

Clinical-therapeutic and recuperatory features in a patient with pluripatology: ischemic stroke, ischemic heart disease (sechelar myocardial infarction), chronic kidney disease and monstrous gout

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Abstract

Introduction: gout is a chronic inflammatory arthropathy produced by depositing crystals of monosodium uric acid (in joints and tissues) following an anomaly (genetics or acquired) in the purine metabolism (1,2). The manifestations of the disease are: hyperuricemia, recurrent episodes of acute arthritis, the presence of tophi, chronic kidney disease, urinary lithiasis(2). Stroke represents,, the rapid development of localized or global clinical signs of cerebral dysfunction with symptoms exceeding 24 hours, leading to death, without any other cause, except for vascular origin". (3)

Materials and Methods: with the permission of the THEBA Ethics Commission (no.17464/14.06.2019), we will present the clinical case of a 57-year-old patient admitted to the TEHBA Neuromuscular Recovery Clinic presenting a right hemiplegia and mixed aphasia after an ischemic stroke in the territory of the left middle cerebral artery, on the background of complex polypathology (monstrous gout arthropathy, chronic smoking, arterial hypertension, myocardial infarction with coronary artery stenosis, chronic kidney disease).

Results: the patient did in our clinic a neuro-muscular recovery treatment, adapted to his needs, which consisted of kinetotherapy and speech therapy and received appropriate medical treatment. The clinical evolution of the patient was slowly favorable, with improvement in language disorders and motor control of paralyzed limbs.

Conclusions: the case of this patient has several particularities. This is a patient with a vicious life style, with a severe arthropathy, with severe cardio-vascular sufferers, hospitalized for recovering neuro-muscular deficits after an ischemic stroke. Despite limited prognosis, the patient has improved ADL and the quality of life after recovery treatment.

Key words: *stroke, hemiplegia, poly-pathologic, ischemic,*

Introduction

Stroke is: ,, the rapid development of localized or global clinical signs of cerebral dysfunction with symptoms exceeding 24 hours, leading to death, without any other cause, except for vascular origin"

(3) Stroke is: 80-85% ischemic (thromboembolic: atrial fibrillation, acute myocardial infarction, valvulopathy, congenital heart disease, atherosclerosis, hypercoagulability, arteriopathy) and 10-15% haemorrhage (after traumatic brain injury, broken arterio-venous malformations, hypertensive encephalopathy, coagulopathies) (3)

Ischemic heart disease is: "stable coronary heart disease is characterized by reversible episodes between myocardial demand – supply, which can be attributed to ischemia or hypoxia, induced by exercise, effort or stress, which are reproducible, but which can also occur spontaneously" (4). Cardio-vascular diseases are a very important cause of mortality and morbidity in the modern world (5). Chronic kidney disease (6):

Classification of chronic kidney disease using GFR and ACR categories

GFR and ACR categories and risk of adverse outcomes		ACR categories (mg/mmol), description and range		
		<3 Normal to mildly increased	3–30 Moderately increased	>30 Severely increased
GFR categories (ml/min/1.73m ²) description and range	≥90 Normal and high	G1	A1	A3
	60–89 Mild reduction related to normal range for a young adult	G2	A2	A3
	45–59 Mild-moderate reduction	G3a ¹	A2	A3
	30–44 Moderate-severe reduction	G3b	A2	A3
	15–29 Severe reduction	G4	A2	A3
	<15 Kidney failure	G5	A2	A3

¹ Consider using eGFRcystatinC for people with CKD G3aA1 (see recommendations 1.1.14 and 1.1.15)

Abbreviations: ACR, albumin:creatinine ratio; CKD, chronic kidney disease; GFR, glomerular filtration rate

Adapted with permission from Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group (2013). KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney International (Suppl. 3): 1–150

Gout is a chronic inflammatory arthropathy produced by depositing crystals of monosodium uric acid (in joints and tissues) following an anomaly (genetics or acquired) in the purine metabolism(7,8). Gout affects up to 7% of the population (men: women's ratio is 3.6-1): men aged 75-84 years and women > 85 years of age(9).

2015 ACR/EULAR Gout Classification Criteria

Criteria	Categories	score
C L I N I C A	Pattern of joint/bursa involvement	Ankle OR midfoot (mono-/oligo-)
		1 st MTP (mono-/oligo-)
	Characteristics of episode(s) ever	One characteristic
		Two characteristic
		Three characteristics
L A B	Time-course of episode(s) ever	One typical episode
		Recurrent typical episodes
I M A G E	Clinical evidence of tophus	Present
	Serum Urate	6 - <8 mg/dL
		8 - <10 mg/dL
		≥ 10 mg/dL
S U A	Imaging evidence of urate deposition	Present (U/S DCS or DECT)
	Imaging evidence of gout-related joint damage	Present (X-ray gouty erosion)
SUA=4 -4 / MSU=ve -2		Maximum Total Score
		23

