Dr B / Dr D /

Dr C /

A Public Hospital

A Report by the

Health and Disability Commissioner

(Case 01HDC11283)

Health and Disability Commissioner
Te Toihau Hauora, Hauātanoa

Parties involved

Ms A	Complainant / consumer's sister
Dr B	Provider / Haematologist
Dr C	Provider / Haematologist
Dr D	Provider / Haematologist
Dr E	Customer Services Manager, the second Public Hospital
Ms F	Quality and Risk Manager, the first Public Hospital

Complaint

On 2 October 2001 the Commissioner received a complaint from Ms A concerning the services provided to Ms G, by staff at the second Public Hospital. The complaint is summarised as follows:

- *During Ms G's admission to the second Public Hospital between 26 June 2001 and her death on 7 July 2001, staff did not provide services with appropriate care and skill.*
- *During Ms G's admission between 26 June 2001 and her death on 7 July 2001, staff did not provide adequate information about her diagnosis and changes in her condition.*
- *On 27 June 2001, Ms G was woken at 2am and provided with information about her illness in an inappropriate manner.*
- *On 27 June 2001, files and plasma labelled with someone else's name were supplied for use in Ms G's treatment.*
- *On 4 July 2001, Ms G was given no assistance while toileting, despite being weak and shivering.*
- *On 6 July 2001 and 7 July 2001, Ms G's deteriorating condition was not noted and acted on quickly enough and she was not given appropriate pain relief.*

An investigation was commenced on 10 January 2002.

Names have been removed to protect privacy. Identifying letters are assigned in alphabetical order and bear no relationship to the person's actual name

Information reviewed

- Ms G's medical records from the second Public Hospital
- Ms G's medical records from the first Public Hospital
- Complaint letter from Ms A, dated 29 September 2001
- Response from Dr D, dated 4 February 2002
- Responses from Dr C, dated 31 January 2002 and 6 November 2002
- Response from Dr B, dated 5 February 2002
- Responses from the second Public Hospital, dated 28 February 2002 and 20 November 2002
- Response from the first Public Hospital, dated 15 November 2002
- Independent expert advice provided by Dr Bart Baker, haematologist, dated 4 September 2002

Information gathered during investigation

On 23 June 2001 Ms G, a woman in her twenties, underwent a blood test at the first Public Hospital after suffering dizziness and bruising. A registrar recorded in the medical records that Ms G's platelet count was 25 and that red blood cell fragments had been reported. The note is undated but is likely to have been recorded on 23 June. Ms G was found to be anaemic and thrombocytopenic (suffering a reduction in number of platelets in the blood) with blood film changes indicative of microangiopathic haemolysis (damage to the walls of the smallest blood vessels manifesting in damage to red blood cells).

A general physician at the first Public Hospital discussed Ms G's case by telephone with Dr D, the haematologist on call at the second Public Hospital for the weekend of 23/24 June 2001. She stated: "He actually gave advice which I noted in the notes and gave a differential of either an auto-immune disease, viral or other cause and suggested some investigations." A blood film was available and was discussed on the phone. The physician explained:

> "At that stage, [Dr D] didn't feel that Haematology were to be further involved …
>
> On the basis of their advice we carried out the tests as they suggested and instituted management at their suggestion. It was only due to continued deterioration of her condition and negative investigations which didn't become available until the normal working week that we felt it had to be rediscussed with Haematology at [the second Public Hospital]."

On 26 June 2001 the physician telephoned Dr B, a haematologist at the second Public Hospital, for further advice. Dr B suspected that Ms G's symptoms were caused by thrombotic thrombocytopenic purpura (TTP, a disorder that appears in a chronic form and also an acute form which is fast acting and may be fatal; see Appendix 1 for further details). Dr B arranged for an urgent transfer to a ward at the second Public Hospital. On arrival, Dr B reviewed Ms G's blood film and made a diagnosis of TTP. Dr B instructed the registrar to commence Ms G on a continuous infusion of fresh frozen plasma.

Ms A, Ms G's sister, stated that during the early hours of 27 June 2001, Ms G was woken at 2.00am and given information about TTP that she found very distressing. Dr E, Customer Services Manager at the second Public Hospital, advised that Ms G was not woken to be given information at 2.00am, but that she was already awake. Nursing notes record that Ms G had a headache on 27 June 2001 and was given Panadol at 2.00am that morning.

On 27 June 2001 Ms G was transferred to the care of Dr C, a haematologist. On the morning of 27 June Dr C and the registrar met with Ms G and explained her condition to her. Dr C proposed a plasma exchange and Ms G consented to this procedure. A femoral vascath (a catheter inserted through the groin) was placed to facilitate this. Around the time of the plasma exchange, Ms A noticed that the file and units of plasma in the surgery were incorrectly labelled "[…]" and had an April birth date on them. Ms G's full name was […] and she was born in March. It appears that this was the result of a clerical error and the blood itself was the correct match, having been matched to a blood sample taken from Ms G. The second Public Hospital advised that it has implemented a new labelling system to prevent this problem from recurring.

Further plasma exchanges occurred over the following three days with cryosupernatant plasma (CSP, a plasma component) used as the replacement fluid. Ms G's condition at this point appeared to be improving, although a rash was noted and treated on 30 June 2001.

