CARDIOLOGIE TUNISIENNE

CASE REPORT

Acute coronary syndrome revealing a Goodpasture Syndrome

Syndrome coronarien aigue révélant un syndrome de Goodpasture

Manel Abbassi1, Souad Ferjani2, Taha Lassoued1, Mohamed Abd El Hay Abdelwedoud1, Soumaya Charaa2, Ahmed Mahmoud Cheikh2, Houda Belkhiria1, Firas Nouri1, Oussama Bouahmed3, Sami Milouchi1

1. *Cardiology department, University hospital of Medenine, Tunisia*
2. *Radiology department, University hospital of Medenine, Tunisia*
3. *Department of Anesthesiology and Reanimation, University hospital of Medenine, Tunisia*

Summary

Goodpasture syndrome is a rare autoimmune disease typically presenting with diffuse alveolar haemorrhage and rapidly progressive glomerulonephritis. Initial cardiac involvement is exceptional. We report a 44-year-old asthmatic woman without cardiovascular risk factors presenting with NSTEMI and elevated troponin. Cardiac MRI showed transmural myocardial necrosis. Days later, massive haemoptysis, ARDS, and acute kidney injury revealed anti-GBM disease confirmed by antibodies. Despite corticosteroids and plasmapheresis, the outcome was rapidly unfavourable.

Keywords

Goodpasture syndrome, anti-GBM disease, non-ST-elevation myocardial infarction, cardiac MRI.

Résumé

Le syndrome de Goodpasture est une maladie auto-immune rare, habituellement révélée par une hémorragie alvéolaire diffuse et une glomérulonéphrite rapidement progressive et touche exceptionnellement le cœur. Nous rapportons le cas d'une jeune femme, sans facteur de risque cardiovasculaire, admise pour un SCA sans sus-décalage du ST. L'IRM cardiaque a montré une nécrose myocardique transmurale. Quelques jours plus tard, une hémoptysie massive et une insuffisance rénale aiguë ont conduit au diagnostic de Goodpasture.

Mots-clés

Syndrome de Goodpasture, syndrome coronarien aigu, IRM cardiaque

Correspondance

Manel Abbassi

INTRODUCTION

Goodpasture syndrome is a rare autoimmune disease characterized by antibodies directed against the glomerular basement membrane (anti-GBM), responsible for the classic presentation of pulmonary-renal syndrome combining diffuse alveolar hemorrhage and rapidly progressive glomerulonephritis (1,2).

However, cardiovascular involvement in Goodpasture syndrome remains exceptional and poorly described in the literature (2,3).

This case report describes an unusual presentation of Goodpasture syndrome initiated by acute coronary syndrome without ST-segment elevation (NSTEMI), preceding by only days the classic revelation by pulmonary hemorrhage and renal involvement.

CASE PRESENTATION

Clinical history and medical background

A 44-year-old female patient presented with progressive, severe anginal chest pain without clearly identified trigger factors. The patient had asthma treated with beta-2 agonists, without other significant medical or surgical history, nor classic cardiovascular risk factors (smoking, diabetes, hypertension, or dyslipidemia…)

Initial presentation and laboratory investigations

On admission in our cardiology department, the patient had persistent chest pain with typical anginal features. The electrocardiogram showed ST-segment depression in the high lateral leads, consistent with myocardial ischemia. Laboratory blood tests revealed extremely elevated troponin (10,000 ng/L), suggesting an acute coronary syndrome. An NSTEMI was suspected, although acute myocarditis could not be excluded given the patient's profile (young age, without cardiovascular risk factors). Clinical evolution is summarized in table 1.

Initial transthoracic echocardiography demonstrated a normal-sized left ventricle with preserved ejection fraction at approximately 54%, without obvious segmental wall motion abnormalities on apical four-chamber view.

Biology findings on admission:

- Hemoglobin: 11.4g/dL.
- White blood cells: 11.7×10^9/L.
- Neutrophils: 7.2×10^9/L.
- Platelets: 249×10^9/L.

Table 1. Clinical Chronology and Key Events

Day	Clinical events	Investigations	Key findings
0	Admission with anginal chest pain	ECG, Troponin	ST depression high lateral, Troponin: 10,000 ng/L
0–2	Hospitalization, relative stability	Echocardiography, Cardiac MRI	LVEF 54%, Transmural LGE segments 6/5/12/11
2–3	Massive hemoptysis, ARDS onset	Chest CT	Diffuse alveolar hemorrhage
3–4	Progressive acute kidney injury, intubation	Immunology panel	Anti-GBM positive → Goodpasture diagnosis
4–7	Intensive treatment (methylprednisolone, plasmapheresis)	Serial monitoring	Progressive deterioration → death

Abbreviations : ARDS : Acute Respiratory Distress Syndrome, CT : Computed Tomography, ECG : electrocardiogram, LGE: late gadolinium enhancement, LVEF: left ventricule ejection fraction, MRI : magnetic resonance imaging, GBM: glomerular basement membrane.

