



CASE REPORT

Acute Pericarditis in Chronic Dialysis Patients in Military Hospital of Morocco: About 8 Cases

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Abstract

Uremic pericarditis was described by R. Bright in 1836, several factors were incriminated in the occurrence of this complication in this population in particular an inadequate kidney substitution treatment.

It is a retrospective study of the clinico-biological and ultrasound characteristics as well as predictive factors for surgical drainage of the pericardium, performed on 8 chronic hemodialysis patients followed in our nephrology-dialysis unit.

Optimization of quality of dialysis and Drainage of the pericardium is the rule in the face of abundant effusion and signs of poor hemodynamic tolerance.

Periodic cardiological monitoring, rigorous dose dialysis evaluation, fast diagnosis and treatment of any unexpected medical conditions are the pillars of prevention of uremic pericarditis.

Keywords

Chronic renal failure, Hemodialysis, Pericarditis, Uremia, Tamponade

to raise this cardiac complication through 8 cases of acute pericarditis and to describe its clinical and paraclinical abnormalities to better recognize and manage it in a timely and effective manner in our dialysis patients, before reaching the pre-tamponade or tamponade stage, requiring pericardiocentesis or pericardial surgery.

Materials and Methods

This retrospective study included all cases of acute pericarditis between January 2014 and December 2019 at the Military Hospital Mohammed V, Rabat, Morocco. Out of 567 chronic dialysis patients hospitalized in our dialysis unit, 8 cases of pericarditis were reported.

All patients were > 18-years-old. They were dialyzed in other dialysis centers except one patient. We recorded the following data for each patient: Age, gender, presence of fever, polypnea, dyspnea, chest pain, hypotension, pericardial friction, biological parameters (CRP, troponine, plasma urea, plasma creatinine, plasma albumin, serum calcium, phosphoremia), electrocardiogram, chest radiography, echocardiography abnormalities. The treatment consisted of an increase in the dialysis dose or a pericardial drainage in agreement with the cardiologist. All our patients benefit from an etiological investigation in search of a secondary cause of pericarditis which included immunological assessment (ANCA, AAN, Ac antiphospholipid, FR, C3C4), Infectious assessment (with hepatitis B, C and HIV serologies) and the cytobacteriological study of the pericardial puncture. Follow-up was ensured until the pericarditis disappeared with a second check by echocardiography.

Introduction

Acute pericarditis is a clinical syndrome associated with severe hemodynamic changes in intracardiac fluids and pericardial space. It is a complication of kidney disease which can occur in acute and/or chronic renal failure in pre-dialysis or after renal replacement therapy. In the first years of initiation of renal replacement therapy, its incidence was high and it was considered as a terminal event linked to a high rate of morbidity and mortality [1]. Its potential morbidity and mortality remains considerable, therefore it seemed to us relevant

Ethical approval for this study was provided by the Institutional Ethical Committee of our hospital.

Statistical analysis

Statistical analysis was carried using the Statistical Package for the Social Sciences version 17 (IBM Corp., Armonk, NY, USA). Numerical data were expressed as a mean \pm standard deviation and categorical data in percentage and numerical values.

Results

The prevalence of acute pericarditis was 1.42% including 2 tamponades. The average age is 47 years (22-68 years), 4 women and 4 men. Initial nephropathy: Is hypertensive nephropathy in 03 patients, 01 case of diabetic nephropathy, 01 case of glomerulopathy on segmental and focal hyalinosis complicating sickle cell anemia and one case of chronic tubule-interstitial nephritis. The initial nephropathy remains unknown in 02 patients.

Vascular access: 4 out of 8 patients had femoral catheterization the rest of the patients had radial arterio-venous fistulas. The dialysis dose: For 4 patients, the weekly duration of dialysis was 12 hours spread over 3 sessions per week, the other 4 were not yet on dialysis before hospitalization. The average time to onset of pericarditis after the start of dialysis is 62.4 weeks (0-204 weeks). The circumstances of discovery of acute pericarditis are clinical in 7 patients and incidental in one patient. The functional signs are dominated by chest pain and polypnea, pericardial friction is only present in 50% and hypotension is only present in 5 patients/8.

