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Improved detection of acute HIV-1 infection in sub-Saharan Africa: development of a risk score algorithm

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Abstract

Objective—Individuals with acute (preseroconversion) HIV infection (AHI) are important in the spread of HIV. The identification of AHI requires the detection of viral proteins or nucleic acids with techniques that are often unaffordable for routine use. To facilitate the efficient use of these tests, we sought to develop a risk score algorithm for identifying likely AHI cases and targeting the tests towards those individuals.

Design—A cross-sectional study of 1448 adults attending a sexually transmitted infections (STI) clinic in Malawi.

Methods—Using logistic regression, we identified risk behaviors, symptoms, HIV rapid test results, and STI syndromes that were predictive of AHI. We assigned a model-based score to each predictor and calculated a risk score for each participant.

Results—Twenty-one participants (1.45%) had AHI, 588 had established HIV infection, and 839 were HIV-negative. AHI was strongly associated with discordant rapid HIV tests and genital ulcer disease (GUD). The algorithm also included diarrhea, more than one sexual partner in 2 months, body ache, and fever. Corresponding predictor scores were 1 for fever, body ache, and more than one partner; 2 for diarrhea and GUD; and 4 for discordant rapid tests. A risk score of 2 or greater was 95.2% sensitive and 60.5% specific in detecting AHI.

Conclusion—Using this algorithm, we could identify 95% of AHI cases by performing nucleic acid or protein tests in only 40% of patients. Risk score algorithms could enable rapid, reliable AHI detection in resource-limited settings.

Keywords

acute HIV infection; detection; diagnosis; risk score algorithm; screening

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Introduction

Acute HIV infection (AHI), the 3–4-week period between HIV acquisition and antibody development [1,2], is an important target for HIV prevention, surveillance, and possibly treatment [1]. During AHI, transmission risk is high [3–5] as a result of ongoing high-risk behavior and elevated viral concentrations in the blood [2,6,7] and genital secretions [8]. Prevention efforts targeted at AHI can have a substantial public health impact by reducing this secondary transmission.

Acutely infected individuals cannot be identified with traditional antibody tests. Instead, HIV RNA or p24 antigen must be detected in the blood before antibody development [2]. Unfortunately, these tests are too expensive for routine use in many resource-limited settings.

Targeting HIV-RNA or p24 testing at individuals with an increased probability of AHI may be feasible. We developed a risk score algorithm for identifying probable cases of acute HIV-1 in a Malawian sexually transmitted infections (STI) clinic, using clinical, behavioral, and real-time laboratory-based predictors.

Methods

Study setting and population

The study population comprised adults seeking care at the outpatient STI clinic of Kamuzu Central Hospital in Lilongwe, Malawi, from February 2003 to October 2004 [9]. Eligibility criteria were: age greater than 17 years, antiretroviral inexperience, and willingness to provide written informed consent, to be HIV tested, and return for follow-up. The University of North Carolina School of Medicine Committee on the Protection of Human Subjects and the Malawi Health Sciences Research Committee Review Board approved the study.

Data and specimen collection

This cross-sectional analysis is restricted to the initial clinic visit. After obtaining informed consent, study staff provided HIV counseling, drew blood for HIV-1 testing, and verbally administered a brief questionnaire with items related to demographics, sexual history, medical history, and recognized AHI symptoms [10,11]. Clinical staff performed a physical examination, including a pelvic examination in women. Patients received treatment for STI according to the Malawi syndromic management guidelines [12].

We performed HIV-1 antibody testing according to the Malawi AIDS Counseling and Resource Organization scheme [12] using two parallel rapid HIV antibody tests: Determine (Abbott Laboratories, Abbott Park, Illinois, USA) and Unigold (Trinity Biotech, Wicklow, Ireland). We manually pooled plasma from patients with discordant or dual-negative rapid tests for HIV-RNA testing (Roche HIV-1 Amplicor Monitor, version 1.5; Pleasanton, California, USA), using a 50 : 5:1 pooling scheme described previously [9,13]. We used a standard kit (Bio-Rad Laboratories, Hercules, California, USA) to perform Western blot testing on all individuals with discordant rapid tests and detectable RNA, using World Health Organization criteria [14] to define a positive result.

