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CASE REPORT

CLINICAL CASE

Acute Heart Failure During the Peripartum Period Due to Aggravated Tricuspid Regurgitation

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ABSTRACT

Latent valvular heart disease may be aggravated or demasked during pregnancy because of physiologic hemodynamic changes, including higher circulating volume, heart rate, and cardiac index, as well as stress during labor. The presence of valvular heart disease increases the risk of maternal and fetal/newborn adverse events. Early diagnosis, risk assessment, and specific management are crucial. We present a case of acute peripartal heart failure caused by idiopathic severe tricuspid regurgitation in a 38-year-old woman. (J Am Coll Cardiol Case Rep 2023;27:102071) © 2023 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 38-year-old female patient (gravida II, para I) at 38 weeks of gestation (in vitro fertilization) was admitted to the gynecology department because of contractions. The pregnancy was uneventful. Spontaneous vaginal delivery occurred without complications. The day after discharge (fifth day postpartum), she presented to the emergency department because of shortness of breath and leg edema. Her blood pressure was 135/85 mm Hg, heart rate was 60 beats/min, oxygen saturation was normal (96%) with a respiratory rate of 20/min, and body temperature was 37 °C.

LEARNING OBJECTIVES

- To highlight that acute HF can be consecutive to pre-existent or aggravated VHD during the peripartum period.
- To apply a multimodality imaging approach for VHD assessment VHD in the peripartum period.
- To emphasize the need for regular follow-up of women with VHD detected during pregnancy to determine the most appropriate timing for an intervention if indicated.

PAST MEDICAL HISTORY

The patient was born and raised in Haiti. She moved to central Europe at the age of 25 years. She had 1 previous uneventful pregnancy and delivery 11 years earlier, resulting in a spontaneous vaginal delivery. She has been experiencing hypothyroidism for 3 years and was euthyroid under substitution on admission. The family history revealed a history of arterial

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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ABBREVIATIONS AND ACRONYMS

HF = heart failure

IV = intravenous

LV = left ventricle

RV = right ventricle/ventricular

TR = tricuspid regurgitation

TTE = transthoracic echocardiography

VHD = valvular heart disease

hypertension, and there were no congenital heart defects.

DIFFERENTIAL DIAGNOSIS

Tricuspid regurgitation (TR) in women of childbearing age may be related to congenital heart disease (eg, Ebstein anomaly, atrioventricular canal defects, arrhythmogenic right ventricular cardiomyopathy, tricuspid valve dysplasia), secondary to annular dilatation in the setting of right ventricular (RV) volume overload, or from mechanical valve damage (eg, prior infectious endocarditis). Moreover, in the presence of arterial hypertension and edema, preeclampsia must be ruled out.

