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RESEARCH ARTICLE

DIABETIC KETOACIDOSIS IN THE PEDIATRIC INTENSIVE CARE UNIT : CLINICAL, EPIDEMIOLOGICAL AND EVOLUTIONARY PROFILE

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Abstract

Diabeticketoacidosis (DKA), a seriousmetabolic complication of diabeteslinked to insulindeficiency,isstillcommoninAfrica,particularlyinchildren,andrepresentsamajormedicalemergency. It represents a real public healthproblem in ourcontext. Our study highlights the epidemiological and clinical profile of childrenadmitted to intensive care for DKA, underlining the importance of early recognition of warning signs, rapid and protocolized management, and close monitoring of potential complications. A retrospectivestudywascarried out over a 12- monthperiod in the pediatric intensive care unit at the Abderrahim Harouchimotherchild Hospital, Chu ibn Rochd, Casablanca, and included 43 cases of DKA. The meanage of the patients was 6.7 years, with a femalepredominance. DKAwas inaugural in 58% of cases, and 39% of episodesweretriggered by infections, mainlyurinary and pulmonary. Clinically, 83% of childrenpresentedwithneurologicaldisorders,and68%withKussmauldyspnea.Severeacidosis (pH< 7.1)wasobservedin41% of cases,whileelectrolyteabnormalitiesincludedhyponatremia and hypokalemia. Treatmentwasbased on rehydration, insulintherapy, correction of fluid andelectrolyteimbalancesandtreatmentofthecause.Progressionwasfavorablein93%of patients,butthreedeathswererecorded,underliningtherisksassociatedwithdelayeddiagnosisandcomplications.Thisstudyhighlightstheimportanceofraisingawarenessofearlydiagnosis and prevention of precipitatingfactors, notably infections and therapeutic non- compliance, in order to improve management and reduceDKA-relatedmortality.

Introduction:-

Diabeticketoacidosis(DKA),describedin1874byAdolfKussmaul,wasonceamajorcauseof mortality in diabetics, beforedecreasingsignificantlyafter the discovery of insulinin 1921 by Banting and Best[1]. The term "ketoacidosis" replaces "ketoacidosis", as the accumulation ofketonebodiesprecedeacidosis[2].Diabeticketoacidosisis the mostseveremetabolic complication of diabetes, linked to insulindeficiency, causingketone body accumulation and sometimes fatal metabolic acidosis[3]. It is inauguralin15-70%ofcasesinthepediatricpopulation,andlinkedtoanimbalancein1-10% of knowndiabetics[4]. Itsfrequencyremains high

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in Africa, withsignificantmorbidity and mortality, linked in mostcases to cerebraledema (0.3 to 1% of cases),responsible for 60 to 90% of deaths and neurocognitive sequelae[5,6]. The cost of treatment per episodeisestimated at between$6,500and$7,500intheUnitedStates[7]

Given the importance of this public healthproblem, the aim of thisworkis to specify the incidence of thesediabeticketoacidosisattacks in ourcontext, as well as to frame thisphenomenonwithepidemiological,clinical,biological,therapeuticandevolutionarydata,with the aim of betterunderstandingthispathology and improving the management of this type of patient.

Materialsand Methods:-

Thisisaretrospectivedescriptivestudyofcasesofdiabeticketoacidosisinpediatrics, carried out over a 12-month period in the pediatric intensive care unit of the Mère-EnfantAbderrahimHarouchiHospital,ChuibnRochd,Casablanca.Weincluded43cases, regardless of age or sex. Using an evaluationformbased on patients' medical records, weanalyzedtheepidemiological,clinical,etiologicaland evolutionary profile, as wellas therapeutic management.