Cardiac imaging by magnetic resonance imaging (MRI)

During hospitalization, cardiac MRI with FIESTA cine sequences, double IR-T2 sequences, FAT-SAT, and late gadolinium enhancement (LGE) sequences was performed. It demonstrated a non-dilated left ventricle with preserved global systolic function (ejection fraction 56%, end-diastolic volume 107 mL).

Cardiac MRI revealed a transmural late gadolinium enhancement in segments 6/5/12/11 (basal to mid-lateral segments), associated with segmental hypokinesis at these segments, without focal or global myocardial hypertrophy and without endocavitary thrombus. This pattern of transmural LGE, systematized to a coronary territory (left circumflex for segments 6 and 5, left anterior descending for segments 12 and 11), was compatible with an ischemic-type transmural myocardial necrosis (4). (Figure 1)

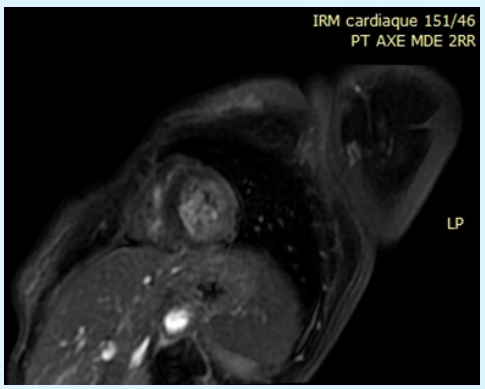

Figure 1. Cardiac MRI – Late Gadolinium Enhancement (LGE)

Cardiac MRI demonstrating transmural late gadolinium enhancement in the basal and mid-lateral wall segments (segments 6, 5, 12, 11), consistent with ischemic-type myocardial

necrosis. After 12 hours, the symptoms resolved and, he recovered completely without any neurological deficit or cardiac complication. Transient cerebral ischemia appeared unlikely given the non-localizing neurological symptom complex. Contrast-induced encephalopathy (CIE) was the most likely diagnosis.

Pulmonary and renal acute complications

During hospitalization, the patient suddenly developed massive hemoptysis, rapidly complicated by acute respiratory distress syndrome (ARDS) requiring intubation and mechanical ventilation. In parallel, acute kidney injury with rapidly progressive elevation of plasma creatinine ensued.

Facing this atypical clinical evolution combining cardiac involvement with acute pulmonary-renal complications, an urgent thoracic CT scan was performed. It demonstrated diffuse and massive alveolar hemorrhage, manifesting as diffuse bilateral ground-glass opacities, roughly symmetric, centered on the hilar regions, extending to lower pulmonary zones, compatible with diffuse alveolar hemorrhage. (Figure 2).

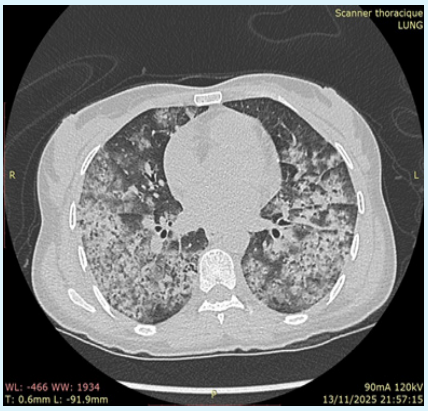

Figure 2. High-Resolution Computed Tomography of Thorax – Axial View
Non-contrast CT of the thorax demonstrating bilateral, diffuse, ground-glass opacities with confluence in the lower lobes and hilar regions, consistent with acute diffuse alveolar hemorrhage.

Diagnostic workup: Goodpasture Syndrome

Given the unusual association of acute coronary syndrome and diffuse alveolar hemorrhage with acute kidney injury, a pulmonary-renal syndrome was strongly suspected, specifically an anti-GBM disease (i.e. Goodpasture syndrome).

Immunological testing has confirmed this hypothesis, demonstrating positive anti-GBM antibodies.

Evolution of biological findings

Progressive biological deterioration was noticed with occurrence of an acute kidney failure (Urea: 9.56mmol/L, Creatinine: 254.1µmol/L) and positive proteinuria.

Treatment and clinical evolution

The patient was intubated and underwent controlled mechanical ventilation. Aggressive immunosuppressive therapy was initiated, combining:

- High-dose intravenous corticotherapy (methylprednisolone boluses)
- Two urgent sessions of plasmapheresis, aimed at eliminating circulating anti-GBM antibodies

These therapeutic modalities corresponded to recommended regimens for severe Goodpasture syndrome with major pulmonary involvement and rapidly progressive renal failure (1,5). Despite intensive management, the clinical course deteriorated rapidly, with progressive worsening of respiratory distress, renal failure, and development of multiorgan failure. The patient deceased in a context of refractory acute respiratory distress syndrome and multivisceral dysfunction.