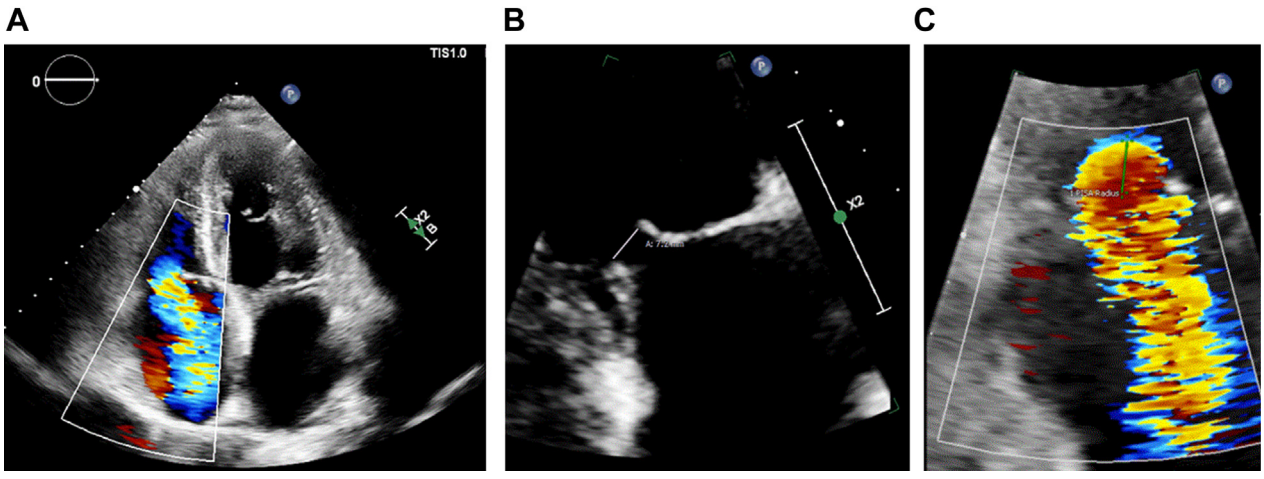
INVESTIGATIONS

The clinical examination revealed lower limb edema. Pulmonary auscultation was clear, and cardiac auscultation found a holosystolic murmur with punctum maximum at the lower left sternal edge. Laboratory analyses revealed mildly elevated C-reactive protein, mild anemia (hemoglobin, 102 g/L) without signs of hemolysis, normal platelet count, and elevated N-terminal pro-B-type natriuretic peptide (501 pg/mL) and D-dimer (3,431 μg/L). Renal function was normal, but liver parameters were mildly elevated (aspartate amino transferase: 42 U/L; alanine amino transferase: 45 U/L; alkaline phosphatase: 139 U/L). Urinary examinations revealed no abnormalities, in particular no proteinuria. The electrocardiogram showed sinus rhythm with no other abnormalities. Deep vein thrombosis and pulmonary embolism were ruled out using leg ultrasound and thoracic multislice computed tomography, which also excluded pleural effusions, pulmonary infiltrate, and aortic dissection. A transthoracic echocardiography (TTE) revealed visually slightly impaired systolic function of the RV with possible ballooning of the right apex but normal tricuspid annular plane systolic excursion and normal function of the left ventricle (LV). This resulted in severe TR caused by leaflet tethering with tricuspid annular dilatation (45 mm), leading to a coaptation gap of 7.2 mm with signs of RV volume overload and diastolic D-shaping of the LV (**Figures 1A to 1C, 2A, and 2B**, Videos 1 to 3).

MANAGEMENT

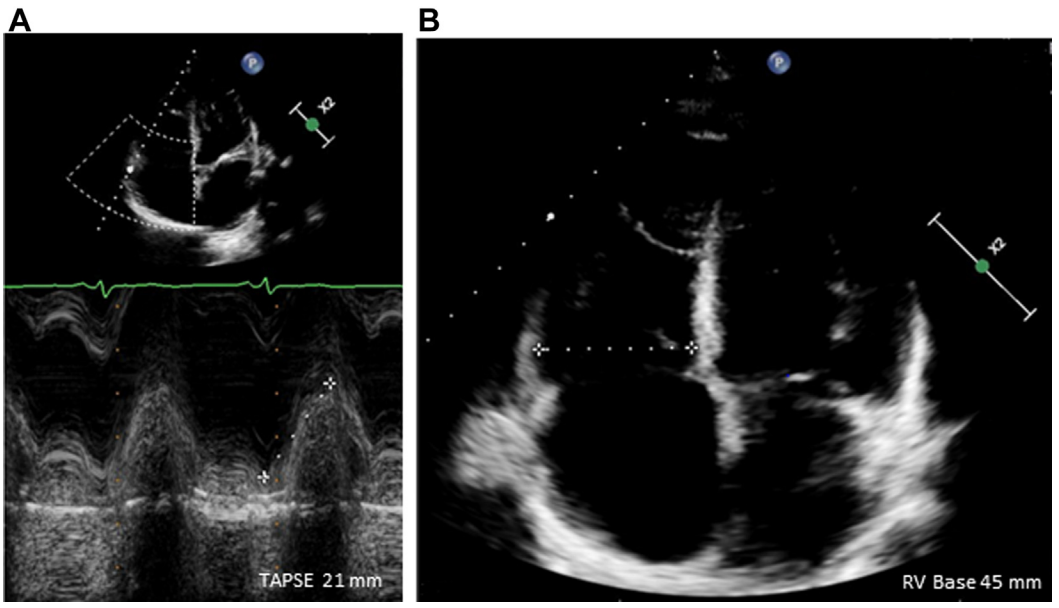
The patient was admitted to the mother-child unit with the preliminary diagnosis of acute cardiac decompensation caused by valvular heart disease (VHD), and forced diuresis was initiated with oral torsemide. However, because of hypertensive blood pressure readings (up to 190/130 mm Hg), she was transferred to the intensive care unit. Blood pressure was controlled with intravenous (IV) urapidil and oral amlodipine, and volume overload was treated with IV furosemide. Recompensation was rapidly achieved, and blood pressure was controlled, allowing the patient to be transferred to