Repolarization disorders are present and consistent in all leads and without mirror images in all patients, 4 out of 8 patients (50%) have stage I of Holzmans versus 4 with stage III. All standard chest radiographs performed on patients show an enlarged heart shape achieving the typical aspect of carafe cardiomegaly. In addition, 2 X-rays show an associated bronchial syndrome.

All of our patients have had a cardiac ultrasound. Tamponade was found in 2 patients, associated with collapse of the right cavities and dilation of the inferior vena cava which is not compliant. Pericardial effusion is very abundant in 6 patients, medium abundance in two patients (Observations are summarized in [Table 1](#)).

Troponin Ic is high in 2 patients (less than 0.5 ng/ml). CRP is high in all patients. All of our patients have elevated levels of both urea and serum creatinine. Albuminemia is low in 4 patients (< 31g/l). (Biological data: [Table 2](#)). The immunological and infectious assessments were negative.

The cytobacteriological study of the pericardial puncture with etiological aim shows a hematous aspect in 2 patients, sero-hematous in 2 others and citrin in 3 patients. All samples have hyper-cellularity (> 1000/ml),

one of which is predominantly lymphocytic. It is exudate in 5 of our patients. The bacteriological search was negative in 6 patients against a single BK positive in one patient. The anatomo-pathological study of the pericardial puncture fluid in search of malignant cells, this is negative for all patients. In addition, the discovery of an epitheloid and giganto-cellular granuloma without caseous necrosis confirmed the diagnosis of tuberculous pericarditis in a patient.

Therapeutically, 7 patients underwent pericardium drainage by pericardo-centesis in intensive care on the day of hospital admission with an average delay of 2.8 hours (2-4 hours), with an average of drained fluid 1006 ml (250-2000 ml). The clinical and ultrasound evolution was remarkable in 7 patients with 80% decrease in effusion on second ultrasound checks against a single death not directly attributable to tamponade case no 4 ([Table 1](#)).

Discussion

Pericardial effusion is a major complication of the terminal stage of chronic renal failure, it was first described by Richard Bright in 1836 whose prognosis is directly linked to the occurrence of tamponade, which represents the fatal development of pericarditis acute [[1](#)].

The incidence at the beginning of dialysis was very high, 41% of a sample of 83 patients at Peter Brigham hospital, More recently, its incidence has decreased significantly to reach 5-20% in chronic hemodialysis patients, following the establishment of an early, adequate dialysis and the advent of new dialyzers, pericarditis has become a very rare entity, currently, in 13,000 dialysis patients per year, there are only 1 to 2 cases of pericarditis per year which joins the results of our study limited by the number of cases [[2](#)].

Classically, two types of pericarditis are defined in patients with end-stage chronic renal failure, uremic pericarditis which occurs before the initiation of the replacement therapy or in the 8 weeks after its start which is due to the uremic syndrome of patients not yet on dialysis, and dialytic or chronic dialysis patient's pericarditis which occurs beyond the first 8 weeks of locum therapy and which is due to improper dialysis or hydro-sodium overload [[3,4](#)].

Four of our patients were therefore considered uremic pericarditis and the other four had chronic dialysis pericarditis.

Acute non-uremic pericarditis often presents with sudden, anterior chest pain; aggravated by inspiration accompanied by pericardial friction, with over ST shift and change in the T wave. While acute uremic pericarditis has more insidious installation with pericardial friction and the symptomatology is often poor. Hyperleukocytosis and fever are rare [[5,6](#)].

Table 1: Summary of patient observations.

