

SOUTH AFRICA / JUDGMENT

M.L v Member of the Executive Council for Health, Eastern Cape (501/2017) [2022] ZAECBHC 40; [2023] 1 All SA 475 (ECB) (15 March 2022) [2022] ZAECBHC 40

COURT

Eastern Cape High Court, Bhisho

BEFORE

M Notyesi

CASE / FILE NUMBER

501/2017

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IN THE HIGH COURT OF

SOUTH AFRICA

(EASTERN

CAPE LOCAL DIVISION, BISHO)

REPORTABLE

Case

Number: 501/2017

Date

of Hearing: 26, 28, 29 & 30 April 2021

18

November 2021

Date

of Delivery: 15 March 2022

In

the matter between: M[....]
L[....]
PLAINTIFF
and
MEMBER
OF THE EXECUTIVE COUNCIL FOR
HEALTH,
EASTERN
CAPE
DEFENDANT

JUDGMENT

NOTYESI AJ:

Introduction

[1] The plaintiff, Ms M[....] L[....], claims delictual damages in her own name and on behalf of her minor child, E[....] (E) against the defendant, the Member of the Executive Council for Health, Eastern Cape Province (the MEC). The claim emanates from the child suffering cerebral palsy as a consequence of a hypoxic-ischaemic event during the birth process. The plaintiff bases the claim on allegations of medical negligence by the medical and/or nursing staff of Mbekweni

Health Care Centre, Vidgesville Clinic, Mthatha General Hospital and/or Nelson Mandela Academic Hospital.

[2] The main contention of the plaintiff is that the medical practitioners and nursing staff at the Vidgesville Clinic, the Mbekweni Health Care Centre, Mthatha General Hospital and or Nelson Mandela Academic Hospital treated her and her child in a substandard manner and were in many respects negligent in carrying out their obligations towards the plaintiff and her child and that such substandard or negligent treatment caused the plaintiff and E to suffer damages for which the defendant must be held liable. The plaintiff further contends that the substandard treatment and negligent acts were committed by those nursing staff and medical practitioners who were acting within the course and scope of their employment.

[3] In response to the plaintiff's case, the defendant made a general plea in which negligence and causality are denied. The parties had narrowed down the trial issues to the question of causality pursuant to the pre-trial procedures and expert advice. The main dispute being whether E had a pre-existing medical condition that had caused the cerebral palsy or whether the cerebral palsy was caused as a result of the negligence by the medical staff and/or nurses.

- [4] By agreement between the parties, the question of the quantum of the plaintiff's claim is to stand over and at the commencement of these proceedings, the parties sought an order separating the merits from the quantum.
- [5] Although I heard the evidence in this matter on 26, 28, 29 and 30 April 2021, I directed the parties to file heads of argument and that agreement be reached on when I could hear oral submissions. When the parties could not reach an agreement on a mutual date, I directed the parties to prepare a joint practice note for the determination of the trial issues based on the heads of argument. In the joint practice note received on 18 November 2021, the parties requested for the disposal of the matter based on their respective heads of argument. This court is indebted to counsel for their comprehensive heads of argument, which has assisted in the preparation of this judgment.

Background

- [6] The allegations of negligence during the birthing process of E, child of the plaintiff, between the Mbekweni Health Care Centre, Vidgesville Clinic, Mthatha General Hospital and, later Nelson Mandela Academic Hospital is the crux of adjudication. Unfortunately, there are no complete official records from the hospitals and clinics for the labour and birth period. In this regard, I do emphasize that there is an obligation upon the defendant's employees to keep clinic and hospital notes pertaining to the plaintiff's treatment. This aspect will be dealt with later in the judgment.

Admissions

- [7] The parties had reached an agreement on several issues pertaining to the trial. Briefly, I set out some of the agreed facts:

7.1 The plaintiff made about four antenatal visits to the Clinic prior to the birth of E on 17 December 2007.

7.2 On 15 December 2007, at approximately 23h00, the plaintiff started to experience labour pains.

7.3 On 16 December 2007, the plaintiff arrived at Vidgesville Clinic at 07h00 and was examined soon after arrival at about 07h30 by way of a vaginal examination, a foetal heart rate and blood pressure tests.

7.4 The plaintiff's water broke at 18h00 and the "toilet like liquid" was coming out. This is identified as meconium.

7.5 The plaintiff was advised that she would be transferred to Mthatha General Hospital.

7.6 The plaintiff arrived at the Mthatha General Hospital at 20h00. At that stage, she was advised that her womb is still closed and she was admitted to the ward.

7.7 The plaintiff was left on her own until she felt something pushing in her vagina and this took place at approximately 04h00 on 17 December

2007. According to the plaintiff, she was assisted by a student nurse with the delivery of E.

7.8 E did not cry at birth and was rushed to an incubator. E started having seizures almost immediately. E was then transferred to Nelson Mandela Academic Hospital.

7.9 E was nursed in an incubator.

7.10 E had been on treatment with Phenobarbitone to suppress seizures since then, but the seizures are still recurring.

7.11 The road to health chart indicates that E was born on 17 December 2007 with a birth weight of 3 400 grams. E suffered from meconium aspiration, low APGAR, however the actual APGAR scores were not given.

7.12 On a proper calculation of time, the plaintiff was in labour for a period of more than 22 hours prior to the birth of E and that there was no monitoring by nursing or medical staff at the hospital.

The Pre-Trial Conference

[8] In a pre-trial conference minutes dated 26 November 2019, several helpful admissions were also made by the parties with a view to narrow down the trial issues. I do record these admissions, which were confirmed by the parties in a meeting of 25 February 2020. The importance of the pre-trial minute is that it later forms the basis of the expert joint minute.

“Pre-Trial Minutes of 26 November 2019

1. The parties agree that the plaintiff who is also known as N[...] was in labour and presented at Mbekweni Health Care Centre where she was admitted.
2. The parties agree that at Mbekweni Health Care Centre the plaintiff registered herself as N[...] L[...] and her admission took place at approximately 06h00 on 16 December 2007 and was assessed for the first time at approximately 07h30 and found to be 2 cm dilated.
3. The parties agree that the plaintiff was assessed for the second time in the evening of 16 December 2007 and a toilet like liquid was running out of her vagina upon breaking of waters.
4. The parties agree that the plaintiff was then transferred to Mthatha General Hospital where she arrived at approximately 20h00.
5. The parties agree that at Mthatha General Hospital the plaintiff, who was in labour with strong contractions was admitted at the labour ward overnight with the IV line running and was found to be 2 cm dilated and was informed that she was not ready to deliver.

6. The parties agree that the plaintiff gave birth approximately 04h00 on 17 December 2007 at Mthatha General Hospital by normal vaginal delivery.
7. The parties agree that the plaintiff was in labour for a period of more than twenty two (22) hours with no proper monitoring.
8. The parties agree that the National Guidelines state that the fetal heart rate should be monitored half-hourly in labour and that a second stage of an hour or longer should be reported to a doctor.
9. The parties agree that the birth weight was 3.4 kg, and Apgar scores were recorded as low and the diagnosis recorded in the Road to Health Chart was “Meconium Aspiration with low Apgar”.
10. The parties agree that at birth E[....] did not cry, was weak, had swelling on the head, immediately had seizures, was rushed and placed in the incubator, given oxygen and was transferred to Nelson Mandela Academic Hospital.
11. The parties agree that at Nelson Mandela Academic Hospital, E[....] was admitted in the neonatal high care unit, the plaintiff was informed that E[....] had seizure soon after birth, was floppy, had an intravenous drip and was fed via a nasogastric tube which was eventually removed on 21 December 2007.
12. The parties agree that E[....] was discharged from Nelson Mandela Academic Hospital on 27 December 2007.
13. The parties agree:
 - 13.1 That the Road to Health Chart discovered by the plaintiff be admitted as evidence and is what it purports to be subject to the right of either party to dispute the correctness or authenticity thereof on notice to the other party (which may be in medico-legal report).
 - 13.2 That pursuant to the medico-legal report of defendant’s expert, Dr Keshave, -
 - 13.2.1 At the time of birth the following problems were noted;
 - 13.2.1.1 MAS – Meconium Aspiration Syndrome; and
 - 13.2.1.2 Low Apgars.
 - 13.2.2 That based on the RTHC the presence of Meconium Aspiration Syndrome seems the most likely cause to have resulted in the low apgars and this would have caused the neonatal Encephalopathy.
 - 13.2.3 That based on the first genetic test, CGH Array – done at Ampath Laboratories on 18 March 2019 indicated that there was no underlying abnormalities, this would make an underlying genetic

condition and also a neurometabolic condition less likely.