FIGURE 1 TR Assessment at Presentation in Decompensated State



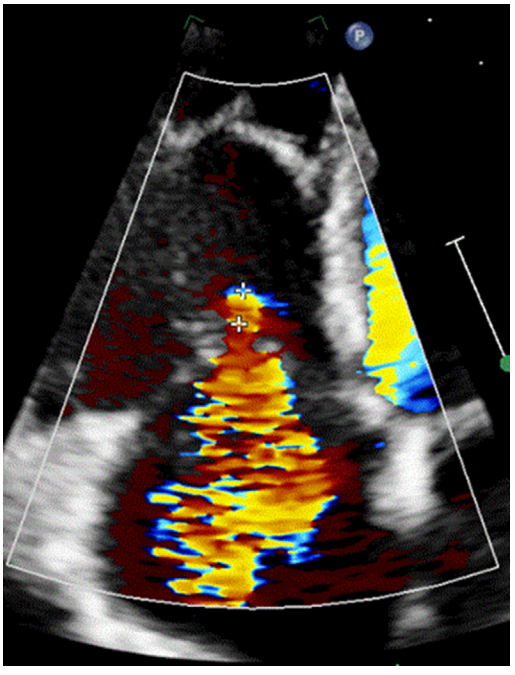
(A) Four-chamber view with color Doppler showing severe TR. (B) RV inflow view showing a coaptation gap of 7.2 mm. (C) RV-focused 4-chamber view with color Doppler proximal isovelocity surface area radius: 0.9 cm. RV = right ventricle; TR = tricuspid regurgitation.

FIGURE 2 **Right Ventricular Dimension and Longitudinal Function at Presentation in Decompensated State**



(A) Tricuspid annular plane systolic excursion within the normal range, 21 mm. (B) Four-chamber view with slightly enlarged right ventricle basal diameter of 45 mm.

FIGURE 3 **Echocardiographic Follow-Up at 1 Month**



Right ventricle–focused 4-chamber with color Doppler; proximal isovelocity surface area radius: 0.5 cm.

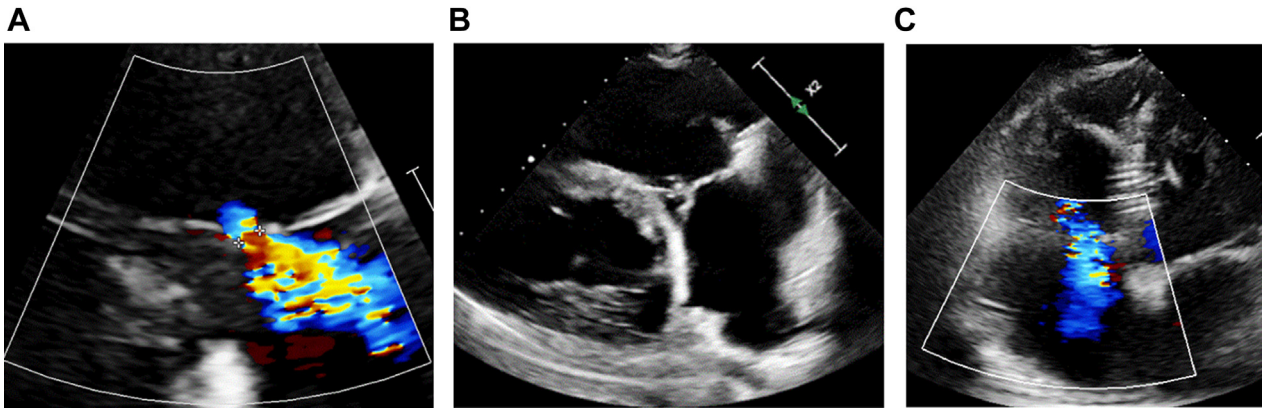
the cardiology ward. The antihypertensive medication was changed to oral hydrochlorothiazide and amlodipine, while IV furosemide was replaced by oral torsemide. No rhythm disturbances occurred. The laboratory and urine analysis results remained within the normal ranges. The patient did not develop symptoms suggestive of pre-eclampsia such as respiratory distress, abdominal pain, or new-onset and persistent headache or visual symptoms, and remained afebrile during the hospital stay, without signs of infection.