Results:-

Epidemiologicalaspects:

Overthe12-monthstudyperiod,43patientswerehospitalized for diabeticketoacidosis. The total number of patients hospitalizedduringthisperiodwas 723. The prevalence of diabeticketoacidosiswas 5.94%. Among the ketoacidosis, 58.13% were inaugural, and 41.48% of the patients wereknown type 1 diabetics. The averageage of theirdiabeteswas 3.32 years ,10 childrenhad a diabetic parent, i.e. 23.25%.Therewere25girlsand 16boys,witha femalepredominanceof58% anda sex ratio of 1.56. The averageageof ourpatients was 6.7years,withextremesof 9 months and14 years.Figure1Ishowstheprevalence ofketoacidosisbyagegroup.

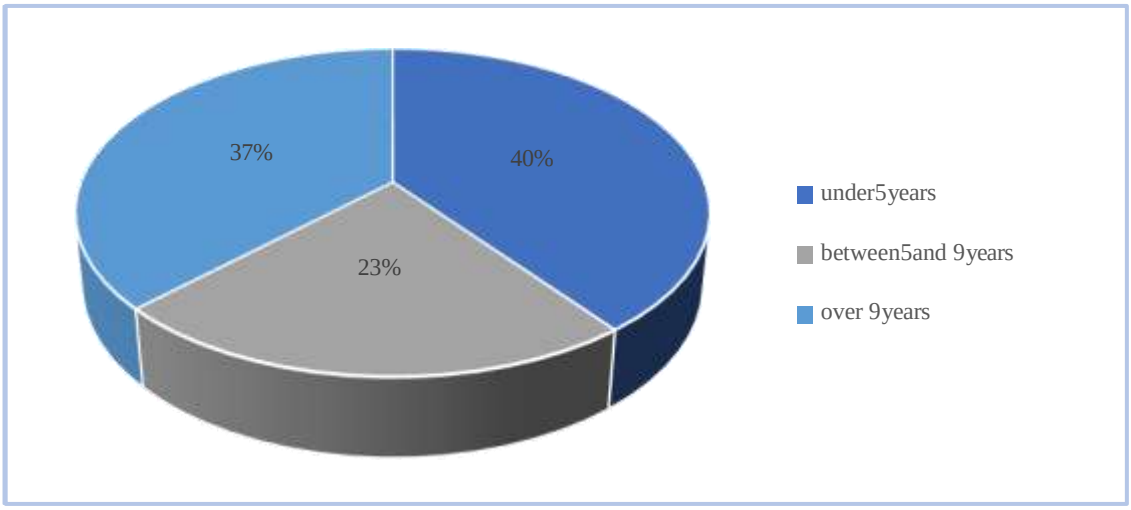

Figure 1:-Prevalenceofketoacidosisbyagegroup.

Clinicalandparaclinicalaspects

The average consultation time in ourserieswas 6.35 days. Weobserved 36 cases ofneurologicaldisorders, representing 83.72% ofthepatients studied. Thesedisordersoftenmanifested as obnubilation or somnolence,notedin 66.66%,while 27.9% of patients had a GCS score of lessthan or equal to 12/15ths. Kusmaul'sdyspneawasalsofrequent, notedin 68.13% of cases, while more thanhalf the childrenweredehydratedandpresented anabdominal picture,notably abdominal pain and vomitingin 48.7% of cases. Figure 2 shows the differentclinical manifestations of acidketosisimbalancepresented by our patients.