13.2.4 That Dr Keshave accepts that the clinical picture in the MRI scan is supportive of prolonged partial hypoxic ischemic injury.

14. The parties agree that from the available hospital records, the clinical picture and the MRI picture, there are no antenatal or postnatal causes that contributed to the brain injury of E[...].
15. That pursuant to the medico-legal report of the plaintiff's expert Prof Lotz, the parties agree that-

15.1 The report of Prof Lotz reflects that genetic disorders and inflammatory, brain diseases are unlikely as causes of the child's brain damage.

15.2 The MRI is supportive of a diagnosis of prolonged partial hypoxic ischaemic injury in a brain.

16. That pursuant to the medico-legal report of defendant's expert Dr Redfern, the parties agree that –

16.1 the perinatal period is the most likely time period when brain injury may have occurred.

16.2 the presence of a neonatal encephalopathy in the first few hours to days after birth strongly points towards a perinatal brain injury.

16.3 the evidence suggests that, when a neonatal encephalopathy is present after delivery, intrapartum factors can almost always be said to be the main cause of the encephalopathy.

16.4 the history of seizures, and the plaintiff's description of E[...]'s condition after birth, suggests the presence of a neonatal encephalopathy even in the absence of the neonatal medical records. Specifically, the description of E[...] as not moving or crying, and being unable to breastfeed and requiring tube feeds, all point towards a diagnosis of NE, together with the history of neonatal seizures.

16.5 the most likely explanation for E[...]'s neonatal encephalopathy and subsequent brain injury was hypoxic ischaemic brain injury, which most likely occurred during the intrapartum period.

17. That pursuant to the medico-legal report of defendant's expert Dr Mugerwa- Sekawabe, the parties agree that –

17.1 although the plaintiff was not advised on the reason for the transfer to Mthatha General Hospital the description of the colour of the amniotic fluid which was draining, suggests that it was meconium staining of the liquor which necessitated the transfer.

17.2 thick meconium stained liquor is associated with an increased risk of fetal distress.

17.3 it is well known that contractions during labour lead to a reduction in oxygen supply to the placenta and in turn to the fetus.

17.4 in view of the increased risk of hypoxia and fetal distress in setting of meconium stained liquor, as was the case in this matter, the non- monitoring of the Fetal Heart Rate probably resulted in abnormalities of the Fetal Heart Rate which accompanied fetal acidosis going undetected.

17.5 if there was a proper monitoring it would have been possible to detect those unfavourable factors which would have necessitated interventions.

17.6 the failure to undertake these observations, in line with acceptable standards and also not instituting measures of clearing the baby's airways of the meconium fluid at delivery so as to mitigate against meconium aspiration amounted not only to substandard care but was also a breach of the duty of care.

18. Subject to the foregoing, in the absence of maternity records and any grounds for dispute, it is agreed that the medico legal reports of Professor Lotz, and Dr Mugerwa-Sekawabe are admitted as evidence without formal proof, including all the opinions, facts, reported facts and conclusions reflected therein.”

Expert Joint Minutes

[9] The joint minutes were signed by Dr A Redfern and Dr A Keshave dated 3 March 2020. Dr A Redfern is a developmental paediatrician expert for the plaintiff. Dr A Keshave is a paediatric neurologist for the defendant. The two experts based their joint agreement on the admissions contained in the pre-trial minute signed on 25 February

2020. That pre-trial minutes embodied the agreements set out in the pre-trial of 26 November 2019, as set out above. I highlighted the admissions in the joint minutes and the agreement of both the experts in relation to the admitted facts.

“9.1

(a) The parties agree that the plaintiff was in labour for a period of more than twenty-two hours with no proper monitoring.

(b) The parties agree that the birth weight was 3.4 kg, and Apgar scores were recorded as low and the diagnosis recorded in the Road to Health Chart was “Meconium aspiration with low Apgar.

9.2

(a) With regards to the possibility of a neurometabolic cause of the NE, the experts agree that the presence of encephalopathy from immediately after birth is not in keeping with an inborn error of metabolism (IEM).

9.3

(a) With regards to the results of the metabolic tests currently available:

· The amino acids and organic acid profiling done on 3/12/19 were “not suggestive of an amino aciduria, amino academia or organic

aciduria.”

· The homocysteine level was very slightly elevated, and red cell folate and Vitamin B12 levels were normal.

· The experts agree that the homocysteine level is not clinically significant, and may be the result of technical factors such as lack

of fasting, or due to E[....] being on sodium valproate therapy for treatment of her epilepsy.

9.4

(a) The experts agree that before a final decision can be made regarding the likely aetiology of the child’s current condition, the results of the following investigated should be reviewed:

(i) Serum lactate / pyruvate ratio;

(ii) Carnitine profile (already performed);

(iii) Repeat serum amino acids (already performed);

(iv) Whole exome sequencing.

9.5 The experts further agree that Prof Mary Rutherford, expert neonatologist and neonatal neuroradiologist, should be asked to comment on the MRI images.

9.6 The experts agree that based on the neonatal history, the clinical examination of the plaintiff’s child, the MRI findings, should the above investigations not be conclusive of any underlying condition, then prolonged partial intrapartum asphyxia resulting in hypoxic ischaemic encephalopathy after birth is the most probable cause of E[....]’s neurodisability.”

[10] Pursuant thereto, E had been subjected to several tests at the instance of Dr Keshave. In this regard, I refer to the order issued by Justice Stretch, of which I quote therefrom the relevant paragraphs set out below:

“1. It is recorded that previously the defendant subjected the minor child for some tests and the reports dated 10 April 2019, 3 December 2019 and 25 February 2020 were filed in this Court.

2. It is further recorded that the defendant has, for the fourth time, subjected the minor child with some tests and the results are still outstanding despite the fact that the defendant as ordered by this Court on 3 March 2020 to provide the test to the plaintiff within 30 days of the order.”

- [11] The final test was performed by Centogene Laboratory in Germany at the instance of Dr Keshave, who suspected genetic conditions. Out of the Centogene test results, Dr Keshave was of the opinion that E had AGS which led to her brain damage. It is on the basis of this suspicion that this court must determine:

“Whether the child’s brain damage was caused by a hypoxic ischemic injury during the intrapartum period (the period from start of labour to birth) or whether the brain damage was caused by the genetic syndrome contended for by the defendant.”