Data processing and analysis

We entered questionnaire, physical examination, and HIV test data into duplicate Microsoft Access or Excel databases (Microsoft Corporation, Redmond, Washington, USA). We conducted statistical analyses with SAS 8.2 (SAS Institute, Inc., Cary, North Carolina, USA) and LogXact 4.1 (Cytel Inc., Cambridge, Massachusetts, USA).

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Participants with concordant-positive rapid tests or strongly positive Western blot were classified as having established (postacute) HIV-1 infection. Given our goal of developing an algorithm to distinguish between AHI and HIV-negative participants, participants with established HIV infection were excluded from analyses. HIV-negative status was defined as discordant or dual-negative rapid tests with undetectable HIV RNA. AHI was defined as detectable HIV RNA and either: (i) dual-negative rapid tests; (ii) discordant rapid tests and negative or indeterminate Western blot; or (iii) discordant rapid tests and weakly positive Western blot with subsequent band evolution. AHI was confirmed by repeated rapid tests and Western blots at weeks 1, 2, 4, 8, 12, and 16 after baseline.

To identify predictors of AHI, we calculated unadjusted prevalence odds ratios for each questionnaire and physical examination variable, with AHI as the outcome. Given the small number of AHI outcomes, we then constructed a separate logistic regression model in each of the following domains: (i) self-reported demographics, sexual history, and medical history; (ii) symptoms (previous month); and (iii) physical examination findings. Each model contained the six variables with the highest unadjusted prevalence odds ratios in the relevant domain [15]. We then conducted backward selection to create parsimonious domain-specific models, using likelihood ratio tests with a stopping rule of $P < 0.05$.

We constructed a full, combined model including discordant rapid test results and the variables from the reduced, domain-specific models. We conducted backward elimination to yield a final model, using a stopping rule of $P < 0.15$ to maintain predictive ability and reduce the likelihood of omitting important variables. We assessed model accuracy using the area under the receiver operating characteristic curves.

We assigned each variable in the final model a predictor score equal to its beta coefficient (natural log of the adjusted prevalence odds ratio), rounded to the nearest integer. We summed the predictor scores to obtain an easy-to-calculate risk score for each participant. In clinical implementation of the algorithm, all antibody-negative or discordant individuals with risk scores equal to or greater than a prespecified cut-off would be identified as likely AHI cases and selected for p24 or RNA testing. We calculated risk score sensitivity, specificity, and the percentage of patients who would be referred for p24 or RNA tests at each possible cut-off.

Results

Among 1450 participants, two were excluded because of missing questionnaires. Of the 1448 remaining participants, 588 (40.6%) had established HIV infection and were excluded from algorithm development. The final study population thus comprised 839 HIV-negative participants (97.6%) and 21 AHI cases (2.4%). Ages ranged from 18 to 60 years (HIV-negative median age 25 years; AHI median age 24 years), and 69% were men.

The six predictors with the largest unadjusted prevalence odds ratios in the self-reported behaviors/demographics domain were: multiple sex partners in the previous 2 months, any previous condom use, no education, receipt or provision of sex for payment in the previous 2 months, alcohol use at the last sex act, and receipt of medical injections in the previous 2 months (Table 1). STI history, marital status, and years in current residence were less strongly associated with AHI. The symptoms most strongly associated with AHI were diarrhea, fever, body ache, sore throat, night sweats, and weight loss. Nausea, stomach ache, cough, headache, and joint pain were less strongly associated with AHI status. Signs associated with AHI were genital ulcer disease (GUD); tender or swollen lymph nodes; tender, red, or swollen genitals; rash; genital warts; and genital discharge. Discordant rapid test results were very strongly associated with AHI (unadjusted prevalence odds ratio 29.5,

95% confidence interval 8.56–92.48). Among the AHI cases, 33% (7/21) had discordant rapid test results, compared with 2% (14/839) of HIV-negative participants.

Of the seven variables in the full, combined model (Table 1), all but one (tender or swollen lymph nodes) remained in the final model, which included: more than one sexual partner (previous 2 months), diarrhea (one month), fever (one month), body ache (one month), GUD, and discordant rapid test results. Corresponding adjusted prevalence odds ratios and predictor scores are shown in Table 1. The area under the receiver operating characteristic curves for the full and final combined models was 0.89.