A second TTE showed normal RV dimensions with normal RV systolic function; however, there was persistence of severe secondary TR caused by leaflet tethering, resulting in a central jet. RV/right atrial gradient was at the upper limit (30 mm Hg) in the context of peripartum hemodynamic changes. There were no echocardiographic signs suggestive of congenital heart disease. In addition, secondary causes of systemic hypertension were excluded, including pre-eclampsia or hyperthyroidism. The patient was discharged home 2 days later with oral hydrochlorothiazide and amlodipine.

DISCUSSION

TR is of secondary etiology in >90% of the patients,[1] and TR of any severity is often observed during

FIGURE 4 Echocardiographic Follow-Up at 5 Months

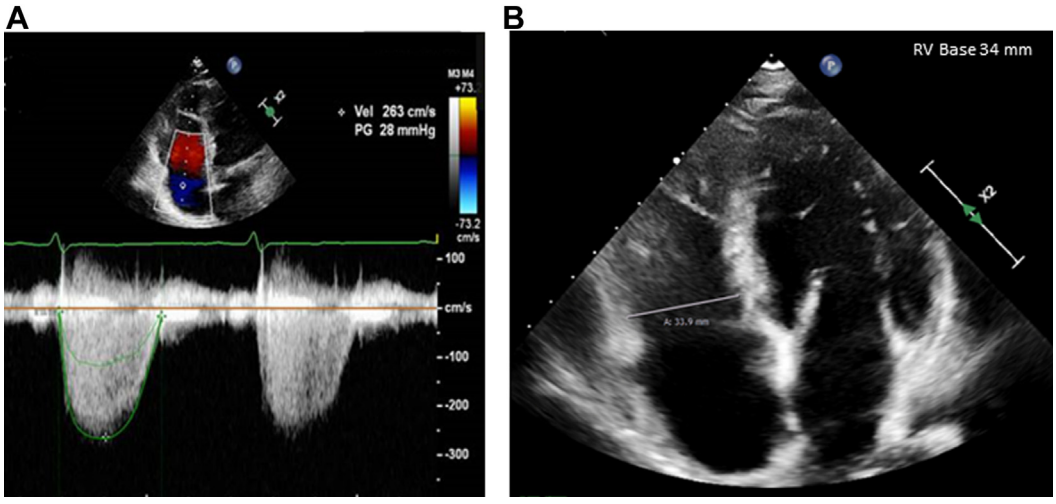


(A) Right ventricle inflow view with color Doppler; proximal isovelocity surface area radius: 0.4 cm. (B) Right ventricle inflow view: no relevant coaptation defect visible after diuretic therapy. (C) Four-chamber view with color Doppler showing moderate tricuspid regurgitation.

pregnancy[2] as a result of increased circulating volume, heart rate, and cardiac output, with or without consecutive dilatation of the tricuspid annulus. TR is usually well tolerated during the peripartum period,[3] and an acute presentation like the one described in this report is rare in an otherwise healthy woman and should prompt further investigations. In contrast, in women with pre-existing congenital heart disease, moderate/severe TR may be associated with maternal cardiac complications because of chronic volume overload of the RV that may not be able to accommodate the new hemodynamic conditions.[4]

The presence of VHD in general—in particular, moderate or severe chronic mitral regurgitation, TR, multivalve disease—LV systolic dysfunction, or pulmonary hypertension (PHT) diagnosed before pregnancy predicts cardiac adverse events during pregnancy.[5] Furthermore, adverse fetal and neonatal events are increased in the presence of all types of

FIGURE 5 Echocardiographic Follow-Up at 5 Months



(A) Four-chamber view with continuous-wave Doppler. (B) Four-chamber view with right ventricle basal diameter within the normal range, 34 mm.