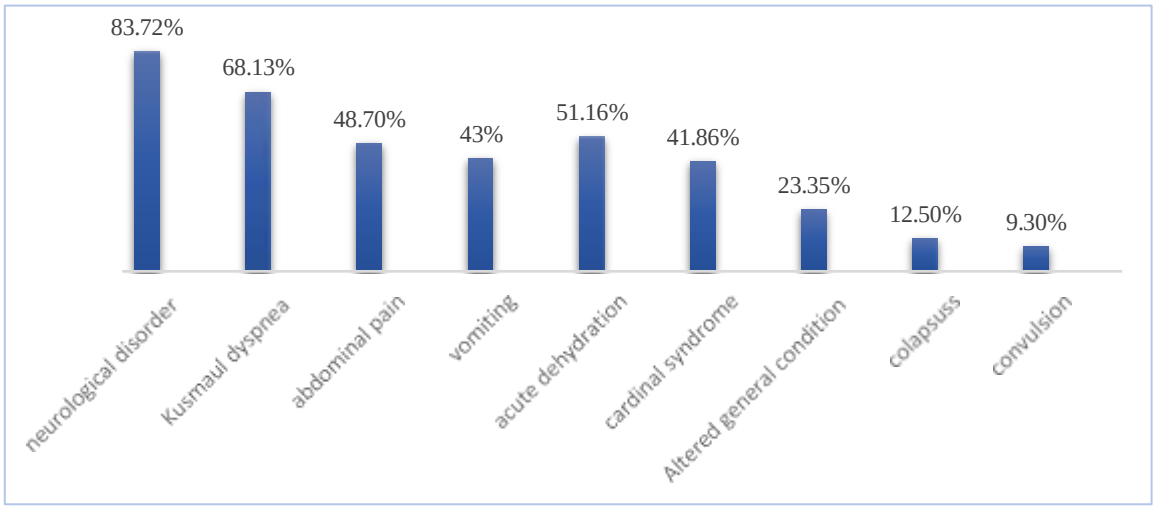

Figure2:- Distributionof clinicalmonkeyson admission.

Allpatientshadbloodglucoselevels≥ 3g/l,associatedwithketonuriaandglycosuriaat twocrosses.Geometrywasperformedin22patients,revealingsevereacidosiswithpH< 7.10 in 40.9% of cases. In the absence of geometry, a systematic bicarbonate assaywascarriedoutinallpatients.Ofthese,37.20%hadbicarbonatelevelsbelow5mmol/l, 34.88% between 5 mmol/l and 9 mmol/l, and 27.9% between 10 and 18 mmol/l. The fluid and electrolyte balance on admission showedtruehyponatremiain 16.27% of patients, and hypokalemiain 9%. Hyperkalemiawasobserved in only one patient. CerebralCTscansperformedonadmissionrevealedcerebraledemain11.62%ofcases.

Etiologicalaspects:

Althoughtwo-thirdsofourpatientspresentedwithacid-ketoticdecompensationlinked to inaugural diabetes, a causecouldbeidentifiedin 83.16% of cases. The infectiouswork-up, guided by clinical and biologicalelements, establishedthat infection wasresponsible for decompensationin 17 cases, i.e. 39.35% of patients. Amongthese infections, urinary tract infection was the mostfrequent (41.17%), followed by pleuropulmonary infections (35.29%) and intrameningeal infections (23.5%). ENT infectionswerefoundin5.83%ofcases.Inaddition,non-adherencetotherapyanddietdeviationswereobservedin 44% of patients. Figure 3 shows the different groups of decompensationfactors.