Expert Evidence During Trial

- [12] During trial, only the expert witnesses testified. The medico-legal reports of experts were filed of record. They form part of the evidence. The report of Dr Mugerwa-Sekawabe, including all the facts and opinions contained therein, was accepted by the defendant. The report forms the basis of the admitted facts.
- [13] Dr Mugerwa-Sekawabe is a Specialist Obstetrician and Gynaecologist. Professor Lotz (neuroradiologist), Dr Gericke (Geneticist), Dr MacDonald (radiologist) and Dr Redfern (paediatric neurologist) all testified on behalf of the plaintiff.
- [14] Dr Zikalala (radiologist) and Dr Keshave (paediatric neurologist) testified on behalf of the defendant.
- [15] The report of Dr Mugerwa-Sekawabe is uncontested. Dr Mugerwa-Sekawabe provides an analysis of the assessment interview conducted with the plaintiff on 11 February 2019, a review of available documents, literature and gave his conclusions and opinions. His testimony on facts is largely based on the interviews with the plaintiff. Dr Mugerwa-Sekawabe’s report indicated that he had received the identity copy of the plaintiff, birth certificate of E, hospital notes, the medico-legal report by Prof Lotz and medico-legal report by Dr Redfern. Pursuant to the interview, he established that the plaintiff started with her antenatal visits at the Ntshale Clinic, Mthatha from when she was 5 months pregnant. The plaintiff made at least four visits to the clinic and during that period, no problems were identified.
- [16] The plaintiff had no medical or surgical problems prior to her falling pregnant. She was a primigravida. She started experiencing labour pains on 15 December 2007, during the night at approximately 23h00. On 16 December 2007, she presented to Vidgesville Clinic at 07h00, where she was examined. The examination included vaginal examinations, fetal heart rate and blood pressure. She was not advised on how far dilated the cervix was. There were no further examinations undertaken for the day. Her water broke at 18h00 and a toilet like liquid was coming out of her vagina. She was transferred to Mthatha General Hospital.
- [17] The plaintiff was taken to Mthatha General Hospital by ambulance and arrived at the hospital at 20h00. At the Mthatha General Hospital, she was assessed and advised that her womb was still closed. During this time, the labour pains were getting progressively stronger. She cannot confirm whether an FHR test was done on admission. The plaintiff was

admitted to the ward. Soon thereafter, she was left on her own. Approximately at 04h00, she felt something pushing in her vagina. The nurses were called to assist her. A student nurse assisted her with the delivery. The baby was then born. The plaintiff was thereafter put on oxygen via a face mask.

[18] E did not cry at birth. The nurse who was assisting the plaintiff showed the plaintiff the baby and pointed out to her a swelling on the baby's head. The baby was then rushed to an incubator as she started having seizures. Pursuant thereto, the baby was then transferred to Nelson Mandela Academic Hospital. The baby was nursed in an incubator, had an intravenous drip, oxygen was administered via nasal prongs and a nasogastric tube for feeding. The baby was discharged from the hospital on 27 December 2007. She has been on phenobarbitone treatment to suppress seizures. In spite of this, the seizures are still recurring.

[19] Dr Mugerwa-Sekawabe complains strongly about the unavailability of the relevant records pertaining to care during the antenatal period, as well as management during labour. The doctor emphasized the importance of these records for purposes of analysis. He then gave an analysis based on the available information and interviews of the plaintiff.

[20] The conclusion by Dr Mugerwa-Sekawabe in relation to the antenatal period is that there were no complications or untoward factors which could be associated with her condition. In relation to the labour, Dr Mugerwa-Sekawabe again pointed out the importance of the Maternity Case Records, which include the PARTOGRAM. These records are not available. The Maternity Care Record is the principal record of antenatal history, intrapartum history as well as the birth and immediate postpartum records. The PARTOGRAM relates to all findings of maternal and fetal conditions and progress in labour. All those records were not made available.

[21] Dr Mugerwa-Sekawabe highlighted the consequences of the missing clinical records and those consequences, according to Dr Mugerwa-Sekawabe, are that:

"21.1 Information on the important parameters of maternal and fetal

conditions as well as the progress of labour is not available. It is therefore not possible to ascertain what pertained during the intrapartum period;

21.2 Information on medications and/or medical interventions which were provided and/or instituted, is lacking;

21.3 Information on whether there were any obstetric and/or neonatal complications and what measures (if any) were instituted thereof is not available; and

21.4 The withholding of contemporaneous clinical records could be a deliberate measure to shield substandard care because those records could have brought such substandard care to light."

- [22] The opinion expressed by Dr Mugerwa-Sekawabe is that the transfer of the plaintiff to Mthatha General Hospital was necessitated as a result of the meconium staining of the liquor (MSL). Dr Mugerwa-Sekawabe was critical about the fact that the plaintiff was not referred for MSL and FHR tests on admission until the baby was born after 04h00 on 17 December 2007.
- [23] Dr Mugerwa-Sekawabe is of the opinion that the baby suffered from aspiration of MSL and was born with Apgar scores that were probably below normal. This conclusion is drawn by Dr Mugerwa-Sekawabe from the fact that the child did not cry at birth and it was recorded in the Road to Health Chart under “Problems during pregnancy/birth/neonatology” – “meconium aspiration, low Apgar”.
- [24] According to Dr Mugerwa-Sekawabe, the accepted standards addressed the problem of MSL and what monitoring of the FHR ought to be. In this regard, Dr Mugerwa-Sekawabe referred to the standards, which I quote below:
- “[T]hin meconium staining requires no special management. Thick [MSL] is associated with an increased risk of fetal distress. Transfer from community health centre to hospital unless delivery is imminent, monitor the fetus with a [CTG] if available, [and] when the head extends at delivery, thoroughly suck the infant’s mouth and then nose before delivering the trunk.”*
- [25] Dr Mugerwa-Sekawabe submitted that the management of the plaintiff’s labour, both at the clinic and at the hospital, were substandard, because:
- “1. MSL is an indication for CTG monitoring. Even in the absence of a CTG, the FHR ought to have been checked with a hand-held Doppler instrument every 30 minutes, before, during and after contractions.*
2. The baby aspirated meconium during the delivery. It would appear that the baby’s airways were not cleared of MSL as provided in the protocols.”
- [26] According to Dr Mugerwa-Sekawabe, contractions during labour lead to a reduction in oxygen supply to the placenta and in turn to the fetus. In relation to the uterus, the reduced pressure on the uterine arteries results in an improvement in the blood supply to the placenta, and a restoration of oxygenation to the fetus. In view of the increased risk of hypoxia and fetal distress in a setting of MSL, as was the case in this matter, the lack of monitoring of the FHR probably resulted in abnormalities of the FHR which accompany fetal acidosis going undetected. This resulted in E being born with low Apgar scores – one of the signs of a hypoxic ischaemic insult.
- [27] The conclusions of Dr Mugerwa-Sekawabe, pursuant to his analysis of the facts and literature, were that:
- “1. In all probability pointers to intrapartum hypoxia went unnoticed, leading the fetus to suffer from a hypoxic ischaemic injury. Prof Lotz is of the opinion that the fetus suffered from a global insult of a prolonged partial nature.*

2. There appears to be a causative link between the substandard management of labour and the hypoxic ischaemic brain insult suffered by E[....] L[....].”

[28] Dr Lotz, a Neuroradiologist, also testified in support of the plaintiff’s case. He testified in respect of the MRI scan of E’s brain and also testified about the CT scan which he had analysed with Dr MacDonald. He concluded that the neuroradiologic features of AGS and TS are not present on E’s MRI and CT scans. According to him, the salient points, in this regard, are summarised as follows:

“1. Intracranial calcification: The characteristic feature of AGS is intracranial calcification, which is present in 100% of cases. E[....]’s CT showed no intracranial calcifications.

2. White matter changes: The white matter changes of AGS occur predominantly in the lobar regions, with relative sparing of the periventricular area, corpus callosum and optic radiations. Contrary to this, the white matter affected in E[....]’s MRI is almost the exact opposite of this pattern.
3. Cerebral atrophy: Cerebral atrophy is present in the vast majority of AGS cases, in contrast, no cerebral atrophy was present in E[....]’s MRI.
4. The MRI and CT scan features essentially exclude a diagnosis of AGS.”