Algorithm performance is shown in Figure 1. With a risk score cut-off of 2, only 40.9% of our population would be referred for RNA or p24 testing, and 95.2% of AHI cases would be detected. With a cut-off of 3, fewer patients (20.1%) would be referred for RNA or p24 testing; however, substantially fewer AHI cases (81.0%) would be identified. Given the negative consequences of missing AHI cases, a risk score cut-off of 2 appears to achieve the most favorable balance between sensitivity and testing. We note that all individuals with discordant rapid tests (predictor score 4) would be referred for RNA or p24 testing at this cut-off.

Discussion

AHI is an important target for public health interventions, because as many as half of all sexual transmission events may be attributable to index cases with acute or early HIV infection [5]. Furthermore, emergent anti-retroviral treatment of patients with AHI might help to prevent, slow, or reverse the immunological decline seen with HIV infection [16].

AHI screening in STI clinics has led to the detection of large numbers of AHI cases in short periods [17–19]. Although routine screening for HIV RNA or p24 is technically feasible, it has not been economically feasible in resource-constrained settings. A simple AHI risk algorithm could allow targeted AHI screening with RNA or p24 tests in selected individuals, thereby reducing costs and false positive results arising from these tests. In this study, we developed a rapid-assessment-based risk score algorithm that identified AHI cases with 95% sensitivity and 60% specificity in a Malawian STI clinic.

The performance of our algorithm compares favorably with other algorithms developed for similar purposes among sex workers in Kenya [20], high-risk individuals in the United States [21], and symptomatic, antibody-negative individuals in the United States [22]. The limited predictive power of these previous algorithms may have been related to a low incidence of HIV, to a high background prevalence of the particular predictors assessed, or to the sampling strategies of other studies. We used an efficient strategy for cross-sectional AHI screening among STI clinic attendees in an area with a high HIV incidence.

The predictors of AHI that we identified are consistent with the biology and transmission of HIV infection. The presence of GUD in 71% of AHI cases is consistent with the co-transmission of HIV and ulcerative STI, or with the facilitation of HIV acquisition by existing genital ulcers [23,24]. The association of AHI with multiple sexual partners in the previous 2 months corresponds to an increased likelihood of recent HIV exposure. Furthermore, the identified symptoms are consistent with those of acute retroviral syndrome, seen in up to 90% of recently infected individuals [10,11].

As we have reported previously [9], 33% of AHI cases had discordant rapid test results, compared with 2% of HIV-negative participants. Available rapid tests differ in sensitivity and specificity, with more sensitive tests detecting antibodies earlier in the course of

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seroconversion. The predictive ability of discordant rapid tests in identifying AHI cases will vary depending upon the particular combination of tests that is used.

Our algorithm is intended for use with similar HIV tests in sub-Saharan STI clinics, where the HIV incidence, predictor patterns, and timing of clinic presentation are likely to be comparable with those in the study setting. Clinical staff could implement the algorithm simply and quickly during routine care, first completing a brief checklist of the final algorithm predictors and then summing predictor scores to calculate a risk score for each patient. Patients with one or more negative rapid test and a risk score of 2 or greater would be referred for HIV-RNA or p24 testing. We caution that generalizability to dissimilar settings is likely to be limited, and that model development was based on a small number of cases. Validation of the algorithm in larger, site-specific field trials is essential.

Our findings suggest that risk score algorithms for identifying AHI cases in high-risk, resource-poor settings could be powerful tools in HIV prevention and control. By guiding the efficient use of RNA or p24 tests, risk score algorithms could improve the feasibility of AHI screening in these settings, enabling the identification of AHI cases that otherwise would be missed.