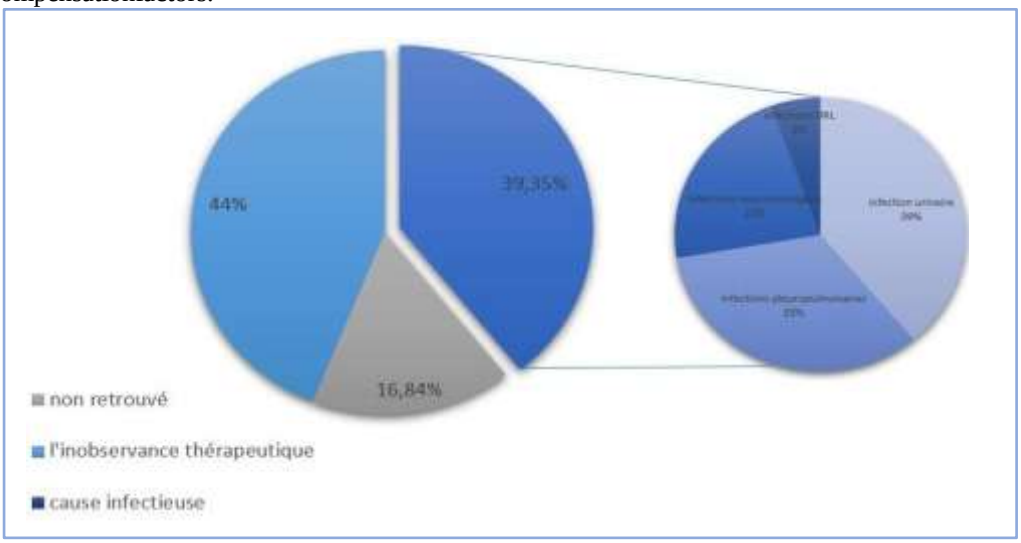

Figure3:-Factorsleadingtodecompensation.

Evolutionaryaspects:

The averagehospitalstaywas 4.3 days, withextremesrangingfrom 2 to 22 days. The outcomewasfavorablein93.03%ofcases.However,theoutcomewasfatalin6.97%of cases (3 patients). The first patient

was a 6-month-old infant whodied of respiratorydistress due to congenitalheartdisease. The othertwo patients, aged 5 and 14 respectively, died of septicshock.

Discussion:-

Diabeticketoacidosisis the mostseveremetabolic complication of diabetes. It iscommon in Africa, affecting 20 to 50% of diabeticchildren, whereasitis rare in the West, with an incidence of 0.3 to 1.3%, inauguratingdiabetesin 25 to 30% of cases [8,9]. In Morocco, the inauguralDKArateis77%accordingtothestudybyElYoubiS. inFez[10],and59.13% inourseries, highlighting the lack of awarenessamongfamilies and the importance of this complication, whichconstitutes a major public healthproblem.

Overthepast30years,theprevalenceoftype1diabeteshasincreasedconsiderablyin childrenunder 5 years of age[11,12]. However, the frequency of ketoacidosisincreaseswithage, peakingaroundpuberty (10-14 years) [13,10].These data are consistent withourseries, where 37.20% of patientswereover10yearsold,thiscouldbeexplainedby insulinresistancecausedbygrowthandsexhormones,reducingitsefficacyby30-50%[14,15].Inaddition,thesocialchangesandstress associatedwiththisperiod can affect adherence to treatment and glycemic control.

Theliteratureshowsthatthereisnosignificantdifferencebetweenthesexesintheoccurrence ofdiabeticketoacidosis,althoughslightvariationsexistbetweencountriesduetodemographicfactors[13,16]. Our seriesshowed a femalepredominance, with an F/M ratio of 1.56.

Afamilyhistoryofdiabetesenablesdiabetestobediagnosedmorequicklyinchildren,therebylimiting the severity of complications. In ourstudy, 23.25% of cases had a familyhistory of diabetes, a figure similar to thatobserved in European studies[17].

Clinically,theconditioninitiallymanifestsitselfasaworseningofclassicdiabeticsymptomssuch as polyuria, polydipsia and weightloss,with or withoutpolyphagia. Thesesigns are followed by various complications, includingdehydration,sometimesdifficult todetect, and vitalsignssuch as tachycardia, and Kussmaul'sdyspnea, characterized by deepbreathing and acetone-smellingbreath, isalsocommon. Othersymptomsincludenausea and vomiting, often the reason for consultation, and abdominal pain thatmaysuggest a surgical abdomen. At an advanced stage, confusion and somnolence appear, and mayprogress to loss of consciousness[18,13,10,19]. In ourseries,symptomsonadmissionweredominatedbyadisturbanceofconsciousnessin92.25%of cases, of which 32.25% hada Glasgow score of lessthan 12/15, followed by dyspnoea and moderate to severedehydration in more thanhalf the patients.