TABLE 1 Summary of RV and TR Measurements at Presentation, Baseline, and Follow-Up

	TTE at Presentation	TTE at Discharge	TTE at the 5-Month Follow-Up	CMR at the 5-Month Follow-Up
RVEDD base, mm	45	39	38	
RA ESV index, mL/m^2	40.8	29.13	22.10	
TAPSE, mm	21	21	28	
RV S′, cm/s	15.1	N/A	14.6	
FAC, %	33	35	43	
RV/RA gradient, mm Hg	25	25	24	
IVC diameter during expirium, mm	25	13	11	
IVC diameter during inspirium, mm	19	11	8	
Estimated central venous pressure, mm Hg	15	10	5	
Estimated sPAP, mm Hg	40	35	29-30	
TR severity	Severe	Moderate	Moderate	
TR Vmax, m/s	2.5	2.5	2.45	
Coaptation gap, mm	7.2	0	0	
TR EROA, cm^2	0.78	0.20	n.a.	
Regurgitation volume, mL	58.93	20.33	n.a.	
Vena contracta, mm	7.5	4.2	4	
RV EDV index, mL/m^2				90 (normal range: 48-104)
RV ESV, mL				38 (normal range: 13-48)
RV SV index, mL/m^2				53 (normal range: 29-66)
RV CO, L/min/m^2				3.7
RVEF, %				58
TR regurgitant fraction, %				16
Q_p/Q_s PA/Ao				1

CMR = cardiac magnetic resonance; CO = cardiac output; EDV = end-diastolic volume; EROA = effective regurgitant orifice area; ESV = end-systolic volume; FAC = fractional area change; IVC = inferior vena cava; N/A = not applicable; PA/Ao = main pulmonary artery/aorta; RA = right atrium; RV = right ventricle; RVEDD = right ventricular end-diastolic diameter; RVEF = right ventricular ejection fraction; sPAP = systolic pulmonary artery pressure; SV = stroke volume; TAPSE = tricuspid annular plane systolic excursion; TR = tricuspid regurgitation; TTE = transthoracic echocardiography; Vmax = maximal velocity.

maternal regurgitant valve lesions.[5] The risk of maternal cardiac events during pregnancy can be further stratified using the CARPREG II (Cardiac Disease in Pregnancy Study II) risk score, a 10-predictor score comprising the aforementioned high-risk features.[6]

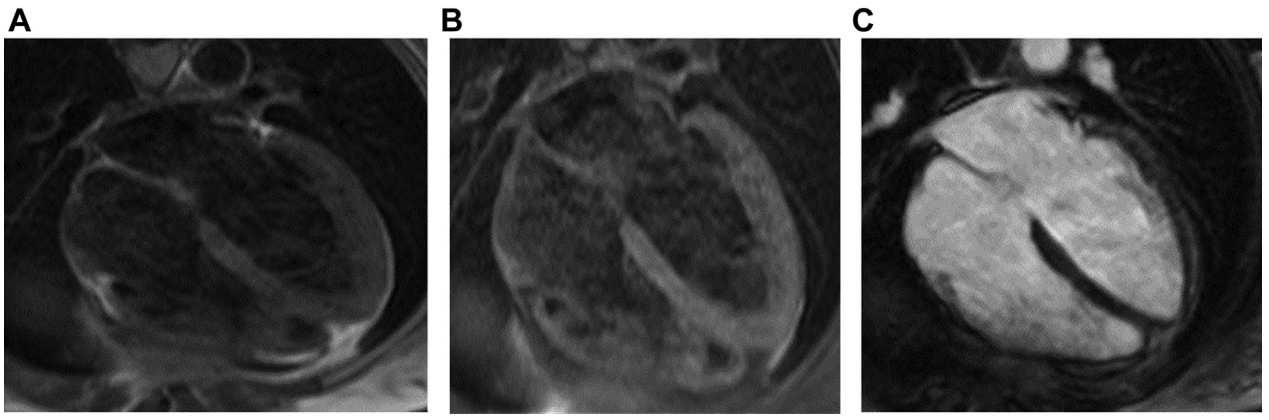
In our case, the exact etiology of TR remains unclear, and pregnancy accompanied by physiologic volume overload may have acted as the demasking factor of a so far subclinical condition. The presence of tethering with minimal annular dilatation confirms the acute presentation of VHD. Dyspnea linked to insufficient exercise cardiac output represents an early symptom of TR that should lead to further investigations and the use of multimodality imaging, in particular cardiac magnetic resonance, to exclude a congenital etiology, confirm TR grading, and assess RV function. The acute presentation of acute heart failure (HF) with visually impaired RV function and slight apical ballooning might also suggest isolated RV stress cardiomyopathy, possibly caused by stress during labor, which resolved in the follow-up visits with remaining moderate TR.