Score ≥ 8 ⇒ High sensitivity (92%) and specificity (89%)

Arthritis Rheumatol. 2015;67:2257-68

(10)

Clinical forms of gout are: asymptomatic / symptomatic hyperuricemia, acute gout attack, intercritical gout, chronic tophaceous gout (11). Complication of gout can be: metabolic syndrome, cardiovascular diseases, chronic kidney disease, urinary lithiasis, atherosclerosis (8). Gout treatment is done by: dietary and lifestyle (physical exercises, weight loss)(9) , limiting excessive consumption of purines (seafood, meat, beer)(10), drugs: acute phase (Colchicine, NSAI's, CS's) and chronic phase (Allopurinol, Febuxostat, Probenecid, Sulpyrinazone, Benzbromarone, Losartan, Fenofibrat)(11), surgical methods: radical debridement of tophi, joint replacements, resection arthroplasty, joint arthrodesis, primary amputation (9).

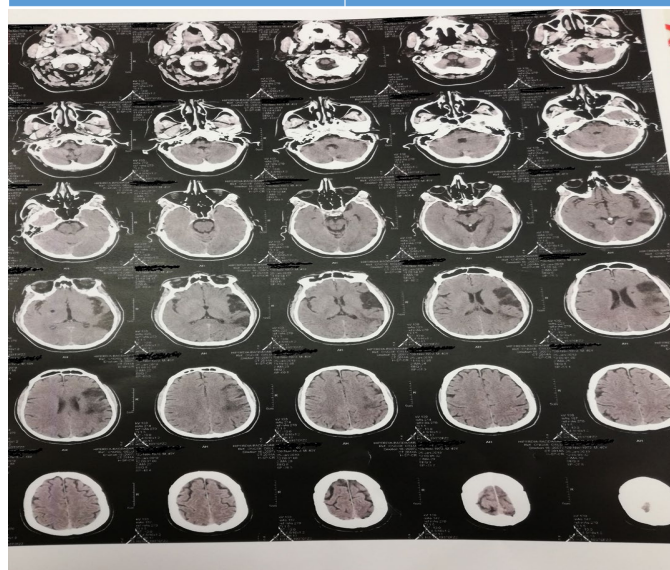
Case presentation we present the clinical case of a 57-year-old patient admitted to the TEHBA Neuromuscular Recovery Clinic for: right spastic hemiplegia, mixed aphasia, specialized evaluation and treatment. From the history of the disease we find next pathology: in 1996: gout (treated with Allopurinol, Colchicine), in 2012: acute anterior transmural myocardial infarction (treated with anterior interventricular artery PTCA stent), in 20.12.2018: ischemic stroke in the middle cerebral artery (superficial territory), essential hypertension, chronic kidney disease, urinary lithiasis, systemic atherosclerosis, chronic smoking. In general clinical examination, we see: slight facial asymmetry, numerous tophi (hands, feet, knees, elbows, right ear), IV right hand finger amputation , BP= 170/90 mmHg, VA= 62/min.

The neurological examination showed: right spastic hemiplegia, central facial paresis, mixed aphasia, relatively good motor control proximal-intermediate-distal right limbs, walking independently possible on short distances, on flat ground. Patient evaluation scales are: FIM (Functional Independent Measure): 35 (admission) –

55 (discharge), GOS (Glasgow Outcome Scale): 3 - severe disability (admission and discharge), modified Rankin Scale: 3 -moderate disability (admission and discharge), Ashworth Scale: 1 - discrete spasticity in the right limbs, Penn Scale: 0 - without spasms.



GPT	65 mg/dl
GOT	35 mg/dl
GGT	62 mg/dl
Creatinine	1,74 mg/dl
Uric acid	7,5 mg/dl
Urea	38 mg/dl
Fibrinogen	854,246 mg/dl
Hemoglobin	11,5 g/dl
GFR Value	43 mL/min/1.73 m2





The established diagnosis was: right spastic hemiplegia, mixed aphasia, sechelar ischemic stroke in the middle cerebral artery (superficial territory), essential hypertension –stage 3, acute anterior transmural myocardial infarction - anterior interventricular artery PTCA stent (2012), monstrous gout, chronic kidney disease -G3b stage. Clinical-functional evolution of the patient was favourable. The patient received medical treatment (with: injectable anticoagulants, analgesics, alpha blockers, platelet anti-aggregates, hypotensive agents, hypolipemic agents, xanthine oxidase inhibitors, vitamins, antibiotics), has practiced logopedy and a suitable kinetotherapeutic program (passive and active exercises at the bed level, then at the gym).

Discussion and conclusion: I have presented the complex case of a patient with multiple comorbidities, who had a stroke that complicated the clinical picture. However, neurological deficits were not complete. And, despite the limited prognosis, the patient progressed in the recovery process. This is a contradictory case that raises etiological and evolutive questions

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