DISCUSSION

In our case, the initial clinical presentation showed an anginal chest pain, without any renal or respiratory manifestation in a young patient without recognized cardiovascular risk factors. Markedly elevated troponin (10,000 ng/L) could suggest either true ischemic NSTEMI or acute inflammatory myocarditis.

Cardiac MRI played a decisive role in the differential diagnosis. The transmural LGE pattern concorded with an ischemic infarction. In this case, the subsequent manifestation of systemic disease (Goodpasture) suggests that the underlying mechanism may be related to microvasculitis or myocardial ischemia secondary to anti-GBM systemic inflammation. CT thorax demonstrating diffuse bilateral alveolar hemorrhage is compatible with alveolar hemorrhage, the pathognomonic presentation of vasculitis with pulmonary involvement, particularly Goodpasture syndrome (6).

Anti-GBM disease confirmed by positive anti-GBM antibodies is a small-vessel vasculitis, autoimmune in nature, of unknown etiology, characterized by production of autoantibodies directed against the NC1 (non-collagen-1) domain of the alpha-3 chain of type IV collagen, present in both renal and pulmonary basement membranes (1,5).

Medical literature classically describes Goodpasture syndrome through its respiratory and renal manifestations (2,7). Typical presentation modes include isolated hemoptysis, then hemoptysis-renal insufficiency association, or more rarely, inaugural renal involvement with secondary pulmonary manifestation.

Cardiovascular involvement in anti-GBM disease is very rarely documented. A few isolated reports describe cardiac involvement,

including cases of myocarditis, pericarditis, or arrhythmias, but rarely acute coronary events documented by troponin elevation and myocardial necrosis on imaging (2,3).

This case is distinguished by the fact that cardiac involvement constitutes the inaugural presentation of Goodpasture syndrome, preceding by only days the classic revelation by diffuse alveolar hemorrhage and acute kidney injury. This raises the question of a continuum of anti-GBM vasculitis affecting first the myocardium, then manifesting pulmonary and renally.

Published observations of cardiac involvement in Goodpasture syndrome remain extremely rare. To our knowledge, presentation of Goodpasture syndrome initiated by NSTEMI with transmural necrosis on cardiac MRI, before classic revelation by alveolar hemorrhage and acute kidney injury, constitutes a highly original observation.

CONCLUSION

This case report documents an unusual presentation of Goodpasture syndrome, initially revealed by NSTEMI documented by elevated troponin and transmural myocardial necrosis on MRI, before the classic manifestation of pulmonary-renal syndrome. This observation highlights an unusual differential diagnosis for acute coronary syndromes in young patients without classic cardiovascular risk factors. It also contributes to enriching the very limited literature on cardiovascular involvement in Goodpasture syndrome.

REFERENCES

1. Goodpasture EW. Landmark publication from The American Journal of the Medical Sciences: The significance of certain pulmonary lesions in relation to the etiology of influenza. Am J Med Sci. 2009 Aug;338(2):148–51. doi:10.1097/MAJ.0b013e31818fff94 PubMed PMID: 19680020.
2. Rout P, DeVrieze BW. Goodpasture Syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited 2026 Jan 25]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK459291/ PubMed PMID: 29083697.
3. Chinniah C, Pyronneau A, Stepman G, Ali R. A Unique Case of Goodpasture's Syndrome-Induced Cardiorenal Syndrome. Cureus. 16(7):e64269. doi:10.7759/cureus.64269 PubMed PMID: 38988901; PubMed Central PMCID: PMC11236434.
4. Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U, et al. Cardiovascular Magnetic Resonance in Nonischemic Myocardial Inflammation: Expert Recommendations. J Am Coll Cardiol. 2018 Dec 18;72(24):3158–76. doi:10.1016/j.jacc.2018.09.072 PubMed PMID: 30545455.
5. Kuang H, Jiang N, Jia XY, Cui Z, Zhao MH. Epidemiology, clinical features, risk factors, and outcomes in anti-glomerular basement membrane disease: A systematic review and meta-analysis. Autoimmunity Reviews. 2024 Apr 1;23(4):103531. doi:10.1016/j.autrev.2024.103531
6. Travis WD, Costabel U, Hansell DM, King TE, Lynch DA, Nicholson AG, et al. An official American Thoracic Society/European Respiratory Society statement: Update of the international multidisciplinary classification of the idiopathic interstitial pneumonias. Am J Respir Crit Care Med. 2013 Sep 15;188(6):733–48. doi:10.1164/rccm.201308-1483ST PubMed PMID: 24032382; PubMed Central PMCID: PMC5803655.
7. Henderson SR, Salama AD. Diagnostic and management challenges in Goodpasture's (anti-glomerular basement membrane) disease. Nephrol Dial Transplant. 2018 Feb 1;33(2):196–202. doi:10.1093/ndt/gfx057 PubMed PMID: 28459999.