On 1 July 2001, Ms G developed an infection at the site of her femoral vascath. The vascath was removed, plasma exchanges were temporarily stopped, and Ms G was commenced on antibiotics. Ms G's family reported that she developed a high fever and was vomiting.

On 2 July 2001, Ms G's condition began to deteriorate. Dr C advised me that he "instructed the medical staff to give her 10 units of intravenous cryo-supernatant as therapy until a further plasmapheresis cannula could be inserted". The entry in Ms G's notes for this order records:

> "[Seen by] Reg[istrar] and Dr C
>
> …
>
> P[lan] IV cryoprecipitate."

The signature on these notes is illegible.

Dr C advised me that the notation in Ms G's notes was an error and that his instructions had been incorrectly recorded. He acknowledges that "10 units of cryoprecipitate were dispensed, in contravention of my instructions". He advised me that there is no evidence to support the use of cryoprecipitate in the treatment of TTP and that the appropriate treatment is CSP, which is what he ordered. He also advised me that the terms for blood products at issue in this complaint are confusing because they both begin with the prefix "cryo".

Records from the New Zealand Blood Service indicate that 10 units of cryoprecipitate (a plasma component used in the treatment of haemophilia) were dispensed for Ms G. It appears that 10 units of cryoprecipitate were administered to Ms G in error.

In response to my provisional opinion, Dr C advised me: "The fact that cryoprecipitate had been given instead of cryosupernatant was picked up by me the day after the ward round on 3 July 2001, following a comment from the medical registrar to Dr B, that cryoprecipitate had been given. Dr B drew this matter to my attention." He further stated: "I told the medical registrar that this was not the treatment that I had prescribed during the ward round the previous day."

Dr C acknowledged that there was a communication error and that his instructions were not followed. He commented: "[I]t is not feasible or practical to follow up every oral instruction to junior staff. If such a duty were imposed, it would place an impossible supervisory and monitoring burden on all hospital specialists under current staff resource realities. Within the current system, the day to day detailed management of inpatients is the delegated responsibility of the haematology registrar on the ward."

On 3 July 2001 Dr C handed over Ms G's care to Dr B, as he was leaving the country. The two consultants had planned to install a further femoral vascath that day, but attempts to do so were unsuccessful and a line was placed in Ms G's jugular vein instead. Over the next three days there were further plasma exchanges using CSP and fresh frozen plasma (FFP).

Ms A claimed that on 4 July 2001 Ms G was not given any assistance while toileting, despite being weak and shivering and having asked a nurse for help. Ms E, Customer Services Manager at the second Public Hospital, advised me that while nurses cannot be with patients 24 hours a day, if Ms G had sought assistance it should have been provided. The second Public Hospital subsequently advised me that no request for assistance with toileting was documented, and that any verbal request would have been met in a timely manner, even if not immediately.

On 6 July 2001 Dr B noted that Ms G's blood parameters had worsened and she had developed rectal bleeding. That afternoon Ms G suffered an epileptic fit. A CT scan of Ms G's head showed no evidence of intracranial bleeding and Dr B arranged for Ms G to have intravenous immunoglobulin therapy in addition to her plasma exchange programme. Ms A stated that although she was told that Ms G was "very sick" and "we are prepared for the worst", she was not told that Ms G was going to die.

It was clear that Ms G was in a great deal of pain and very distressed that night. However, pain relief was limited to paracetamol.

In the early hours of 7 July 2001 Dr D, haematologist, ordered intravenous broad-spectrum antibiotics to be instituted to cover the possibility of a recurrence of infection. Despite these measures, Ms G's condition continued to deteriorate and Epilim (an anticonvulsant) was administered and an urgent CT scan of the head was sought. There also appears to have been some debate between staff about whether to transfer Ms G to the Intensive Care Unit at this time, and it was eventually decided that she should remain in the Haematology Ward. Ms A reported that she and her family were told that Ms G was going to be transferred and want to know why this did not occur.

Ms G went into cardiac arrest at around 6.00am and passed away shortly thereafter.

Independent advice to Commissioner

The following expert advice was obtained from Dr Bart Baker, an independent haematology advisor:

"**Conflict of Interest**
As I discussed with … of the HDC's office before agreeing to act as an advisor, the Haematology Community in New Zealand is a small one, and virtually all consultant haematologists in this country are well known to each other. The haematologists named in this complaint are all familiar to me as colleagues and acquaintances. However, the same constraints are likely to apply to any expert Haematological opinion obtained in New Zealand and, to the best of my ability, I have compiled the following report with complete impartiality, as instructed in the HDC's 'Guidelines for Independent Advisors'.

Material Reviewed
In order to complete this report I have reviewed all of the documents forwarded to me by the Commissioner's Office, including:

- Correspondence between the family of Ms G and the office of the Health and Disability Commissioner (HDC)

- Correspondence between the family and a manager of the second Public Hospital

- Several documents outlining meetings held by the staff involved in Ms G's care
- A response from a clinical leader and a manager to the concerns raised by the family
- The responses to the family's concerns from Dr D, Dr C and Dr B
- A response by Ms E on behalf of the second Public Hospital
- Photocopies of what I understand to be the complete medical records of Ms G's admissions to the first Public Hospital (23-26 June 2001) and to the second Public Hospital (26 June-7 July 2001).