Case	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Age	22	45	68	32	68	65	59	27
Sex	Female	Female	Female	Male	Male	Male	Male	Female
CRF Etiology 's	HSF+ drépanocytose	NH	NH	inconnue	NTIC	ND	N Ind	NH
Weekly duration of dialysis	12 H	Not yet on dialysis	12 H	Not yet on dialysis	12 H	Not yet on dialysis	12 H	Not yet on dialysis
Dialysis adherence	Good	-	Mediocre	-	Good	---	---	----
Vascular access	fémoral KT	fémoral KT	radial AVF	radial AVF	radial AVF	jugular KT	radial AVF	jugular KT
Time of pericarditis after onset of dialysis (weeks)	8	inaugural	320	inaugural	100	inaugural	30	inaugurale
Chest pain	+	+	+	+	-	+	-	+
Dyspnea	+	+	+	+	-	+	+	+
Hypotension	+	+	+	+	+	-	-	-
Pericardial friction	-	-	+	-	-	+	+	+
Electrical signs	Micro-voltage Holzman I	Microvoltage Holzman I	Microvoltage Holzman III	Microvoltage Holzman III	Microvoltage Holzman I	Microvoltage Holzman III	Microvoltage Holzman III	Microvoltage Holzman I
ETT effusion size	-Great abundance -collapse of the RV + OD dilated non-compliant ICV	-Great abundance -collapse of the RV + OD dilated non-compliant ICV	-average abundance -no RV + OD collapse	- Great abundance - no RV + OD collapse	-average abundance -no RV + OD collapse	-Great abundance - no RV + OD collapse	-Great abundance - no RV + OD collapse	-Great abundance - no RV + OD collapse

Pericardial puncture	Aspect	Hematic	Sero-haematic	Hematic h	Sero-haematic	Citrine	citrine	citrine	NOT DONE
	Biochemistry	Exsudate	Exsudate	Exsudate	Exsudate	Exsudate	transudate	Transudate	---
	Cyto-bacteriology	BF (-) BK-	BF (-) BK-	BF (-) BK-	BF (-) BK-	Predominantly lymphocytic hypercytosis BK +	BF (-) BK-	BF (-) BK-	----
	Anatomopathology	MC (-)	MC (-)	MC (-)	MC (-)	-MC (-) -cellular epithello giganteoGE granuloma sans caseous necrosis	MC (-)	MC (-)	----
Treatment		PD	PD	PD	PD	PD	PD	PD	Daily HD
		1500 ml	2000 ml	300 ml	2000 ml	250 ml	1100	300	NON
Evolution		Recovery	Recovery	Recovery	death	Recovery	Recovery	Recovery	Recovery

BF (-): Absence of bacterial flora; MC (-): Absence of malignant cells; PD: Pericardial drainage; CHD: Conventional hemodialysis.

Table 2: Biological results.

Biological parameters	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Initial plasma urea (g/l)	2.8	1.4	1.1	6.12	2.3	4.3	1.8	3.7
Initial plasma creatinine (mg/l)	136	90	74	230	111	157	87	120
Plasma Albumin (mg/l)	23	29	30	32	30	27	23	32
Calcemia (mg/l)	95	88	92	94	98	67	88	71
PTH (pg/l)	45	396,7	90	102	99	318	750	600
Crip (mg/l)	12	18	20	12	33	13	9	28
Troponin (ng/ml)	0.2	0.3	0.36	0.41	0.42	0.35	0.13	0.34

In our patients 80% had presented immediately in a table of tamponade and pericarditis of great abundance announcing by a noisy symptomatology with precordial chest or left basi-thoracic pain.

The specificity of pericardial friction is high, but its low sensitivity as a diagnostic element has already been established [7-9]. Indeed, pericardial friction was only present in 20% of our patients, which agrees with study data [10,11].

The ECG is rarely normal and is the key element of the differential diagnosis with coronary syndromes. EKG abnormalities are common in acute non-uremic pericarditis; rare in uremic pericarditis. Repolarization disorders are mainly limited to an isolated and diffuse sus-ST found in 65 to 85%, was noted in 60% of patients in our series. The ST segment shift into V1 and AVR, classic "auricular mirror", is variably present in 37 to 76% of cases. We can have a normalization of ST with a diffuse inversion of the T wave at a more advanced stage [12,13].

Pericardial inflammation is affecting both the epicardial ventricular and atrial regions. It is a sensitive, early and concomitant sign of ST elevation, but its specificity remains poorly assessed. It was not found in our patients [14].