[29] Dr Lotz confirmed that WES failed to detect an alternative explanation for E’s brain injury. Also, on this basis, he agreed with other experts that the most likely cause of E’s brain injury and neurodisability is a hypoxic ischaemic injury sustained during the intrapartum period. Dr Lotz came to the conclusion that the case of E indicates that there was a hypoxic ischemic insult to a term brain which caused the injuries seen in E. He further explained that when there is a partial prolonged insult, it means that the blood flow to the brain is interrupted at times by way of both hypoxia (not enough oxygen in the blood) and ischemia (not enough pressure in the flow of blood) to the brain.

[30] Dr Lotz dealt in his evidence with AGS extensively. He dealt with calcification and showed the slides where there is calcification and not.

[31] Dr Gericke also testified in support of the plaintiff’s case. He is the only qualified genetic expert that has been called in the case. I must indicate though that Dr Keshave, a paediatric neurologist, had informed this court that he can testify about genetics as well. That aspect is dealt with later.

[32] Dr Gericke is a specialist clinical geneticist. Broadly speaking, his evidence was not challenged on material aspects. His testimony related to the issue of genetics. In his testimony, Dr Gericke excluded AGS presence on E. He directly responded to the Centogene report, an aspect to which I deal with later. He firstly indicated that the Centogene report indicates that E has a genetic result of no functional significance, for it is of unknown significance and as such, it is not diagnostic of AGS. According to Dr Gericke, the sequencing results from the testing performed by Centogene indicate two ‘variants of uncertain

significance' (VOUS or VUS) relating to non-disease causing, incomplete changes in genes involved with Aicardi Goutieres syndrome and tuberous sclerosis II complex are just that, they are of uncertain significance. VOUS are graded according to whether there is much available information (Classes 2 and 4), but the described variants in this instance are associated with 'scarce information'. Of the VOUS, Class 3 is the most numerous one, comprising about 40% of all VOUS variants discovered.

- [33] E's family pedigree, medical history, neurodevelopmental course, MRI findings and genetic testing do not demonstrate any of the known clinical features or TREX1 or TSC2 mutations that are associated with Aicardi Goutieres Syndrome of Tuberous Sclerosis respectively.
- [34] In summary, the presented genetic sequencing results are not informative and cannot be used in any way to even remotely suggest that they relate to the patient's adverse neurological presentation.
- [35] Dr MacDonald, an Adjunct Professor of Radiology, also testified for the plaintiff. He testified on the MRI brain and CT brain scans. According to Dr MacDonald, the CT and MRI brain scans demonstrate that there are no calcifications, effectively excluding AGS as a possible diagnosis. According to him, the white matter damage seen in E is in a typical watershed distribution, specifically sparing the temporal lobes and there is a severe cortical thinning in the watershed regions.
- [36] In his testimony, Dr MacDonald explained that AGS is rare and thereafter outlined what is expected in a neuroimaging diagnosis of AGS. He draws the court's attention to the following aspects:

"1. Brain calcifications

1.1 The study by Ugetti was 100% of 36 patients and La Piana of 121 patients but 97.5% of 112 patients where CT scans were available. CT is more sensitive for calcification than MRI and is the gold standard for demonstrating brain calcification.

1.2 The result of this is in 98% of the cases where there is AGS calcification will be present. This is a percentage which would mean that for this theory of the defendant to work, the child must by chance fall within the 2% that does not show calcification.

2. Leukoencephalopathy (abnormal white matter)

2.1 Ugetti showed 100% of 36 patients and La Piana 90% of 121 patients.

2.2 The cortex is preserved. In a patient with AGS, the cortical thickness is preserved (as opposed to what we see in E[....], where the cortex is severely thin in the watershed regions)."

- [37] Dr MacDonald is of the opinion that for the defendant's proposition to work, E must, first of all, fall within the 2% with no calcification and then further fall into the miniscule percentage with no leukoencephalopathy. In addition, thereto, E would be one of the first cases (if not the first) where the cortex is not preserved. The CT and MRI for E therefore demonstrate none of the features expected for AGS.
- [38] Dr Redfern, a paediatric neurologist, also testified on behalf of the plaintiff. In his testimony, he indicated that E does not display the necessary clinical features to suggest a diagnosis of TREX1 related AGS. E's neuroimaging is completely not compatible with a diagnosis of AGS. The most probable cause of E's disability is hypoxic ischaemic brain injury sustained in the perinatal period.
- [39] That was the plaintiff's case.
- [40] The defendant called two expert witnesses, Dr Keshave and Dr Zikalala.
- [41] Dr Keshave, in his testimony for the defendant, informed this court that he is a paediatric neurologist.
- [42] Dr Keshave testified that he assessed E on 28 February 2019. He had also considered documents submitted to him, such as the Road to Health Card, medico-legal report by Dr Zikalala, a radiologist and the Ampath Laboratory genetic results. In his neurological or developmental assessment, Dr Keshave prepared his reports. I quote below from his report dated 31 October 2019, and as amended on 25 February 2020:

"16. Summary of Neurological/Developmental Assessment

E[...] is a 12 year old female with Microcephalic Spastic Quadriplegia with features of Autistic Spectrum Disorder complicated by Global Developmental Delay, Intellectual Disability, Symptomatic Epilepsy and a pseudo bulbar palsy.

The addition of the radiologist report – indicates that there is cystic encephalomalacia. This would support the presence of neonatal encephalopathy at the time of birth – however the aetiology of neonatal encephalopathy is vast and includes the following conditions:

- 1) Metabolic Conditions
- 2) Infectious
- 3) Toxin and Drugs
- 4) Perinatal Stroke Syndrome
- 5) Congenital Anomalies
- 6) Vasculopathies
- 7) Genetic Syndromes

8) Hypoxic Ischemic Encephalopathy

Based on the RTHC the presence of Meconium Aspiration Syndrome seems the most likely cause to have resulted in the low apgars (actual score not documented) and this would have caused the Neonatal Encephalopathy.

I also strongly feel that there is an underlying genetic condition – in view of the strong Autistic Spectrum Disorder features that are showed by E[...] – one would need to exclude an underlying genetic condition. As the severity of the MRI brain does not match the clinical picture noted in E[...] at age 12 years of age.

Based on the genetic test, CGH Array – done at Ampath Laboratories on the 18th March 2019 indicated that there was no underlying abnormalities, this would make an underlying genetic cause for E[...]’s condition less likely. Although the genes have returned negative for the common South African genetic mutations for children with ASD – this does not totally exclude an underlying genetic disorder, as mentioned in the final comment on the genetic results from Ampath Laboratory.

However in view of the lack of medical records it is still very difficult to predict what would have caused E[...]’s current clinical condition, as mentioned before the presence of Neonatal Encephalopathy does not singly handily indicate Birth Asphyxia other conditions that still need to be excluded include:

16.1 Metabolic Conditions

16.2 Infectious

16.3 Toxin and Drugs

16.4 Perinatal Stroke Syndrome

16.5 Vasculopathies”

[43] Dr Keshave made a recommendation for a neurometabolic screening to be done to exclude possible aetiology, following which the most likely diagnosis would then be birth asphyxia. He complained as well about the lack of medical records, which records, in his opinion, would have assisted with determining the underlying cause. Notwithstanding the production of various laboratory reports, Dr Keshave had a suspicion about the existence of some other medical conditions in “E”, and he called for further metabolic and genetic tests. One test that led Dr Keshave to amend his first report, was the Centogene test report.

[44] The Centogene report, contained the following observations under the heading “Interpretation”:

“A heterozygous variant of uncertain significance was identified in the TREX1 gene. The genetic diagnosis of autosomal dominant Aicardi-Goutieres syndrome 1 is possible.