I have also requested a complete summary of the blood products given to Ms G during her admissions from the New Zealand Blood Service.

Summary

In my opinion, throughout a two-week hospital stay, the provider met the standard of care and skill reasonably expected of such a provider in the circumstances, with three exceptions:

- Between 23 June and 26 June 2001, after admission to the first Public Hospital, Ms G's condition remained undiagnosed, despite features of thrombotic thrombocytopenic purpura being clearly described in her medical records. Given the rarity of this condition, the fact that she was not in a specialist haematology ward at this time, and that the diagnosis was eventually made when she failed to improve, I consider this departure to be minor and it would incur mild disapproval of other peers.

- On 26 June 2001, after arriving at the second Public Hospital, Ms G received plasma labelled incorrectly, apparently because of a clerical error in the Emergency Department. Provided that the usual precautions for labelling samples for compatibility testing and for administering blood components were followed, this did not pose a significant risk to Ms G. However, I believe that this clerical error represents a minor departure and it would incur moderate disapproval of other peers. It is of note that this departure has been recognised by the second Public Hospital and that steps have been taken to prevent a recurrence.

- On 2 July 2001, after removal of her first vascath because of infection, Ms G received 10 units of cryoprecipitate, which I consider to be a major departure from the standard of care expected under the circumstances and that this departure would incur moderate-to-severe disapproval of other peers.

There are numerous other aspects of her care during this long and complicated hospital admission that have concerned her family. While the nature of her illness (particularly in

the final stages) and the outcome were deeply upsetting for her family and for the staff involved, I do not believe, from the information provided to me, that there were other significant departures from the standard of care and skill reasonably expected of such a provider under the circumstances. In the main body of this report I have tried to address these concerns, as well as the three major issues outlined above.

Introduction

Thrombotic Thrombocytopenic Purpura (TTP) is an extremely rare and confusing disease. Understanding the issues relating to Ms G's treatment requires some understanding of this disease. I have provided a summary of the condition and what I consider to be the appropriate management as Appendix 1 of this report.

In order to facilitate my review of this case, I have also summarised the important clinical events and laboratory results, in chronological order, from the clinical records. This summary is presented as Appendix 2. There are some differences from similar summaries presented in the responses from the second Public Hospital and the staff involved in Ms G's care. I have discussed some of these differences in the main body of this report.

Specific Questions

I note that the family of Ms G have complained about a number of aspects of the care that she received during her illness. These have been summarised in the HDC's request for Medical/Professional Expert Advice, and I have been asked to provide an opinion on three questions relating to their concerns. I have framed my report around these three questions and attempted to comment specifically on the major treatment decisions made during her illness.

1. In your opinion, did the staff at the second Public Hospital provide services to Ms G with appropriate care and skill?

Prior to her transfer to the second Public Hospital, Ms G had been managed at the first Public Hospital for several days. My comments on her management during this admission are detailed under Question 3 below, since the staff at the second Public Hospital were not involved directly in her care during that admission.

A diagnosis of TTP was made by Dr B of the second Public Hospital on Tuesday 26 June 2001, after she had been contacted by the medical team looking after Ms G at the first Public Hospital and had had the opportunity to review her blood film. When the diagnosis was apparent, arrangements were made to transfer her to the second Public Hospital, and she was commenced on an infusion of fresh frozen plasma (FFP) overnight. I believe this to have been entirely appropriate management, while arranging plasma exchange therapy.

I note that one of the family's complaints relates to the fact that, during her first night in the second Public Hospital, she was "woken at 2am and provided with information

about her illness in an inappropriate manner". However, the nursing notes state that she was given Panadol at 0200 for a headache, implying that she was awake at this time, as outlined in the response from the second public hospital. Although it is not explicit in the notes, I assume that the information sheet provided was that obtained from the University of Oklahoma web site, copies of which appear in her notes. In my opinion, this is a very complete and useful summary of this rare and frightening disease. I believe that provision of this information sheet to patients with TTP and their families is appropriate, although I am unable to comment on the manner in which it was presented to Ms G, the time that it was presented, and the presence or absence of supportive discussions at the time.

On Wednesday 27 June 2001 a femoral vascath was inserted by the anaesthetics team, to enable plasma exchange to proceed. I believe this decision was appropriate for the following reasons:

- Plasma exchange is the cornerstone of treatment for TTP (George, 2000) and has been shown in one of the few randomised trials ever published in this condition to be superior to plasma infusions (Rock *et al* 1991).

- Plasma exchange requires large-bore intravenous access.

- The first Public Hospital Emergency Dept. Nursing Assessment Sheet notes that it was 'difficult to find a vein' which had necessitated the placement of a cannula in the antecubital fossa of the elbow. A large haematoma had subsequently developed around this site. Therefore, while using peripheral veins initially for plasma exchange is possible for some patients, this does not appear to have been an option for Ms G, whose peripheral veins were documented to be difficult to cannulate.