VHD is increasingly recognized as a cause of acute HF or a contributing factor that may precipitate the onset of HF symptoms. Recent recommendations encourage the interdisciplinary management of patients with HF and VHD.[7] Symptoms or progression of chamber dilatation should trigger early referral to a center with competence in valve treatment, including emerging minimally invasive treatment options.

FOLLOW-UP

The patient presented for a clinical visit at the adult congenital heart disease department 4 weeks, 3 months, and 5 months postpartum. Follow up TTE at 1 month (**Figure 3**, Video 4) and 5 months (**Figures 4A to 4C, 5A, and 5B**, Videos 5 and 6) showed reduction to moderate TR under medical treatment as prescribed at discharge. RV/right atrial gradient (23 mm Hg) was not suggestive of pulmonary

FIGURE 6 Cardiac Magnetic Resonance at the 5-Month Follow-Up Showing Normal Dimensions and Function of the Heart Chambers



(A) Cardiac magnetic resonance 4-chamber view without fat saturation. (B) Cardiac magnetic resonance 4-chamber view with fat saturation. No myocardial fatty deposits are visible, and there are no signs of arrhythmogenic right ventricular cardiomyopathy. (C) Cardiac magnetic resonance 4-chamber view with application of gadolinium magnetic resonance contrast. No late gadolinium enhancement could be observed, suggesting a structurally normal heart with no fibrosis or signs of inflammatory or systemic heart disease.

hypertension. The right basal diameter decreased to 34 mm. In addition, cardiac magnetic resonance revealed normal LV and RV function with normal dimensions (indexed right ventricular end-diastolic volume: 90 mL/m^2; indexed right ventricular end-systolic volume 38 mL/m^2) (**Table 1**), and no structural abnormalities, in particular no sign of tricuspid valve and RV dysplasia, were observed (**Figures 6A to 6C**, Videos 7 and 8). Moderate TR was confirmed (regurgitant fraction: 16%). No intracardial shunts were seen (Q_p/Q_s: 1.0). Systemic blood pressures normalized under hydrochlorothiazide monotherapy. In the absence of clinical factors suggesting secondary causes (hyperthyroidism, hyperparathyroidism, coarctation of the aorta, and sleep apnea were excluded), hypertension was considered of gestational etiology. Yearly echocardiographic controls are planned to detect symptoms, TR progression, and signs of RV dilatation early.

CONCLUSIONS

Our case report first raises awareness around the possible occurrence of TR during the peripartum period caused by increased circulating volume. Heart conditions like VHD that are unmasked during pregnancy may pose a cardiovascular risk factor later in life. An increasing body of evidence also suggests that pre-existing heart conditions in pregnant women increase the risk of adverse events not only in women but also in the fetus/newborn. According to current American College of Cardiology/American Heart Association[8] and European Society of Cardiology[9] guidelines, women with suspected VHD who are considering pregnancy should undergo a clinical evaluation and TTE before pregnancy (Class I indication). If VHD is first detected during pregnancy, early referral and interdisciplinary follow-up at a specialized Heart Valve Center are indicated.

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KEY WORDS acute heart failure, multimodality imaging, peripartum, pregnancy, tricuspid regurgitation, valvular heart disease

APPENDIX For supplemental videos, please see the online version of this paper.