A chest X-ray can provide information about the presence of cardiomegaly and pericardial effusion. A chest CT scan and a cardiac MRI are not essential for diagnosis if a two-dimensional ultrasound is available [7].

The dosage of troponin must, according to the recommendations of the European Society of Cardiology be systematic in this pathology in order to seek an associated myocardial attack. In our patients it is only very small elevations of troponin which have been recorded (all less than 0.5 ng/ml) and which have not been considered as authentic acute myo-pericarditis but simply as a minimal myocardial reaction [8].

Roubille, et al. observed a normal dosage of reactive protein C (CRP) in 27% of cases among 103 acute pericarditis. In our study; we found that patients with elevated troponin, had also a high initial CRP. This result is concurring with the Hooper, et al. study's [12] which found 40% of high CRP in the initial phase of the symptomatology. This probably indicates a more active inflammatory process [15].

Establishing the kinetics of CRP under treatment, or at least the rate of decrease could therefore be correlated with the control of the inflammatory process, and possibly with the subsequent course. This hypothesis remains to be evaluated.

The evaluation of pericardial effusion is important to confirm diagnosis, assess the prognosis by looking for signs of collapse of the cardiac cavities and poor compliance of IVC, and to evaluate the therapeutic efficacy

insofar that the importance of pericardial effusion is a predictor of surgical drainage of the pericardium.

The importance of pericardial effusion is: Weak if detachment < 10 mm (< 300 mL); Medium between 10 and 20 mm (300 to 500 mL); Important if detachment > 20 mm (> 500 mL) [10].

The immediate prognosis of acute pericarditis is progression to tamponade, which is a diagnostic and therapeutic emergency. Its occurrence appears mainly according to the etiology of pericarditis. Tuberculous, purulent and neoplastic forms are thought to be complicated by tamponade in nearly two thirds of cases. The occurrence of such a complication must therefore lead to an exhaustive etiological assessment which is sometimes invasive [13].

The prevalence of this complication varies from 19 to 22% for the older series to 5% for the most recent series [14,16]. Tamponade is suspected in the presence of tachycardia, hypotension, distension of the jugular veins and an increase in paradoxical pulsations, as well as signs of right heart failure, the progression to tamponade depends on the speed of installation of the pericardial effusion [3].

In our study, tamponade was the method of revelation of acute pericarditis in 2/8 cases. This proportion should raise the alarm signal and lead to more rigorous cardiological monitoring in patients with chronic renal failure. In whom the incidence of tamponade is higher compared to the general population.

The cause of uremic pericarditis is still not well elucidated, however, by comparing our data with those of the literature, it appears that hemodialysis pericarditis is always the consequence of unsuitable purifying treatment, whether it is: Insufficiency related to the dialysis technique: Quality of the vascular access (low flow of arteriovenous access or high recirculation rate), duration of the sessions, dose and quality of dialysis. Indeed 50% of our patients (n = 4) were dialysed from a central catheter, all the patients on dialysis had an insufficient dialysis dose due to poor compliance, especially since the urea level observed in all of our patients testified to ineffective dialysis, or an inadequacy, linked to an increase of catabolism by concomitant pathological condition, in particular infection, previous cardiac condition, surgical procedures, hyperparathyroidism, hypercalcemia and hyperuricaemia. In fact 62.5% of our patients presented with a concomitant pathology with the appearance of pericarditis [2,3].

Several medical treatments have been tested in the management of uremic pericarditis including aspirin, steroidal and non steroidal anti-inflammatory drugs and colchicine, the efficacy of which is controversial [17].

For aspirin and non steroidal anti-inflammatory drugs, they regulate fever but do not improve chest

Table 3: Published work on pericardial effusions in uremic patients.