- The choice of a femoral site in the first instance was appropriate because of the markedly low platelet count, which increased the risk of bleeding. This increased risk of bleeding would have made placement of the vascath in the subclavian or jugular vein more hazardous. Platelet transfusions, which might be given to improve the platelet count in other circumstances, are generally contraindicated in TTP, because they tend to worsen the underlying condition (summarised in Rock, 2000). In my opinion, the increased risk of infection associated with femoral placement was justified under these circumstances.

Plasma exchange was started later that day and her condition was improving over the next three days, with approximately three litre plasma exchanges, using cryosupernatant plasma (CSP) as replacement fluid. The choice of CSP (variously referred to in the notes as: 'plasma cryoprecipitate depleted'; 'cryosupernatant'; 'plasma cryosupernatant depleted' [*sic*]; 'PCS'; 'cryoprecipitate depleted plasma'; and 'cryo depleted plasma') as replacement fluid was appropriate, in my opinion, and the reasons for choosing this component are outlined in Appendix 1.

By Saturday 30 June, this therapy was clearly proving effective, with Ms G's platelet count rising to 45 x 10^9/L, and her haemoglobin rising to 127 g/L.

Unfortunately, the following day, her condition deteriorated, with evidence of an infection in her vascath, necessitating abandonment of her plasma exchange, removal of the vascath, and the commencement of antibiotics. Problems with venous access devices are unfortunately common in this condition (Rizvi *et al*, 2000) and I do not believe that this infection, which led to her final deterioration, can be attributed to a lack of appropriate care or skill. Attempts at insertion of a peripheral line were unsuccessful and a midline catheter was inserted to allow parenteral antibiotic therapy. I consider these steps to have been appropriate, under the circumstances. Dr C's response notes that arrangements were made for 'supplementary plasma' to be given and, although I can't find evidence of this being prescribed, the nursing notes suggest that 2 units of FFP were given during the evening shift that day, after removal of the vascath, and it would be appropriate to maintain an infusion of FFP or CSP while awaiting the insertion of a further vascular access device to allow recommencement of plasma exchange.

On Monday 2 July, her blood indices had deteriorated significantly, her platelet count falling to 8 x 10^9/L and her haemoglobin to 92 g/L. At a ward round that day, attended by 'Reg and Dr C', the decision appears to have been made to give 'IV cryoprecipitate'. Dr C's recollection in his response dated 31 January 2002 was that he 'instructed the medical staff to give her 10 units of intravenous cryo-supernatant as therapy until a further plasmapheresis cannula could be inserted'. He further recalls that 'she was given 3 units of cryoprecipitate and 7 units of cryo-supernatant plasma, not 10 units as cryoprecipitate as is actually stated in the House Surgeon notes.' However, I believe that 10 units of cryoprecipitate were given that day, for the following reasons:

- The clinical record shows reviews by 'Haem Reg.' at 1700 after 3 units of cryoprecipitate and by '2nd On' after 7 units of cryoprecipitate. Moreover, in what I take to be the last nursing note of this day, it is stated that '10 units cryoprecipitate given without incident'.

- According to the Intravenous Fluid Order Forms contained in the material provided to me, 10 units of cryoprecipitate were prescribed and administered between about 1600 and 2300 hours on 2 July 2001.

- The separate blood transfusion record sheets appear to confirm that 10 units of cryoprecipitate were issued to her on 2 July between 1530 and 2230 hours. These records also appear to show a further 2 units of cryoprecipitate being issued on 1 July at 1730 and at an illegible time, although I can find no other record in the notes of these 2 units being either prescribed or administered.

- Because there may have conceivably been confusion about the nature of product that was being administered (the multiple different terms used to describe CSP by the

apheresis nurses, as outlined in Appendix 2, suggest this possibility), I have requested from the New Zealand Blood Service a complete record of all blood components issued to Ms G during her admission. These confirm that 13 units of cryoprecipitate were issued to her on 2 July 2001. Although 3 of these were returned, 10 are recorded as having been transfused.

I believe that the administration of cryoprecipitate at this time to be a significant departure from the standard of care and skill reasonably expected in these circumstances. As outlined in Appendix 1, appropriate management of active TTP involves the administration of either FFP or CSP, which contain the VWF-cleaving metalloproteinase, ADAMTS-13 that is lacking in the blood of patients with most forms of TTP (Moake, 2002). Although knowledge about the precise nature of ADAMTS-13 has improved since July 2001 when Ms G was treated, I believe that it was established knowledge at that time that the active therapeutic substance in this disease is 'present in CSP in the same concentration as in plasma – it is not removed in the cryoprecipitate' (Rock, 2000). While there is some uncertainty about whether use of CSP in TTP achieves superior results to the use of FFP (summarised in George, 2000, Rock, 2000 and Blackhall *et al*, 2001), I am not aware of any evidence to support the use of cryoprecipitate in this condition and there are theoretical reasons (most notably the increased concentration of high molecular weight multimers of VWF, and the absence of ADAMTS-13) for believing that it might be potentially harmful. Cryoprecipitate is used to treat low fibrinogen levels, which can be associated with disseminated intravascular coagulation secondary to serious infection. However, the only fibrinogen assays that I can find in her notes are normal (3.1g/L on 29 June and 4.2 g/L on 3 July), making it unlikely that this was the rationale behind the use of this product.