A heterozygous variant of uncertain significance was identified in the TSC2 gene. The genetic diagnosis of autosomal dominant Tuberous sclerosis type 2 is possible. However, further analysis is necessary.”

[45] The Centogene report, however, proposed further testing and other investigations for the confirmation of their suspicions. These included:

“45.1 Clinical correlation.

45.2 Parental carrier testing to establish whether the detected TREX1 and TSC2 variants are inherited or de novo.

45.3 Carrier testing for all informative family members to establish whether the detected variants are associated with the disorder or not. Basic clinic information and relationship for each analysed family member is needed for a comprehensive evaluation of the data.”

[46] Based on the Centogene report, Dr Keshave requested the defendant’s radiologist, Dr Zikalala, to review the earlier findings. In essence, Dr Keshave maintains that there is a suspicion that “E” suffers from Aicardi-Goutieres (AGS) and that, according to Dr Keshave on the interpretation of the Centogene report, the condition is genetic and the AGS caused the insult to the brain of “E”. The family history had shown no genetic conditions.

[47] The defendant further called Dr Zikalala, a radiologist, to testify with regards to the MRI and CT scans. Dr Zikalala had prepared two expert reports. The first report is dated 4 February 2019 and the amended report is dated 27 November 2020. In her testimony, Dr Zikalala dealt with the two reports. In respect of the first report, she made the comment below:

“Cystic encephalomalacia in the anterior and posterior watershed zones. In an appropriate context, this is seen as the sequelae of partial hypoxia of prolonged duration in term infants. These findings need further correlation with clinical history and findings.”

[48] Almost a year later on 27 November 2020, after receipt of Dr Keshave’s amended report which suggests AGS, Dr Zikalala amended her initial comments to read:

“Cystic encephalomalacia in the anterior and posterior watershed zones. Differentials include metabolic conditions, infections and hypoxic ischaemic encephalopathy. However, correlation with clinical history, pediatric assessment, biochemistry and genetic testing confirmed Aicardi-Goutieres syndrome, making the other causes less likely.

The findings in our case are similar to documented cases of Aicardi-Goutieres syndrome which include:

- Abnormal white matter signal intensity with a diffuse frontotemporal anteroposterior gradient, and periventricular distribution
- Cystic areas in the temporal and/or frontal lobes
- Cerebral atrophy with dilatation of ventricles

- Deep white matter cysts
- Although calcifications, mainly in the basal ganglia, lobar white matter, and dentate nuclei are not a dominant feature in our case,

some case reports have reported absence or resolution of calcifications with time.”

- [49] Dr Zikalala, in her evidence, testified that the amendment was necessitated by a report because there were a few changes in the scan that resembles some of the things that are described in the literature of AGS. She indicated that she was informed by the team and the information brought by a paediatric neurologist, who carried out the test on the child and she disputed allegations which suggested hearsay about the AGS. Dr Zikalala, in her evidence, supported Dr Keshave that the conditions, as depicted on the MRI scan did not correlate with the clinical presentation of the child.
- [50] Dr Zikalala, in her evidence, indicated that she was informed that there was genetic testing which suggested AGS or similar cases in the literature of AGS. The entire second report of Dr Zikalala, according to her, is based on the proposition that AGS had been positively diagnosed and therefore, she had to review the first report. Indeed, the first report was reviewed and the second report suggests the presence of AGS.

- [51] That was the case of the defendant.

The Applicable Law

- [52] To obtain a judgment holding the defendant liable to pay delictual damages, the court in Minister of Safety & Security v Van Duivenboden[1] state that the plaintiff must prove, on a balance of probabilities, that the act(s) or omission(s) of the defendant is wrongful and negligent, and have caused loss. The approach in our law to the plaintiff’s claim is not controversial. It is trite that in order to succeed in her delictual claim for damages, the plaintiff must establish that the wrongful and negligent conduct of the province’s nursing and medical staff, acting within the course and scope of their employment, caused her harm. [2]

- [53] In Kruger v Coetzee[3] it was held:-

“For the purposes of liability culpa arises if –

- (a) a diligens paterfamilias in the position of the defendant (or his employees) –
 - (i) would foresee the reasonable possibility of his (their) conduct injuring another in his person or property and causing him patrimonial loss; and
 - (ii) would take reasonable steps to guard against such occurrence; and
- (b) the defendant (or his employees) failed to take such steps.”

- [54] In Naude NO v Transvaal Boot and Shoe Manufacturing Co[4] it was held –

”Although the onus of proving negligence is on the plaintiff, the plaintiff does not have to adduce positive evidence to disprove every theoretical explanation which is exclusively within the knowledge of the defendant, however unlikely, that might be devised to explain (his paraplegia) in a way which would absolve the defendant and his employees of negligence.”

[55] In *Mitchell v Dixon*[5] it was held –

“A medical practitioner is not expected to bring to bear upon the case entrusted to him the highest possible degree of professional skill, but he is bound to employ reasonable skill and care; and he is liable for the consequences if he does not.”

[56] In *Monteoli v Woolworths (Pty) Ltd*[6] the court confirmed that the onus, nevertheless, remains with the plaintiff. The defendant has an evidential burden to show what steps were taken to comply with the standards to be expected.

[57] In *Minister of Safety & Security & Another v Carmichele*[7] where the court confirmed that causation has two elements –

“1. The factual issue to be established on a balance of probabilities by the plaintiff by using the “but for” test would involve the mental elimination of the wrongful conduct in the posing of the question as to whether upon such hypothesis, the plaintiff’s loss would have ensued or not;

2. The legal causation, namely whether the wrongful act is linked sufficiently closely or directly to the loss for legal liability to ensue or whether, as it is said, the loss is too remote. This is a juridical problem and considerations of policy may play a part in the solution thereof.”

[58] In *Caswell v Powell Duffryn Associates Collieries*[8] Lord Wright remarked –

“Inference must be carefully distinguished from conjecture or speculation. There can be no inference unless there are objective facts from which to infer the other facts from which it is sought to establish. In some cases, the other facts can be inferred with as much practical certainty as if they had been actually observed. In other cases the inference does not go beyond reasonable probability. But if there are no positive proved facts from which the inference can be made, the method of inference fails and what is left is mere speculation or conjecture.”

[59] The expert witnesses who had testified are experienced. As a result of the expert reports and testimonies, the issues were narrowed down to the question of what the factual cause of E’s condition was. The starting point would be to evaluate and resolve the conflict in the testimony of the experts for the plaintiff and for the defendant. In *J Afrikaner on behalf of DMA v The MEC for Health*.[9]

“The opinion of a witness is generally inadmissible. ‘In the law of evidence, ‘opinion’ means any inference from observed facts, and the law on the subject derived from the general rule that witnesses must speak only to that which was directly observed by them.’ Opinion is admissible if it is relevant. Relevance is in turn determined by the issues in the matter. If the opinion can assist the court in determining an issue, it has probative value, otherwise it is superfluous. Expert opinion evidence is

received when the issues require special skill and knowledge to draw the right inferences from the facts stated by the witnesses.

[60] The Full Bench of the Eastern Cape in *J Afrikander* discuss several types of conflicts in expert evidence that may present itself in any given case.[10] The first is a conflict with regard to the assumed facts. Expert opinion must have a factual basis. The facts upon which an expert

opinion is based must be proved by admissible evidence. A second conflict in the expert opinion may lie in the analysis of the

established facts and the inferences drawn therefrom by the opposing witnesses. The cogency of the expert opinion depends on its

consistency with proven facts and on the reasoning by which the conclusion is reached. The source for the evaluation of this evidence

for its cogency and reliability are (i) the reasons that have been provided by the expert for the position adopted by him/her; (ii) whether that reasoning has a logical basis when measured against the established facts; and (iii) the probabilities raised on the facts of the matter. It means that the opinion must be logical in its own context, that is, it must accord with, and be consistent with all the established facts, and must not postulate facts which have not been proved.