Classically,the diagnosisofDKAisbasedonbiologicalcriteria:bloodglucose ≥2g/L(11mmol/L), glycosuria ≥2 crosses, ketonuria ≥2 crosses, pH < 7.30 or alkalinereserve< 15 mmol/L[20].

In general, capillaryblood glucose levels are oftenabove 4g/l, and mayevenexceed 10g/l. However,whenhigherlevelsarereached,hyperosmolardecompensationistobesuspected, despiteitsrarity in children[2(1).]

According tothe recommendations of ISPAD (InternationalSociety of Pediatric and Adolescent Diabetology)[23]:theseverityofketoacidosisisthenclassifiedintothreegroups,accordingtothe degree of acidosis, often indicative of a delay in diagnosis (table 1)

Severityofdiabetic ketoacidosis	**Acidosis**	**Bicarbonate**
Mild	PH7.2-7.29	10mmol/Lto<18 mmol/L
Moderate	PH7.1-7.19	5mmol/Lto9mmol/L
Severe	PH<7.1	<5mmol/L

Table1:-Determiningtheseverityofdiabeticketoacidosis.

Identifying the triggers of decompensationrequires a full investigation, fromquestioning to paraclinicalexaminations. Failure to recognizethem can compromise prognosis,andleadtopersistenceorrecurrenceofdecompensationdespiteappropriatetreatment. The frequency of infection as a triggering factor is a constant fact in Africa, sometimesaccounting for over 70% of cases [22]. These infections are highly variable in nature, and the localizationsclassicallyfound are ENT, bronchopulmonary, urinary, gastric,

cutaneous and gynecological[22]. Non-compliance withtherapy, includinginsulin discontinuationorincorrectdosageanddietaryerrors[25,2],wasobservedin20.6% ofour patients,a higher ratethan in otherMoroccanstudies: 10.41% inFez[14]and 5% in Rabat [22],aswellasinMaliwith13%[13].Thisfactor,frequentindevelopingcountries,underlines theimportanceofimprovingthesocio-economiclevelanddiabeticeducationofpatients and theirfamilies. Otherfactorsthat can beidentifiedinclude situations of physical or psychologicalstress,suchasatrauma,oremotionalshocks,surgicalintervention,aswell ascertainmedications,suchasglucocorticoids.Eatingdisorders,particularlyingirls,and denial of the diseaseduring the peripubertalperiodmayalsobepotential causes [24].

The cause ofdecompensationmust betreatedsimultaneouslywiththe ketoacidosis. In children, episodes of DKA are often inaugural, althoughtriggeringfactors, such as infections and poor compliance withtreatment, can beidentified. This justifies the administration of appropriateantibiotics, the resumption of diabeteseducation, or supportivepsychotherapy for adolescents in denial of the disease[25].

Emergency treatmentisbased on four key actions: rehydration, insulintherapy, correction of hydro electrolyticdisorders and treatment of the triggering cause.

According to ISPAD (Clinical Practice Consensus Guidelines), the correction of fluiddisorders can besubdividedintotwo components[30] : initial filling and intravenousrehydration.

In the absence of initial shock, infusion of 0.9% saline, 10-20 ml/kg over 20-30 min, shouldbestartedimmediately to restore peripheral circulation[23]. If shockispresent, infusionshouldbeperformedwithboluses of20 ml/kgof0.9%saline[23].In practice,the ISPAD[30]recommendationsproposeasimplifiedmethodforensuringrehydration,based on the child'sweight. (Table 2)

Weight	Input
11-20kg	1000mL+50mL/kg/24 hforeachkgbetween11and20
>20kg	1500mL+20 mL/kg/24hforeachkg>20

Table 2:-24-hourmaintenanceflowratebyweight[30]

It isrecommended to start rehydrationwith 0.9% isotonic sodium solutions (physiologicalsaline),changing to5% glucose serum if bloodglucose drops to 2.5-3 g/l.