It would seem from Dr C's response that he had intended 'cryo-supernatant' (CSP) to be given on this day and this would have been appropriate therapy under the circumstances. I can only assume that the administration of cryoprecipitate instead of CSP was due to a communication error.

On 3 July, it was planned for a further femoral vascath to be inserted. Again, this decision seems appropriate for the reasons outlined above. Unfortunately, attempts at placement of a femoral line were unsuccessful and, after an unsuccessful attempt at inserting a subclavian line, a jugular line was placed successfully. Although the changing plans were distressing to Ms G and her family, I believe that these were appropriate, for the following reasons:

- Without adequate vascular access, she would have been unable to receive appropriate therapy (plasma exchange) for her deteriorating, life-threatening condition.

- Because of the risk of bleeding an attempt was made, appropriately, to insert a second femoral catheter.

- When the attempt at placing a femoral catheter failed, further attempts to place the vascath in more central sites were appropriate, despite the risk of bleeding, because the risk of bleeding at this time was outweighed by the risk posed by the disease, in my opinion.

From 3 July, Ms G received appropriate therapy for her TTP, with increasingly aggressive plasma exchange. Despite this, her condition deteriorated, which leads on to the second of the specific questions asked of me.

2. In particular, on 6 July 2001, 7 July 2001, was Ms G's deteriorating condition detected early enough and was the appropriate treatment and pain relief administered?

Because of the clear evidence of a continuing deterioration on 6 July, secondary treatments, including Intravenous Immunoglobulin and Vincristine, were introduced. I consider these to have been reasonable choices, and, although it could be argued that they should have been considered earlier, there are only anecdotal data to support their use in this condition (George, 2000, Rock, 2000). Moreover, her deterioration appears to have been initiated by the *Staph aureus* bacteraemia, which became apparent on 1 July. This was being treated appropriately with catheter removal and antibiotics, while plasma exchange had been recommenced. For several days after such an infection, it would not seem unreasonable to expect that the disease would be brought back under control, and to withhold other potentially toxic therapies of uncertain benefit, while awaiting a response to the reintroduction of plasma exchange.

Consideration had also been given to twice daily plasma exchange, which has been suggested in deteriorating TTP. However, there are no studies to support this option, and it is very difficult to sustain, both for the patient and for the apheresis service. Twice-daily exchange has been described as a 'formidable procedure, requiring nearly full-time personnel commitment' (George, 2000). Furthermore, on 5 July, in addition to the daily plasma exchange, she was given supplementary infusions of FFP. Other options that have been suggested for this condition include splenectomy, cyclophosphamide, and anti-platelet agents. However, I don't believe that these would necessarily have been appropriate for the following reasons:

- Support for these treatments comprises anecdotal reports only.
- She was clearly not fit to undergo splenectomy by this time.
- Treatments that may act by suppressing the immune system, such as cyclophosphamide, are generally relatively slow-acting.
- She was already apparently bleeding from the bowel, making anti-platelet agents a potentially hazardous treatment.

- She had significant problems with nausea, vomiting and diarrhoea, which were likely to have been exacerbated by cyclophosphamide and, possibly, anti-platelet agents.

Despite these measures, Ms G's condition steadily deteriorated, with rapidly rising LDH and bilirubin levels, together with persistently low platelet levels. Moreover, the nursing notes of 'blood-stained +++' plasma collected during apheresis and 'bright red urine' on 6 July probably reflect severe intravascular haemolysis, leading to free haemoglobin in the plasma and haemoglobinuria. I believe that the surprisingly high platelet counts described during the last 24 hours of Ms G's life were probably artefactual, and were most likely caused by severe red blood cell fragmentation. In these circumstances, automated cell counters often count red cell fragments as platelets, because of their similar size.

The clinical manifestations of this deterioration were her seizures and, possibly, her abdominal symptoms, although the latter may have been due to a number of factors, including bleeding, the bacteraemia, and the antibiotic treatment. I believe that the response to her seizures was appropriate, with an urgent CT scan to exclude bleeding and the introduction of intravenous sodium valproate (Epilim).

It is very difficult to comment on the issue of pain relief during the night of 6-7 July. It seems likely that some of her agitation may have been secondary to cerebral irritation due to the microvascular ischaemia caused by her worsening TTP. She was also post-ictal. Although her obvious agitation was very distressing, the desirability of adequate sedation and pain relief was counterbalanced by the need to accurately assess her neurological status, because of her recent seizures and because of the very real possibility of intracerebral bleeding. Powerful sedating or pain-relieving drugs such as morphine would have compromised this assessment and, for this reason, paracetamol was recommended. In addition, according to Dr D's response, intravenous diazepam was also considered, primarily for seizure control.

Had it been clear that Ms G was dying, it would have been unreasonable not to use whatever drugs were necessary to make her last few hours as comfortable as possible. However, the on-call staff did not have the luxury of hindsight and, in my opinion, the decisions made on pain relief appear to have been reasonable under the very difficult circumstances facing them. That Ms G was very agitated during her last hours was an unfortunate consequence of the requirement to monitor her neurological status closely.

