TB in the study area is long lasting.

The slope coefficient of EC(-1) is one period lag error correction term which guides the independent variables of the model to restore back to equilibrium or corrects disequilibrium. From the output of our estimates the slope coefficient of EC(-1) is negative and significant as expected indicating that system corrects its previous period disequilibrium at a speed of 44.62% monthly. This implies that the model have identified a speed of adjustment by 44.62% of disequilibrium correction monthly for attaining long run equilibrium steady state position.

3.6 Pairwise Granger Causality Test Result

Granger causality looks at the direction of causality between TB and HIV. This is necessary because of the strong economic contention that in some cases an increase in one variable may led to an increase in another variable but actually there may be no causality relationship existing between them. The result of Granger causality is reported in Table 6.

The pairwise Granger Causality test shown in Table 6 showed that a dependent relationship exists between TB and HIV infections. What this dependent relationship means is that TB does not Granger causes HIV but HIV Granger causes TB within the study period. The F-statistic in the first null hypothesis is less than 2 which indicates acceptance of the null hypothesis of no causation between TB and HIV, the p-value also confirmed this by given higher value which is greater than 0.05. The F-statistic in the second null hypothesis is greater than 2 which indicate rejection of the null hypothesis of no causation between the HIV and TB. The p-value confirms

Table 5. Parameter estimates of the error correction model

Dependent variable: ΔTB					
Variable	**Coefficient**	**Std. error**	**t-statistic**		**P-value**
C	-1.368052	3.206745	-0.426617		0.6705
ΔTB(-1)	-0.106478	0.107845	-0.987324		0.0256
ΔTB(-2)	0.153552	0.091536	1.677514		0.0269
ΔHIV(-1)	-0.101391	0.044651	-2.270733		0.0251
ΔHIV(-2)	-0.088001	0.045172	-1.948124		0.0359
EC(-1)	-0.446223	0.099865	-4.468249		0.0000
R-squared	0.798241	F-statistic	9.519810		
Adjusted R-squared	0.566913	Prob(F-statistic)	0.000000	DW	1.933365

Table 6. Granger causality test result

S/n	Null Hypothesis	Obs.	F-statistic	P-value
1	TB does not Granger Cause HIV	117	1.16177	0.3277
2	HIV does not Granger Cause TB	117	3.08633	0.0302*

*Note: * denotes the significant of the F-statistic and rejection of the null hypothesis*

this by given lower value which is less than 0.05. The implication is that TB is not the driving force behind the increased rates of HIV prevalence in the study area while HIV is found to have caused increased rates of TB in the study area.

4. CONCLUSION

This study has attempted to model the dynamics of HIV and TB co-infection in Kwande region of Benue state in Nigeria using econometrics time series models. The study utilizes monthly data of serologically confirmed cases of HIV and TB infection from patients attending General hospital Adikpo in Kwande region from January 2007 to December 2016. The study employs Phillips-Perron unit root test to investigate the stationarity characteristics of the study variables, simple correlation and regression analysis were employed to determine the degree of association and the impact of HIV on TB, Engle-Granger cointegration testing procedure was used to determine the long-run relationship between HIV and TB while error correction model as well as pairwise Granger causality test were employed to determine the speed of adjustment for disequilibrium correction as well as the direction of causality between HIV and TB. Results showed that the series are integrated of order one, HIV and TB are found to be strongly and significantly positively correlated. HIV is found to have long lasting, positive and significant impact on TB and every HIV patient in the study area is found to have equal chance of developing TB. The study found a long-run stable relationship between HIV and TB. The study has identified a speed of adjustment by 44.62% of disequilibrium correction monthly for attaining long run equilibrium steady state position. Finally, HIV is found to Granger caused TB in the study area. In the light of these findings, the study recommends policies that would improve health promotion and education of the people, health awareness campaign of the public on HIV and TB be implemented in the study area and beyond, also the importance of HIV and TB screening prior to marriage contracts should be emphasized.

CONSENT

All authors declare that written informed consent was obtained from the patient (or other approved parties) for publication of this paper and accompanying images.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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