[61] The inferences drawn from the facts must be sound. The logic of the opinion must be consistent, and the reasoning adopted in arriving at the conclusion in question must accord with what the accepted standards of methodology are in the relevant discipline. The reasoning will be illogical or irrational and consequently unreliable, if (i) it is based on a misinterpretation of the facts; (ii) it is speculative, or internally contradictory or inconsistent to be unreliable; (iii) if the opinion is based on a standard of conduct that is higher or lower than what has been found to be the acceptable standard; (iv) if the methodology employed by the expert witness is flawed. What flows from this is that the mere fact that an expert opinion is unchallenged, does not necessarily mean that it must be accepted. However, if that evidence is based on sound grounds and is supported by the facts, there exists no reason not to accept it.

[62] Other considerations relevant in this context are (i) the qualifications and the experience of the expert witnesses with regard to the issue he or she is asked to express an opinion on; (ii) support by authoritative, peer-reviewed literature; (iii) the measure of equivocality with which the opinion is expressed; (iv) the quality of the investigation done by the expert; (v) and the presence or absence of impartiality or a lack of objectivity. What is ultimately required is a critical evaluation of the reasoning on which the opinion is based, rather than considerations of credibility. Should it not be possible to resolve a conflict in the expert opinion presented to the court in this manner, that is, when the two opposing opinions are both found to be sound and reasonable, the position of the overall burden of proof will inevitably determine which party must fail. It is worth emphasising that the onus as a

determining factor “can only arise if the tribunal finds the evidence pro and con so evenly balanced that it can come to no such conclusion. Then the onus will determine the matter. But if the tribunal, after hearing and weighing the evidence, comes to a determinate conclusion, the onus has nothing to do with it, and need not be further considered.”

- [63] The third type of conflict, which may arise in expert evidence is that of competing theories of a purely scientific nature. The choice between two conflicting theories is informed primarily by the extent to which the theory is regarded as being established and has gained general acceptance within the specific scientific community in the particular discipline to which it belongs. Whether or not a theory has been sufficiently established must be measured against considerations such as whether it can, and has been tested; whether it is the product of reliable principles and methods that have been reliably applied to the facts of the case; and whether it has been subjected to peer review and publication.
- [64] Fourthly, a conflict may also arise in the context of what the accepted standard of conduct of a medical professional is in certain circumstances. Typically, medical negligence cases deal with the situation where an injury is alleged to be in complete discord with the recognised therapeutic objective and techniques of the operation or treatment involved. Expert opinion, in this context, is aimed at determining whether the conduct of a professional person in a particular field accords with what is regarded as a sound practice in that field. Again, the method adopted is to evaluate opinion evidence with the view of establishing the extent to which the opinions advanced are founded on logical reasoning.
- [65] What is evident from the foregoing is that the evaluation of expert opinion in determining its probative value and the considerations relevant thereto, are determined by the nature of the conflict in the opinion, and the context provided by all the evidence and the issues which the court is asked to determine. In general, it is important to bear in mind that it is ultimately the task of the court to determine the probative value of expert evidence placed before it and to make its own finding with regards to the issues raised. Faced with a conflict in the expert testimony of the opposing parties, the court

is required to justify its preference for one opinion over another by a careful and critical evaluation thereof. Further, the primary

function of an expert witness is to guide the court to a correct decision on questions, which fall within that expert's field.

To that extent, the expert witness has a duty to provide the court with abstract or general knowledge concerning his or her discipline,

and the criteria necessary to enable the court to form its own independent judgment by the application of the criteria to the facts

proved in evidence. Accordingly, the mere “pitting of one hypothesis against another does not constitute the discharge of the functions of an expert.” Finally, it is not the function of the court to develop its own theory or thesis and to introduce on its own accord evidence that is otherwise founded on special knowledge and skill. Ex hypothesi, such evidence is outside the learning of the court. The function of the court is restricted to deciding a matter on the evidence, or accepting or rejecting the proffered expert evidence. [11]

[66] There is a general obligation placed upon the parties on cross examination of witnesses, including experts, to put the parties’ case to the witness being cross examined. The reason for this is to allow the witness to deal with the evidence where he differs with such evidence. In this regard, *Small v Smith*[12] and *President of the Republic of South Africa v SARU*[13] support the position taken by this court. Expert witnesses should provide independent assistance to the court by way of objective,

unbiased opinions. An expert witness is not required to assume the role of a legal practitioner or that of the court. An expert witness must state facts or assumption upon which his or her opinion is based. The expert must not omit to consider the material facts that should detract from his concluded opinion. It is not expected of the court to simply accept the opinions of experts. The expert evidence must be logical and his or her conclusions must be reached with knowledge of all the facts.

[67] In *Schneider NO and Others v AA and Another*[14] Davis J discusses the duties of an expert with reference to some authorities, whereafter he makes the statement, with which I agree:

“In short, an expert comes to court to give the court the benefit of his or her expertise. Agreed, an expert is called by a particular party, presumably because the conclusion of the expert, using his or her expertise, is in favour of the line of argument of the particular party. But that does not absolve the expert from providing the court with as objective and unbiased an opinion, based on his or her expertise, as possible. An expert is not a hired gun who dispenses his or her expertise for the purposes of a particular case. An expert does not assume the role of an advocate, nor gives evidence which goes beyond the logic which is dictated by the scientific knowledge which that expert claims to possess.”

[68] In *Michael and Another v Linksfield Park Clinic (Pty) Ltd & Another*[15] the court had the following to say when considering expert evidence:

“This essential difference between the scientific and the judicial measure of proof was aptly highlighted by the House of Lords in the Scottish case of Dingley v The Chief Constable, Strathclyde Police 200 SC (HL) 77 and the warning given at 89D-E that:

‘(O)ne cannot entirely discount the risk that by immersing himself in every detail and by looking deeply into the minds of the experts, a Judge may be seduced into a position where he applies to the expert evidence the standards which the expert himself will apply to the question whether a particular thesis has been proved or disproved – instead of assessing, as a Judge must do, where the balance of probabilities lies on a review of the whole of the evidence.’

Analysis and Findings

[69] I am obliged to assess and evaluate the entire evidence. However, I am troubled by the fact that the blood test results from Ampath Laboratories and other tests were submitted with no corroborating evidence. The Centogene report, which in my view, is central to the defendant's case, was submitted with no corroborating evidence. The Centogene report called for further tests and information. This was not entirely done. The Centogene report had called for further investigation in order to confirm their findings. The family history had shown no genetic conditions. This court has not been told why it should prefer the Centogene laboratory report over other reports. For that reason I will consider these reports in light of all the evidence and to the extent that they provide admissible evidence. I raise the issue of the Centogene report and the report from Ampath Laboratories because they give contradictory results. Centogene Laboratory suggests the genetic diagnosis of autosomal dominant Aicardi-Goutieres syndrome 1 is possible and that the genetic diagnosis of autosomal dominant Tuberous sclerosis type 2 is possible. The other reports contain no such findings. These contradictions were not explained nor was evidence from laboratories led during the trial. The plaintiff's experts had excluded the presence of AGS.

[70] There is no controversy about the report of Dr Mugerwa-Sekawabe. The entire report has been accepted by the defendant. In such circumstances, I therefore accept that the findings and opinions of Dr Mugerwa-Sekawabe are common cause. Briefly, Dr Mugerwa-Sekawabe mentioned some critical findings which I do repeat below.