Finally, there was some discussion of transfer to the Intensive Care Unit, and there appear to have been conflicting messages given to Ms G's family about this during her final deterioration. I believe that, until shortly before her cardiac arrest when she developed a tachycardia and hypotension, there was little that the ICU could have provided that was not being provided on the Haematology Ward.

3. Are there any aspects of any of the hospital staff which you consider warrant:
- *Further exploration by the investigation officer?*
- *Additional comment?*

In my opinion, there are three other matters in this case that deserve comment.

1. The first is the length of time taken to diagnose or suspect the condition after the initial admission to the first Public Hospital. In retrospect, on the day of her initial admission to the first Public Hospital, Ms G had typical features of TTP documented in her notes, with a haemolytic anaemia, red blood cell fragmentation and thrombocytopenia. Although she did not exhibit a fever, neurological abnormalities or renal impairment (which, together with the existing features, would have constituted the classical 'pentad' of TTP features, described in Appendix 1), the features present on admission were, in my opinion, sufficient to at least suspect the diagnosis (Rock, 2000). However, this is a very rare condition, and the case was discussed with 'haematology' by the physician, presumably on the day of admission (although the entry in the case notes is dated '22/6', the day before admission). It is not clear who 'haematology' was and I am unable to speculate on whether all of the features of her presentation, including the significant RBC fragmentation were discussed. A further three days elapsed before the case was discussed with Dr B and the correct diagnosis was first mentioned in her notes. She was then transferred to the second Public Hospital and appropriate treatment was initiated promptly.

It is acknowledged in the second Public Hospital's response by Ms E that the diagnosis could have been made earlier and I would agree with that assessment. However, there appears to be some confusion in her report about the dates on which abnormalities were noted, with the suggestion that the 'first medical notation is 24 June' and the 'first reference to a low platelet count is on 25 June'. It is my opinion that the first medical notation is that made by the medical registrar, whose signature appears to be ['the registrar'], timed 1750, but undated. It seems very likely that this is the admission note on 23 June and it clearly documents a platelet count of 25 and the presence of RBC fragments reported by the laboratory.

2. The next issue that requires comment is the apparent mislabelling of the plasma given to Ms G on 26 June. This has been addressed in the responses from Dr B, a clinical leader / a manager and Ms E. It would seem that the problem arose when an incorrect set of identity labels were printed by the Admitting Clerk in A&E. Although the family's concern about this is understandable, it seems likely that this did not pose any significant risk. Provided that samples for compatibility testing are labelled from the patient's wrist band and that blood components are checked against that wrist band before administration, there is no increased risk that a component of the wrong blood group will be given, even if the details on the patient's wrist band are inaccurate. This unfortunate error has been acknowledged, and 'the process leading to this error has been

investigated to ensure such an event does not occur in the future', according to Ms E's report.

3. Finally, numerous concerns have been expressed by the family of Ms G about the level of communication with her and the family during her illness. These have been answered in the responses of the doctors involved in her care, as well as in Ms E's response on behalf of the second Public Hospital. Commenting on these concerns by means of a retrospective review of the case notes is very difficult. The following factors contributed to the concerns about communication relating to Ms G:

- The rarity of her illness and the complexity of the treatment
- The rapid changes in her condition, particularly in the last few days of her life
- The need for frequent and, sometimes, unexpected changes in management (*e.g.* the decisions surrounding the placement of her second catheter)
- Apparent conflicts of opinion between the 'on call' staff and the haematology team (*e.g.* the need for ICU transfer on the night before she died)
- The fact that overall responsibility for her care (unavoidably) changed during her stay in hospital, with Dr C's departure overseas
- The doctors' need to balance communicating the gravity of the situation to Ms G and her family, with a desire to avoid causing excessive anxiety and distress

While Ms G's family have expressed the opinion that communication was inadequate, the responses by the specialists involved in her care dispute this. In particular, the response by Dr B describes frequent and regular communication with Ms G and her family during the last day or two of her life and after her death. It also describes offers of further information that were not always accepted.

Under the circumstances, and acknowledging the difficulties in commenting on this aspect retrospectively, I believe that communication was delivered with appropriate care and skill in this case.

APPENDIX 1: SUMMARY OF THROMBOTIC THROMBOCYTOPENIC PURPURA

Thrombotic thrombocytopenic purpura (TTP) is a very rare disorder with an annual incidence in various reports of between 3.7 cases per million and approximately 1 case per 50,000-100,000 (summarised in George, 2000). The reported incidence appears to be rising, possibly because of a greater awareness of the condition (Rock, 2000). Sometimes the name TTP-HUS is used for this syndrome, because of the similarity between TTP and the haemolytic uraemic syndrome (HUS). TTP-HUS is the name used in the information sheet produced by the University of Oklahoma, a copy of which is included in Ms G's hospital notes.

No cause can be identified in most adult cases of TTP but it appears that, in many cases, the initial problem is the production by the patient's immune system of an antibody against endothelial cells (which line the blood vessels) and/or against an important enzyme in the blood, called ADAMTS 13 (Moake, 2002). This enzyme's function is to break down large, high-molecular-weight multimers of von Willebrand factor (VWF), one of the proteins involved in the initiation of blood clotting.