Insulintherapyis the cornerstone of treatment for ketoacidosis, and the latest ISPAD recommendations (2022) insistthatinsulintherapyshouldbestartedafter one hour of intravenous rehydration[30] . Insulin administration shouldbecontinuous and regular, usingaself-propelledsyringeorpump,atarateof0.05to0.1units/kg/hour,takingintoaccount the child'sage and the severity of the acidosis[(23).]

	Children≤5ans	Children> 5 years
LightACD(PH>7.2)	0.03IU/kg/h	0.05IU/kg/h
ModerateorsevereDKA	0.05IU/kg/h	0.1IU/kg/h

Table 3:-Insulin therapydoseaccordingtoISPAD recommendations[30]

If blood glucose levelsfalltoorapidlybeforeacidosisiscorrected, itisadvisable to increasetheserumglucoseflowratewithoutreducinginsulininfusionaslongasacidosispersists, particularly if PH < 7.20[4].

Insulintherapyshouldbecontinuedaslongasacetonemiapersists,maintainingblood glucose levels at around 2 g/l. Switching to subcutaneousinsulintherapy can onlybeconsidered if: the patient can eatproperly, blood glucose< 2.5g/pH>7.30,$Hco3^-$> 18mEq/the switch to subcutaneousinsulin must be made 30 minutes beforestopping the insulin infusion.

Itisadvisable to check blood potassium levelsbeforestartinginsulintherapy,as insulindisplacesintracellular potassium, which can rapidly lead to hypokalemia[29] . Specificrecommendationsexist for potassium intakeaccording tokalemialevels[25,23]. Similarly, althoughserum phosphate concentrations are generally normal at the outset, intracellulardepletionisfrequent, leading to a drop in serum concentrations duringtreatment ofketoacidosis[25]. Thus, phosphate supplementationisrecommendedwhenserum concentration isbelow 10-15 g/dl (0.3-0.5 mmol/l).

Intravenousbicarbonatesarenotroutinelyrecommendedforchildrenwith DKA,asthey have not been shown to improvebiochemicalresolution time, hospitalstay or mortality[25]. Despite the serious complications associatedwithsevereacidosis, such as ventricularrhythmdisturbances,negativeinotropism,peripheralvasodilation,aswellas liver and brainfailure, the use of bicarbonates remainscontroversial. This treatmentmayincreasetheriskofhypokalemia,slowtheresolutionofketosis,causeaparadoxicalincrease in cerebralacidity due to the rise in tissue partial pressure of carbondioxide (pCO2), and increase the risk of brain damage[26,25]

However, as shown in the 2006 study by N. Glaser et al., almost 50% of ketoacidosis patientsdevelopmildorevenasymptomaticcerebraledema[(27).] Inadditiontotheriskof mortality, thesedisorders are associatedwithsignificantmorbidity over the long term(28).

Thankstotheaccumulationofdataondiabeticketoacidosis,understandingthispathology and improvingits management have led to a considerablereduction in itsmortalityrate.Thisrate,whichexceeded95%in1923,isnowlessthan1%indeveloped countries. However, in developing countries, it can stillvarybetween 3% and 13%[25,23].

Conclusion:-

Diabeticketoacidosisrepresents a major metabolic emergency in pediatric intensive care, particularly in developing countries wheredelays in diagnosis and difficulties in access to care worsenitsprognosis. The results of ourstudy are a reminder of the importance of rigorous follow-up of diabetic patients, notablythroughtherapeuticeducation of patients and theirfamilies. Regular follow-up by a multidisciplinary team not onlyhelpspreventDKArecurrences, but alsoensures a betterquality of life for patients and theirfamilies. Efforts must alsobepursued to strengthendiabetesawarenesscampaignsinchildren,developearlydetectionstrategiesandimproveaccess to appropriate care, in order to reduce the incidence and severity of DKAepisodes.

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