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References

1. Pilcher CD, Eron JJ Jr, Galvin S, Gay C, Cohen MS. Acute HIV revisited: new opportunities for treatment and prevention. J Clin Invest. 2004; 113:937–945. [PubMed: 15057296]
2. Fiebig EW, Wright DJ, Rawal BD, Garrett PE, Schumacher RT, Peddada L, et al. Dynamics of HIV viremia and antibody seroconversion in plasma donors: implications for diagnosis and staging of primary HIV infection. AIDS. 2003; 17:1871–1879. [PubMed: 12960819]
3. Leynaert B, Downs AM, de Vincenzi I. European Study Group on Heterosexual Transmission of HIV. Heterosexual transmission of human immunodeficiency virus: variability of infectivity throughout the course of infection. Am J Epidemiol. 1998; 148:88–96. [PubMed: 9663408]
4. Shiboski SC, Padian NS. Epidemiologic evidence for time variation in HIV infectivity. J Acquir Immune Defic Syndr Hum Retrovirol. 1998; 19:527–535. [PubMed: 9859968]
5. Wawer MJ, Gray RH, Sewankambo NK, Serwadda D, Li X, Laeyendecker O, et al. Rates of HIV-1 transmission per coital act, by stage of HIV-1 infection, in Rakai, Uganda. J Infect Dis. 2005; 191:1403–1409. [PubMed: 15809897]
6. Lindbaäck S, Karlsson AC, Mittler J, Blaxhult A, Carlsson M, Briheim G, et al. Viral dynamics in primary HIV-1 infection. AIDS. 2000; 14:2283–2291. [PubMed: 11089616]
7. Schacker TW, Hughes JP, Shea T, Coombs RW, Corey L. Biological and virologic characteristics of primary HIV infection. Ann Intern Med. 1998; 128:613–620. [PubMed: 9537934]
8. Pilcher CD, Tien HC, Eron JJ Jr, Vernazza PL, Leu SY, Stewart PW, et al. Brief but efficient: acute HIV infection and the sexual transmission of HIV. J Infect Dis. 2004; 189:1785–1792. [PubMed: 15122514]

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9. Fiscus SA, Pilcher CD, Miller WC, Powers KA, Hoffman IF, Price M, et al. Rapid, real-time detection of acute HIV infection in patients in Africa. J Infect Dis. 2007; 195:416–424. [PubMed: 17205481]
10. Tindall B, Barker S, Donovan B, Barnes T, Roberts J, Kronenberg C, et al. Characterization of the acute clinical illness associated with human immunodeficiency virus infection. Arch Intern Med. 1988; 148:945–949. [PubMed: 3258508]
11. Schacker T, Collier AC, Hughes J, Shea T, Corey L. Clinical and epidemiologic features of primary HIV infection. Ann Intern Med. 1996; 125:257–264. [PubMed: 8678387]
12. Southern African Regional Poverty Network. Government of Malawi, National AIDS Commission. National HIV/AIDS policy: a call to renewed action. Oct.2003 Accessed: August 2007Available at: http://www.sarpn.org.za/documents/d0000702/index.php
13. Pilcher CD, McPherson JT, Leone PA, Smurzynski M, Owen-O'Dowd J, Peace-Brewer AL, et al. Real-time, universal screening for acute HIV infection in a routine HIV counseling and testing population. JAMA. 2002; 288:216–221. [PubMed: 12095386]
14. World Health Organization. Proposed WHO criteria for interpreting results from Western blot assays for HIV-1, HIV-2 and HTLV-I/HTLV-II. Wkly Epidemiol Rec. 1990; 65:281–284. [PubMed: 2206928]
15. Harrell, FE. Regression modeling strategies. New York: Springer-Verlag; 2001.
16. Kassutto S, Maghsoudi K, Johnston MN, Robbins GK, Burgett NC, Sax PE, et al. Longitudinal analysis of clinical markers following antiretroviral therapy initiated during acute or early HIV type 1 infection. Clin Infect Dis. 2006; 42:1024–1031. [PubMed: 16511771]
17. Pilcher CD, Fiscus SA, Nguyen TQ, Foust E, Wolf L, Williams D, et al. Detection of acute infections during HIV testing in North Carolina. N Engl J Med. 2005; 352:1873–1883. [PubMed: 15872202]
18. Pilcher CD, Price MA, Hoffman IF, Galvin S, Martinson FEA, Kazembe PN, et al. Frequent detection of acute primary HIV infection in men in Malawi. AIDS. 2004; 18:517–524. [PubMed: 15090805]
19. Patel P, Klausner JD, Bacon OM, Liska S, Taylor M, Gonzalez A, et al. Detection of acute HIV infections in high-risk patients in California. J Acquir Immune Defic Syndr. 2006; 42:75–79. [PubMed: 16763493]
20. Lavreys L, Thompson ML, Martin HL Jr, Mandaliya K, Ndinya-Achola J, Bwayo JJ, et al. Primary human immunodeficiency virus type 1 infection: clinical manifestations among women in Mombasa, Kenya. Clin Infect Dis. 2000; 30:486–490. [PubMed: 10722432]
21. Sharghi N, Bosch RJ, Mayer K, Essex M, Seage GR. The development and utility of a clinical algorithm to predict early HIV-1 infection. J Acquir Immune Defic Syndr. 2005; 40:472–478. [PubMed: 16280704]
22. Daar ES, Little S, Pitt J, Santangelo J, Ho P, Harawa N, et al. Diagnosis of primary HIV-1 infection. Ann Intern Med. 2001; 134:25–29. [PubMed: 11187417]
23. Fleming DT, Wasserheit JN. From epidemiological synergy to public health policy and practice: the contribution of other sexually transmitted diseases to sexual transmission of HIV infection. Sex Transm Infect. 1999; 75:3–17. [PubMed: 10448335]
24. Røttingen JA, Cameron DW, Garnett GP. A systematic review of the epidemiologic interactions between classic sexually transmitted diseases and HIV: how much is really known? Sex Transm Dis. 2001; 28:579–597. [PubMed: 11689757]