Damage to the endothelial cells and/or reduction in the levels of ADAMTS 13 lead to the presence of abnormally large forms of VWF in the blood stream, and these forms cause abnormal aggregation of the small clotting cells, called platelets, in small blood vessels (arterioles) around the body. This leads to:

- A reduction in the number of platelet cells in the blood (known as thrombocytopenia), which can lead to abnormal bleeding and bruising.

- Destruction of the red blood cells (RBC) as they travel through the arterioles blocked by platelet aggregates, which can cause anaemia and the appearances of fragmented RBC on blood films. This mechanical fragmentation of RBC is described as 'microangiopathic haemolytic anaemia'.

- Reduced blood supply to organs all around the body, leading to organ damage. This is often most pronounced in the kidneys and the brain, leading in some patients to kidney failure and neurological symptoms, including epileptic seizures.

- Fever can also be a feature of this process

Classically, TTP was described as a 'pentad' of five features: microangiopathic haemolytic anaemia; thrombocytopenia; abnormal kidney function; neurological symptoms; and fever. However, it has become apparent over recent years that most sufferers of this condition do not have all of these features at the time of diagnosis. It is currently felt that the presence of a microangiopathic haemolytic anaemia and thrombocytopenia in the absence of any other possible causes of these phenomena is adequate for a presumptive diagnosis of TTP (Rock, 2000).

TTP used to be fatal for the vast majority of sufferers until trials of plasma infusions and, subsequently, plasma exchange have been shown to be effective therapy over recent decades. Plasma is the liquid component of blood, in which numerous proteins and other substances are dissolved. It is usually stored in the frozen state as fresh frozen plasma (FFP) and thawed just prior to use. It is thought that infusion of FFP replenishes ADAMTS 13 and that plasma exchange may remove the high molecular weight multimers of VWF, as well as allowing increased volumes of plasma (containing ADAMTS 13) to be given as replacement fluid.

When FFP is thawed between 2ºC and 8ºC a precipitate, which is rich in VWF, clotting factor VIII, fibrinogen and some other proteins, forms. This product is called

cryoprecipitate and it was once used to treat haemophilia, before the availability of more purified concentrates of Factor VIII. Its use is largely restricted nowadays to replacing fibrinogen in certain clinical states, including disseminated intravascular coagulation. The plasma component that is left when FFP is thawed under these conditions is called cryosupernatant plasma (CSP). A number of other names are used for this component, including: cryo-supernatant; cryoprecipitate-depleted plasma; cryoprecipitate-reduced plasma; and plasma cryosupernatant (PCS). There are theoretical and clinical reasons to suggest that CSP may be a better replacement fluid than FFP in TTP:

- CSP is relatively depleted of the high molecular weight multimers of VWF that are thought to cause the platelet aggregation that is the primary pathophysiological event in TTP

- CSP appears to be the component in which ADAMTS 13 segregates during the manufacture of cryoprecipitate from FFP (Rock 2000)

- CSP appeared superior to FFP as a replacement fluid in plasma exchange for TTP in one retrospective study although this has not been confirmed in a small, prospective, randomised study (reviewed in George, 2000 and Rock, 2000)

Although plasma-based therapy is the mainstay of treatment for this condition, other treatments for TTP have been reported to be of benefit in some patients, including: steroids; vincristine; anti-platelet agents such as aspirin and dipyridamole; intravenous immunoglobulin; cyclophosphamide; and splenectomy (reviewed in George, 2000 and Rock, 2000).

In 2001, with appropriate treatment, approximately 80-90% of patients with TTP would have been expected to survive the illness although it should be noted that, even with appropriate care, 10-20% still die of this disease.

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APPENDIX 2: SUMMARY OF SIGNIFICANT EVENTS

Saturday 23 June 2001: Ms G was admitted to the first Public Hospital after a blood count had shown her to be anaemic and thrombocytopenic, with evidence of microangiopathic haemolysis ('cell fragments ++' noted in admission record of […], Med Reg, which was undated, but I assume to be written on 23 June)

A further entry, dated '22/6' (I assume this to have been the 23/6) by the physician at the first Public Hosptial; noted: 'D/W Haematology…ΔΔ Autoimmune or 2º to viral or 2º to other. They suggest if Direct Coomb's is +ive to give Prednisone 1 mg/kg'. It is not clear from this entry whether a haematologist or the laboratory was being consulted and whether details of the thrombocytopenia, the anaemia, and the RBC fragmentation were discussed. Subsequently the Direct Coomb's Test was shown to be negative and prednisone was withheld.

Haemoglobin (Hb) 70 g/L; Platelet count (Plt) 25 x 10^9/L; LDH 1894; Bilirubin (BR) 48 µmol/L.

Sunday 24 June 2001: Hb 61 g/L; Plt 29 x 10^9/L; BR 55µmol/L.

Transfused 3 units RBC

Monday 25 June 2001: Hb 90 g/L; Plt 34 x 10^9/L.

Feeling better; plan to D/W Haematology. Large haematoma at site of IV cannula, which had tissued.

Tuesday 26 June 2001: Hb 87g/L; Plt 7 x 10^9/L; LDH 3850U/L; BR 75 µmol/L.