[71] Dr Mugerwa-Sekawabe finds that the management of the labour, both at the clinic and hospital, was of a substandard nature because -

71.1 MSL is an indication for CTG monitoring (this is monitoring by a cardiotocograph machine). Even in the absence of a CTG, the FHR ought to have been checked with handheld Doppler instrument every 30 minutes before, during and after contractions. This did not happen.

71.2 The baby aspirated meconium during the delivery. It would appear that the baby's airways were not cleared of MSL as provided in the protocols.

71.3 The non-monitoring of the FHR probably resulted in abnormalities of the FHR which accompany foetal acidosis going undetected. This resulted in the newborn being born with low APGAR scores – one of the signs of hypoxic ischemic insult.

71.4 The intrapartum management of the unit was flawed.

71.5 If the monitoring had been proper "it would have been possible to detect those unfavourable factors which have necessitated interventions. The failure to undertake these observations, in line with accepted standards – and also not instituting measures of clearing the baby's airways of the meconium fluid at delivery so as to mitigate against meconium aspiration – amounted not only to substandard care but was also a breach of the duty of care."

71.6 In all probability, Dr Mugerwa-Sekawabe concluded that pointers to intrapartum hypoxia went unnoticed, leading the foetus to suffer from a hypoxic ischemic injury.

[72] The defendant did not challenge the report of Dr Mugerwa-Sekawabe and instead, his findings were admitted in the pre-trial minutes. I cannot fault the report by Dr Mugerwa-Sekawabe. Accordingly, I accept the evidence of Dr Mugerwa-Sekawabe.

[73] Dr Mugerwa-Sekawabe relied upon the direct evidence of the plaintiff. The medical staff had a duty to record the treatment that was accorded to the plaintiff. The defendant's employees were obliged to keep punctilious clinic and hospital notes pertaining to the plaintiff's treatment. I agree with the submissions of Mr Van der Walt, counsel for the plaintiff in this regard. The missing hospital records remains questionable. In my view, the defendant's employees had breached their legal duty to maintain official hospital records. In such circumstances, the plaintiff's evidence as summarised in the report of Dr Mugerwa-Sekawabe should prevail.

[74] In *Lungile Ntsele v MEC for Health, Gauteng Provincial Government*[16] it is said:

"(46) Logic and common sense dictates that the plaintiff's labour and A's subsequent birth endured for a longer period of time than the few minutes suggested by the plaintiff under cross-examination. It is unfair and unjust for the defendant's counsel without any cogent evidence from the defendant's employees regarding the treatment accorded to the plaintiff or any reasonable explanation tendered by the defendant's employees regarding the disappearance of the plaintiff's clinic and hospital records, to expect the plaintiff to be precise and specific about the treatment accorded her at the clinic and hospital whilst under anaesthesia.

(115) There is a legal duty on the nurses at the clinic, the doctor and nurses at the hospital to record the treatment accorded to the plaintiff and Ayanda. The defendant's employees were obliged to and must have made and kept punctilious clinic and hospital notes pertaining to the plaintiff's treatment.

(116) The clinic and hospital notes are missing from the plaintiff's

and A's files. There is a duty on the clinic and hospital record custodian staff in terms of sections 13 and 17 of the National Health Act No 61 of 2003 to safeguard the plaintiff's and A's clinic and hospital records."

[75] Prof Lotz analysed the damage to the brain of E and made use of slides in his presentation of evidence. Prof Lotz concluded that there was a hypoxic ischemic insult. The evidence of Prof Lotz is consistent with the report prepared by Dr Mugerwa-Sekawabe which had pointed to a hypoxic ischemic injury. Prof Lotz evidence was not meaningfully challenged under cross-examination. I was satisfied with the evidence of Prof Lotz. He gave logical opinions and he was clear in his evidence. I do accept his evidence, more so that it is in line with the report by Dr Mugerwa-Sekawabe.

- [76] Dr Gericke was the only fully qualified genetic expert. The defendant relied on the evidence of Dr Keshave. The evidence of Dr Gericke was straightforward to the effect that E does not have AGS. In supporting his conclusion, Dr Gericke pointed out that the variant identified in the Centogene report means that E has a genetic result of no functional significance at all. That is because it is of unknown significance and it is not diagnostic of AGS. The defendant's counsel, Mr Maseti, could not challenge in a meaningful way, these conclusions. Accordingly, I accept the evidence of Dr Gericke. It was logical and supported with convincing analysis of objective evidence.
- [77] Dr MacDonald was tasked to perform a CT scan and studied the MRI scan in order to specifically make conclusions with reference to neuroimaging of AGS. In his explanation, he informed this court that AGS is rare and he indicated what is expected in a neuroimaging diagnosis of AGS. According to Dr MacDonald, the CT and MRI scans demonstrate none of these features expected for AGS. There are no calcifications, which effectively exclude AGS as a possible diagnosis. The white matter damage seen in the brain of E is the typical watershed distribution, specifically sparing the temporal lobes and there is severe cortical thinning in the watershed region. I accept Dr MacDonald's evidence. It is logical and an objective analysis of the facts and available information.
- [78] Dr Redfern also came to the conclusion that there was no AGS. In reaching his conclusion, he had researched and considered the clinical and MRI characteristics of AGS. I have no hesitation in accepting his evidence and opinions. I was satisfied with his analysis and his conclusions are logical. I also do consider the fact that there is an agreement amongst all the experts that AGS is a rare genetic disorder.
- [79] I have difficulties in accepting Dr Keshave's evidence. I highlight the following shortcomings in his evidence:
- 79.1 Dr Keshave's evidence was speculative and incapable of throwing any doubt on the otherwise acceptable opinion of the plaintiff's expert evidence. The opinion evidence of plaintiff's expert was based on sound grounds and supported by the basic facts.
- 79.2 I cannot ignore the fact that Dr Keshave had called for several tests to be conducted for as long as the first tests were not giving his desired outcomes. Dr Keshave had sought to qualify himself as a geneticist. Dr Keshave had introduced new material facts which were not contained in his expert opinion. There was no acceptable explanation given by him. In his expert report, Dr Keshave never mentioned that he had a physical possession of the MRI scans. During his oral testimony, he created an impression that he had possession of the MRI scans. During cross examination by Mr van der Walt, counsel for the plaintiff, Dr Keshave was evasive in his answers. I was not impressed with the explanations given by Dr Keshave for introduction of the new facts.

79.3 I cannot find an explanation why Ampath Laboratories results should not be relied upon, according to Dr Keshave. He sought to testify about genetic analysis and thereby discount the expert in the field, Dr Gericke. I find this unacceptable. An objective expert would give concessions where appropriately needed in respect of experts in the field. Dr Keshave was less convincing in doing so.

79.4 Whilst I do appreciate the concession given by Dr Gericke that persons like Dr Keshave and Dr Redfern, may have some knowledge of genetics in their field, genetics is a specialist area. Dr Gericke is a specialist in genetics and I therefore prefer his opinions over those of Dr Keshave.

79.5 For these reasons, I find that Dr Keshave's evidence had shortcomings and it would not be safe for this court to rely thereupon.

[80] From a reading of Dr Keshave's evidence and what he agreed to in the joint minute with Dr Redfern, it is evident that he was not able to raise AGS as a cause of the injury to E, any higher than it being a mere possibility. The suggestion that the prolonged partial intrapartum asphyxia resulting in hypoxic ischaemic encephalopathy after birth is consistent with the uncontroverted evidence by Dr Mugerwa-Sekawabe. Dr Keshave cannot undo this conclusion. The demand for several tests to be conducted on child E, seems to suggest that Dr Keshave had a resolve to find anything that will explain away from the hypoxic ischaemic encephalopathy injury on E. At least, for the court, some measure of certainty on what the cause of the injury suffered by E, was expected from Dr Keshave. Dr Keshave failed dismally in this regard. I have my reservations about the Centogene report. No conclusive evidence about this report was led and that leaves me with many questions than answers. On the other hand, the plaintiff's experts presented logical opinions which is supported by facts.