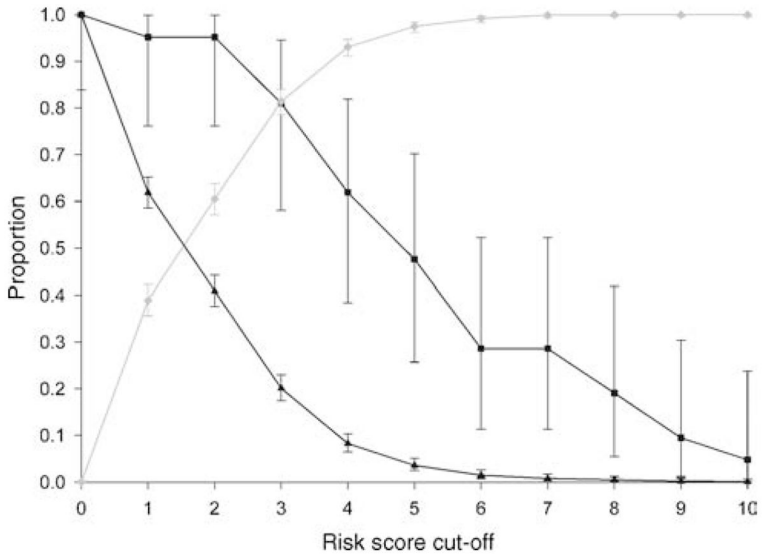

Fig. 1. Sensitivity, specificity, and percentage to be referred for RNA/p24 tests

The horizontal axis displays all possible risk score cut-offs that could be chosen for clinical implementation of the algorithm. These scores correspond to the range of risk scores observed across individuals in this study, based on each individual's predictor profile. In clinical implementation of the algorithm, all antibody-negative or discordant individuals with risk scores at or above a chosen cut-off would be referred for RNA or p24 tests. 'RNA or p24 tests' (—◆—) refers to the proportion of all antibody-negative or discordant patients who would be referred for RNA or p24 testing at a given cut-off (i.e. the proportion of all antibody-negative or discordant patients with risk scores at or above a given cut-off). As the risk score cut-off increases, fewer patients [both with acute HIV infection (AHI) and HIV negative] have risk scores at or above that value, such that fewer patients are indicated for RNA or p24 testing. 'Sensitivity' (—■—) refers to the proportion of AHI cases with risk scores at or above a given cut-off. As the risk score cut-off increases, fewer AHI cases will be referred for RNA or p24 testing, so they will not be detected as AHI cases (sensitivity decreases). 'Specificity' (—▲—) refers to the proportion of HIV-negative participants with risk scores lower than a given cut-off (i.e. the proportion of HIV-negative individuals ruled out as AHI cases). As the risk score cut-off increases, more HIV-negative participants have scores less than the cut-off, so specificity increases. The vertical lines with horizontal bars around each point estimate represent 95% confidence intervals.

