Dept of Cellular Pathology

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Date: June 2018

To: **Leslie Brown**

Head of National Criminal Allegations Against the Police Division
Crown Office and Procurator Fiscal Service.
Scotland.

Re: Sheku BAYOH, died aged 31yrs on 3rd May 2015, in custody

You have asked me to review the autopsy pathology and relevant reports into his death, specifically to address these issues:

- To comment on the supplementary report by Liz Soilleux and in particular her conclusions on the cause and mechanism of death and the significance of sickle cell carrier status;
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- Whether the apparent sickling of cells occurred ante or post mortem;
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- From your own experience conducting autopsies in persons who are sickle cell carriers the extent to which sickling of cells is noted generally;
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- Whether it is possible or likely that sickle cell carrier status contributed to the cause and mechanism of death in this case having regard to all of the relevant factors set out in the reports.

I have been presented with:

- The original autopsy report (F542/2015);
- The supplement dated Sept 2017 that confirms that BAYOH has sickle cell trait;
- The expert witness report of Dr E Soilleux (11th Feb 2016) and her supplementary report (10th May 2018);
- The histology slides from the autopsy.

HISTOPATHOLOGY REVIEW

Brain histo N15/262

Body histo F15-542

BONE MARROW - rib sample, with traction artefact. Normocellular, no sickle cells, no viral inclusion bodies.

BRAIN – early red neurone change only (from hypoxia-ischaemia); some vessels have sickled red blood cells (SRBC).

KIDNEY – very congested; some parts just normal red blood cells (RBC), but much massive SRBC accumulations in arteries, capillaries (including glomerular) and veins. No disseminated intravascular coagulation (DIC) in glomeruli; no casts in tubules. Arteries structurally normal.

LIVER – severe congestion of vessels and sinusoids; much SRBC present (and in sinusoids); architecture normal, no steatosis; no necrosis. Bile ducts normal.

HEART – right ventricle normal; left ventricle normal apart from acute contraction bands scattered (from resuscitation); no scars, no acute ischaemia or myocarditis; much SRBC in vessels and capillaries

THYROID – normal apart from much SRBC.

ADRENAL – normal apart from much SRBC in vessels and capillaries

LUNGS – congested with distended vessels; 6 pieces sampled; areas of alveolar oedema and no associated sickling; areas of sicking in veins and alveolar capillaries; 3/6 pieces have this sickling, 3/6 have mild or no SRBC in the non-oedema areas. No thrombi, no alveolar haemorrhage.

SPLEEN AND SKELETAL PSOAS/QUADS MUSCLE – not sampled (unfortunately)

COMMENTARY

I do not need to repeat the chronology of the circumstances of death of this man who had sickle cell trait (HbAS).

From my review of the gross and histology features of the deceased's tissues, and based on my previous experience of such deaths in HbAS, and knowing the literature as it has been presented by Dr Soilleux, I believe that the HbAS trait did contribute to the death of BAYOH.

There is no doubt HbAS persons have died in custody or under stress (heat, dehydration, exercise) where the main pathogenesis is a sickle crisis affecting the lungs (particularly), the kidneys, skeletal muscle, and the heart, and this has led to death. There is no doubt that the vast majority of persons with HbAS who undergo life's daily stresses do not suffer any such syndrome. The critical aspects are

therefore the level of stress and accompanying elements such as dehydration, drugs, alcohol intake, muscle activity and body temperature.

In evaluating the clinical pathology, the critical aspects are the amount of sicking of red cells and in how many critical organs. There is no rigid morphological case definition – we must acknowledge – that separates harmless sickling from harmful sickling: it is inevitably somewhat subjective, and informed by the observer's previous experience. And it must be acknowledged that changes in the body's tissue post-mortem can contribute to sickling of red cells. But the quantity of sickling here tells me that this is much more than just post-mortem sickling; it happened peri-mortem as part of the death processes.

In the BAYOH case, I am impressed by the quantity of sickling in the organs such as the heart, kidneys, liver, thyroid and adrenal – much more than I expect to see in the organs of those with HbAS who died of unrelated causes. The lung tissues show more variable amounts of sickling, but where it is present, it is again more pronounced (ie the blood vessels are more distended) than one would normally see in HbAS persons dying of other causes.

Dr Soilleux presents a good diagram of the potential clinical pathology of death in this case, with which I do not significantly disagree apart from suggesting a more prominent component from the lung pathology. She concludes with the cause of death as:

1a. sudden cardiac death

1b. sickle cell trait, recreational drug use, struggle against restraint.

I would suggest an alternative

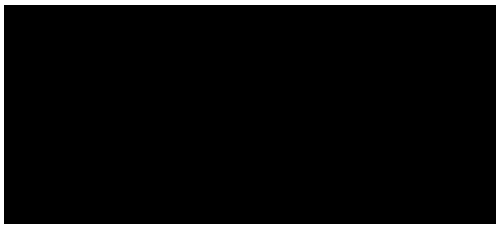
1a. sudden cardio-pulmonary failure

1b. sickle cell trait, recreational drug use, struggle against restraint

The last feature – struggle against restraint – can include positional asphyxia, but as a non-forensic pathologist, I do not wish to be drawn into a more detailed discussion in that area.

Importantly I do not think we can quantify the contribution of the three factors presented in 1b. and state, with rigor, that one is more or less important than the others. It is multifactorial.

Yours sincerely



Professor Sebastian Lucas FRCP FRCPath

Duty to the Court

I confirm that I have made clear which facts and matters referred to in this report are within my own knowledge and which are not. Those which are within my own knowledge I confirm to be true. The opinions I have expressed represent my true and complete professional opinions on the matter to which they refer.

I understand my duty to the court and I have complied with that duty and I am aware of the requirements of Part 35 of the Civil Procedure Rules, the accompanying practice direction and Protocol for Instructions to Experts to give Evidence in Civil Claims.

Qualifications

I have been a consultant histopathologist since 1980, with wide experience in general diagnostic histopathology and autopsy pathology.

I have been MRCPath from 1978, and MRCP since 1975. I have published widely on all aspects of histopathology and autopsy practice, particularly in infectious diseases.

Also since the late 1990s, I have taken a special interest in the morbid anatomy of sickle cell disease. I have performed dozens of autopsies on persons with sickle cell disease, uncountable numbers of sickle trait deaths; and been involved in medico-legal court work on many sickle disease deaths and, specifically, five previous deaths in custody or stress-related deaths among men with sickle cell trait.

GMC number [redacted]; 5-year revalidation, dated 2 Sept 2014.