[81] Dr Zikalala agreed with the plaintiff's expert witnesses that there is no calcification on the CT scan on child E and she confirms that the prolonged partial hypoxic ischaemic injury watershed involvement is the hallmark of prolonged partial hypoxic ischaemic injury. In her evidence, Dr Zikalala testified, I quote:

"Ms Zikalala: I cannot exclude AGS in this case. We look for MRI features, we did not find all of them admittedly. It is a genetic

test, a scientific test, I am not a geneticist, if the person ..[indistinct] is such then I cannot exclude it, unless the person

says something else and that is not my field of expertise. I will leave it with the ...[indistinct]."

[82] The above answer is less convincing when regard is had to the first report of Dr Zikalala when she records the following:

[83] The position and views of Dr Zikalala only changed after the report by Centogene and at the instance of Dr Keshave's intervention. This is clearer when regard is had to the report prepared on 4 April

2019. In the report, Dr Zikalala makes this statement under her findings:

“Cystic encephalomalacia in the anterior and posterior watershed zones. Differentials include metabolic conditions, infections and hypoxic ischaemic encephalopathy. However, correlation with clinical history, pediatric assessment, biochemistry and genetic testing confirmed Aicardi-Goutieres syndrome, making the other causes less likely.”

[84] During questioning by this court, Dr Zikalala made the following concession:

“Court: You do not have a case of AGS, but you cannot ignore the clinical findings of other expert and you believe to them so I must look onto those findings. I must be looking onto those but from your perspective as you sit here, you do not have a case. It is not your case that there is AGS here. That is my last clarity I want from you.”

Ms Zikalala: What I am saying is I do not have classical features of AGS therefore, I will look into the clinicians, the experts and the genetic information given by the geneticist.”

[85] I was not convinced by Dr Zikalala. The inconsistencies in her evidence have not been convincingly justified. The differences between her initial admissions and the subsequent report after the Centogene report, cannot be reconciled. She admits that she is not an expert in genetics. I have already accepted the evidence of Dr Gericke, which I do prefer, because of its logic and common sense based on the admitted evidence. For these reasons, I reject the evidence of Dr Zikalala.

Conclusion

[86] On the whole, the evidence, in particular the report prepared by Dr Mugerwa-Sekawabe, supports the expert opinion that the brain injury sustained by E and the disabilities that later followed, were the result of prolonged partial hypoxic ischaemia during labour as opposed to AGS. The injury is consistent with the conduct of the defendant’s medical staff and nurses, allowing a severely prolonged labour of the plaintiff to continue with no monitoring, exposing the fetus to a risk of hypoxic type brain injury. The plaintiff’s experts have, in my view, objectively evaluated the available facts, the limited medical records and the clinical findings logically and carefully. Both Prof Lotz and Dr MacDonald found that it was a hypoxic ischaemic insult of a partial prolonged nature. The defendant’s expert, Dr Zikalala, had initially agreed with these observations. Dr Keshave could not dispute those observations with certainty. He was non-committal and speculative in his analysis.

[87] I have been invited by the plaintiff’s counsel, Mr Van der Walt, to consider the conduct of Dr Keshave in this case. My attention was drawn to the fact that the behaviour of Dr Keshave failed to meet the basic expected standards from an expert. The suggestion is that he was not independent. Whilst I have reservations about Dr Keshave’s insistence on several tests and the subsequent interactions with Dr Zikalala, who thereafter changed her initial report, I do not believe that there has been some form of mala fides on the part of Dr Keshave. Accordingly, I decline to draw any adverse inference against Dr Keshave.

[88] I agree with the plaintiff's submissions that there had been inadequate monitoring during the birth process when there would have been danger signs such as a prolonged labour process and other signs of distress of the fetus. The child has been born in a compromised position with low APGAR scores. The MRI clearly shows a hypoxic ischaemic insult of a partial prolonged nature. The insult must have taken place during the intrapartum period when regard is given to the entire evidence and opinions of Drs Mugerwa-Sekawabe and Redfern. Dr Gericke, a qualified geneticist, has removed any possible clinical

signs of AGS. The admitted facts in the pre-trial minute, joint expert minutes and the common cause facts established the plaintiff's

case on a balance of probabilities.

[89] I find that the defendant had breached their contractual obligations of care and thereby caused E to suffer hypoxic ischaemic insult of a partial prolonged nature. The net result is that the defendant is liable to compensate the plaintiff.

[90] For these reasons, the plaintiff's case must succeed on the merits. The costs of this part of the trial should follow the results, and I have not been persuaded differently. The plaintiff is entitled to the costs of the experts that have been employed.

Order

[91] In the result, the following order is made:

1. The merits and quantum are hereby separated in terms of Rule 33(4).

1.5cm; margin-bottom: 1cm; line-height: 200%"> 2. The determination of quantum is postponed sine die.

3. The defendant is held liable for the plaintiff's agreed or proven damages arising from the cerebral palsy suffered by the minor child "E".

4. The defendant shall pay the plaintiff's costs relating to the issue of merits, which costs shall include the employment of two counsel, together with all reserved costs, if any, which costs shall include:

4.1 The travelling expenses, reservation and appearance fees, if any, together with the costs of the preparation of their reports and qualifying fees, if any, of the following expert witnesses:

4.1.1 Dr E Mugerwa-Sekawabe – Specialist Obstetrician & Gynaecologist;

4.1.2 Dr A Redfern – Senior Specialist/Paediatric Neurologist;

4.1.3 Professor J Lotz – Professor of Radiology / Neuroradiologist;

4.1.4 Dr G Gericke – Clinical Geneticist; and

4.1.5 Dr A MacDonald – Professor of Radiology.

4.2 The defendant shall pay interest on the aforesaid costs at the current prescribed legal rate of interest from date of allocator or agreement to date of payment thereof.

M NOTYESI

JUDGE OF THE HIGH COURT (ACTING)

Appearances:

Counsel for the plaintiff : Mr Van Der Walt SC

together with V Msiwa

Attorney for the plaintiff : Sakhela Incorporated

East London

Counsel for the respondent : A Maseti

Attorney for the defendant : Office of the State Attorney

[1] 2002 (6) SA 431 SCA at para [12]; LD obo AD v Member of the Executive Council responsible for the Department of Health [2021] JOL 49623 (ECM) at p2

[2] KX v Member of the Executive Council for Health, Western Cape [2021] JOL 51401 (WCC) at p3

[3] Kruger v Coetzee 1966 (2) SA 428 (A) at 430E

[4] 1938 AD 379 at 392(3)

[5] 1914 AD 519 at 525, see also [K...]X[...]supra at p3

[6] 2000 (4) SA 735 (W) at 127

[7] 2004 (3) SA 305 (SCA)

[8]

[1939] 3 All ER 722 (HL) at 733

[9] J Afrikander obo DMA v The MEC for Health, Eastern Cape – Full Bench – Case No: C A & R 8/2021 at p9 para 10, see also cases referred to in the judgment

[9] J Afrikander obo DMA v The MEC for Health, Eastern Cape – Full Bench – Case No: C A & R 8/2021 at p9 para

10, see also cases referred to in the judgment

[10] J Afrikander supra at p9 – p17

[11] See J Afrikander obo DMA supra at p17

[12] 1954 (3) SA 434 (SWA)

[13] 2000 (1) SA 1 (CC) at 36 [para 61 – 65]

[14] 2010 (5) SA 203 (WCC) at 211 E-J, see also Mediclinic Ltd v Vermeulen 2015

(1) SA 241

[15] 2001 (3) SA 1188 (SCA) at par 40

[16] 2013 (2) All SA 356 (GSJ)

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