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Table 1

Predictors of acute HIV-1 infection in Malawian sexually transmitted infection clinic population.

Predictor	AHI cases *n* (%)	HIV-negative *n* (%)	Unadjusted POR (95% CI)	Full, domain-specific model adjusted POR (95% CI)	Full, combined model adjusted POR (95% CI)	Final, combined model adjusted POR (95% CI)[a]	Predictor score[b]
Self-reported behaviors/demographics							
>1 Sex partner in past 2 months	11 (52)	194 (23)	3.65 (1.38, 9.73)	2.11 (0.66, 6.75)	4.17 (1.31, 13.27)	4.27 (1.16, 16.16)	1
History of ever using condom	18 (86)	540 (65)	3.30 (0.95, 17.61)	2.61 (0.70, 14.55)	–	–	–
No education	3 (14)	44 (5)	3.00 (0.55, 10.85)	3.70 (0.62, 14.31)	–	–	–
Paid sex in past 2 months	12 (57)	283 (34)	2.61 (0.99, 7.08)	1.46 (0.48, 4.53)	–	–	–
Alcohol use at last sex act	9 (43)	186 (22)	2.61 (0.95, 6.86)	1.62 (0.53, 4.69)	–	–	–
≥1 Medical injection in past 2 months	7 (33)	158 (19)	2.16 (0.72, 5.81)	2.34 (0.75, 6.41)	–	–	–
Symptoms							
Diarrhea in past month	4 (19)	34 (4)	5.57 (1.29, 18.31)	3.99 (0.77, 12.98)	4.88 (0.99, 24.01)	4.88 (0.55, 25.79)	2
Fever in past month	12 (57)	184 (22)	4.75 (1.80, 12.94)	2.90 (0.97, 8.61)	2.29 (0.76, 6.91)	2.35 (0.66, 7.70)	1
Body ache in past month	10 (48)	144 (17)	4.39 (1.63, 11.59)	2.47 (0.79, 7.29)	2.61 (0.85, 8.02)	2.69 (0.72, 8.77)	1
Sore throat in past month	1 (5)	11 (1)	3.76 (0.08, 28.24)	1.55 (0.02, 18.47)	–	–	–
Night sweats in past month	4 (19)	53 (6)	3.49 (0.82, 11.20)	1.43 (0.24, 5.49)	–	–	–
Weight loss in past month	6 (29)	104 (12)	2.83 (0.88, 7.91)	1.58 (0.41, 4.77)	–	–	–
Clinical examination findings							
Genital ulcer disease	15 (71)	220 (26)	7.03 (2.53, 22.35)	5.71 (1.86, 18.85)	5.20 (1.68, 16.08)	5.40 (1.58, 19.42)	2
Tender or swollen lymph nodes	11 (52)	158 (19)	4.74 (1.79, 12.66)	2.55 (0.89, 7.22)	1.20 (0.41, 3.55)	–	–
Tender/red/swollen genitals	14 (67)	358 (43)	2.69 (1.00, 7.94)	2.35 (0.80, 7.31)	–	–	–
Any rash	7 (33)	142 (17)	2.45 (0.82, 6.63)	1.93 (0.61, 5.52)	–	–	–
Genital warts	2 (10)	40 (5)	2.10 (0.23, 9.21)	2.47 (0.24, 12.40)	–	–	–
Genital discharge	13 (62)	479 (57)	1.22 (0.46, 3.44)	1.40 (0.48, 4.23)	–	–	–
Discordant rapid test results	7 (33)	14 (2)	29.5 (8.56, 92.48)	–	42.45 (9.82, 183.4)	44.21 (7.37, 206.11)	4

AHI, Acute HIV infection; CI, confidence interval; POR, prevalence odds ratio.

[a]For some variables retained as 'statistically significant' in the final model, the 95% confidence intervals span 1.0 because the α level used in likelihood ratio tests was 0.15, rather than 0.05 (see Methods section).

[b]Predictor score is the natural log of the adjusted prevalence odds ratio in the final model, rounded to the nearest integer (see Methods section).

The area under the full, domain-specific models' receiver operating characteristic curves ranged from 0.69 to 0.83.

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