Studies	Year	Country	Population	Drainage (%)	Death (%)	References
Comty, et al.	1971	UK	25H	16	12	[26]
Kramer, et al.	1975	Germany	10H ; 7PD	6	18	[27]
Minuth, et al.	1975	USA	11H	27	0	[28]
Luft, et al.	1980	USA	15H ; 11 CKD	35	Non dispo nible	[29]
Spector, et al.	1983	USA	10H ;5PD ;9 CKD	21	13	[17]
Peraino, et al.	1983	USA	18H ; 4PD	64	9	[19]
De pace, et al.	1984	USA	97 CRF	23	8	[20]
Rutsky, et al.	1987	USA	93H ;20 PD ; 23 CKD	46	10	[24]
Leehey, et al.	1989	USA	32H ; 4PD ; 11 CKD	34	4	[21]
Gafter, et al.	1990	Israel	25 H or DP	52	4	[22]
Banerjee, et al.	2006	UK	5H ; 1DP ; 1 CKD	100	14	[23]
Tseng, et al.	2009	Taiwan	88H	16	11	[25]
Stanislas, et al.	2013	France	19H ; 3DP ; 22 CKD	45	2	[10]
Hajji, et al.	2015	Tunis	46H	0	2	[11]
Notre étude	2016	Maroc	3H ; 2 CRF	7	1	-

H: Hemodialysis; PD: Peritoneal dialysis; CKD: Chronic kidney disease; CRF: Chronic renal failure.

pain, pericardial rubbing, need for surgical treatment, or mortality; with the cost of increasing the adverse digestive effects.

In a Cochrane review on the use of colchicine in pericarditis, Alabed, et al. compared the effect of colchicine with indomethacin and corticosteroids and concluded that it is effective in recurrent pericarditis, but this was based on a small number of trials [18].

There is a general consensus that the initial attitude to uremic pericarditis is to increase the duration and frequency of dialysis without circuit heparinization. Some studies have shown that more often, this attitude is sufficient to manage this pathology with complete disappearance of symptoms and effusion [2,3,19-24].

Few studies have evaluated the optimal time of pericardial drainage, however, most authors necessarily state the indication for emergency drainage in the event of clinical or ultrasound signs of tamponade [25].

S. bataille and Al, tried to determine the predictive factors of pericardial drainage which are the size of the effusion and the signs of poor hemodynamic tolerance, that match with our study in which 25% of patients presented with both clinical and ultrasound signs of tamponade, 50% had high abundance effusion without signs of tamponade, 25% with moderate and low abundance effusion, and 20% with poor hemodynamic tolerance, 7 patients were drained and one patients was daily dialysed (Table 3).

In addition, the albumin level seems to be a risk factor for drainage as well, in a recent study of 44 patients, evaluating the predictive factors of pericardial drainage confirms this theory, 35% of their patients having an al-

buminemia level. Less than 31 g/l were drained against only 7% having a rate greater than 31 g/l, the results of our study seem to confirm this theory with 80% of cases having an albumin rate lower than 31 g/l [8].

Overall, the prognosis for uremic pericarditis is excellent, with a survival rate of 85-90%. Successful pericardial decompression is associated with a rapid improvement in blood pressure and a decrease of tachycardia [26] (Table 3).

Conclusion

The study of 8 cases of acute pericarditis occurring in patients with end-stage chronic renal failure treated by chronic hemodialysis, compared with classic and recent data from the literature, led to the following conclusions:

From an ethiopathogenetic point of view, chronic hemodialysis pericarditis is due to insufficiency linked to the dialysis technique: Deficient vascular approach, an insufficient dose of dialysis; Or by inadequacy linked to a specific increase of the nitrogenous catabolism, in particular by intercurrent infectious process.

The increase in the duration and frequency of dialysis is effective, however pericardial drainage is mandatory if clinical or ultrasound signs of poor hemodynamic tolerance are present, abundant effusion associated with an albuminemia level of less than 31 g/l seems also to be a predictor factor of surgical drainage.

So we propose, in addition to periodic cardiological monitoring, regular control of the dialysis dose by Kt/V, diagnosis and rapidly effective treatment of any unexpected pathological condition, accompanied by a readjustment of the purifying treatment.

Although acute pericarditis is treatable, it also isn't always prevented. Its timely recognition and efficient management are essential elements in the success of the treatment.

Authors Declaration

No conflicts of interest.

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