Discussed with Dr B who was to review the blood film. Platelet count subsequently found to be < 10 x 10^9/L and further efforts to contact haematology team at the second Public Hospital. Transfer to the second Public Hospital agreed 1545hrs after diagnosis of TTP made by Dr B.

Infusion of fresh frozen plasma (FFP) was commenced via a peripheral line and she received 10 units of FFP overnight, complicated by some urticaria (hives).

Wednesday 27 June 2001: Hb 70 g/L; Plt 9 x 10^9/L.

Vascath inserted into R femoral vein by anaesthetics team and plasma exchange of 2.9 litres completed, using 'plasma cryoprecipitate depleted' as exchange fluid. Exchange complicated by symptoms suggestive of hypocalcaemia, managed with slowing rate of exchange and oral Calcium Sandoz.

Transfused 3U of RBC.

Thursday 28 June 2001: Hb 105 g/L; Plt 12 x 10^9/L; BR 44µmol/L.

Plasma exchange of 2.9 litres, using 'cryosupernatant' as exchange fluid. No problems apart from some skin rashes noted subsequently.

Prednisone 40mg daily started.

Friday 29 June 2001: Hb 114g/L; Plt 22 x 10^9/L; BR 30µmol/L; Fibrinogen 3.1 g/L.

Plasma exchange of 2.8 litres, using 'Plasma Cryosupernatant Depleted' [*sic*] as exchange fluid.

Saturday 30 June 2001: Hb127 g/L; Plt 45 x 10^9/L.

Plasma exchange of 3 litres. 'Line site not infected' 'Anxious about prognosis and asking lots of questions'.

Sunday 1 July 2001: Hb 126 g/L; Plt 22 x 10^9/L; LDH 1377 U/L.

Febrile (>38^o) with 'chills'; Vascath site 'puffy, red', 'looking very infected'.

Plasma exchange of 2 litres, using 'cryo-depleted plasma' abandoned after Louise developed coughing, chest pain, hypotension, rigors.

Unsuccessful insertion of peripheral line x3. Vascath removed. Midline catheter inserted and commenced on cefuroxime, vancomycin and single dose of gentamicin.

Monday 2 July 2001: Hb 92 g/l; Plt 8 x 10^9/L.

Fever and chills overnight. 'Vascath site clean'

'P: IV cryoprecipitate'

Nausea, vomiting and diarrhoea during day

Given 10 units of cryoprecipitate from 1600-2245hrs.

Tuesday 3 July 2001: Hb 73 g/L; Plt 6 x 10^9/L; BR 56µmol/L; Fibrinogen 4.2g/L.

Generalised abdominal pain - ? examination findings not recorded

Staph aureus confirmed on blood culture. Flucloxacillin started.

Anaesthetics – failed insertion of left femoral and left subclavian lines. Left Internal Jugular insertion successful.

Transfused 4 U RBC.

Plasma exchange of 2.9 litres with 'PCS' replacement.

Wednesday 4 July 2001: Hb 85 g/L; Plt 6 x 10^9/L; LDH 3003 U/L; BR 60 µmol/L.

Afebrile. Midline catheter removed.

Plasma exchange of 3.3 litres with 'cryoprecipitate depleted plasma' replacement.

Plan for former TTP patient […] to visit in two days.

Thursday 5 July 2001: Hb 113 g/L; Plt 2 x 10^9/L; LDH 2926 U/L.

Nausea++. Feeling down.

Catheter site clean. Fem line site clean. 'Good Bowel sounds'

Folic acid started.

4 Units FFP.

Plasma exchange of 3.2 litres with 'cryo depleted plasma'

Friday 6 July 2001: Hb 81g/L; Plt 10 x 10^9/L; LDH 5401 U/L; BR 142 µmol/L

'Feels awful', 'mind not thinking clear', 'down in mood', Abdo pain – crampy in nature, diarrhoea x 1, vomit x 1

Plasma exchange of 3.8 litres with 'PCS' replacement. Plasma collected 'blood stained +++'

Plan for 4U RBC followed by 4 U FFP overnight.

1730 hrs: probable seizure

Reviewed with consultant: Plan CT scan to exclude IC bleed

IV Valproate

Vincristine 2 mg IV

IVIG 2 g/Kg over 5 days

2230 Nursing notes: 'irritable all duty'; 'Vague and at times unresponsive. Cannot verbalise. Requires nurse constantly present'; 'Incontinent of urine at times – has used commode x1 – bright red urine'; T 39-38.8

Saturday 7 July 2001: Hb 108g/L; Plt 162 x 10^9/L; LDH 8107 U/L; BR 201µmol/L

S/B Night Reg.: Seizure – collapsed while on commode (? 2330 hrs from subsequent nursing note); 'bowel incontinent'; 'very agitated, throwing herself around. Not responding to calls. Responding to pain.'

D/W Dr D: 'Supportive R; Neurology to see mane; Panadol P/R – temp 38ºC, start cefepime 1 gram BD; Epilim 500mg IV stat foll. by oral from tomorrow; routine bloods = blood c/s; ct. FFP / slow intragam'

0420 hrs: S/B (?) Night float re: tachycardia. 'Pt agitated / throwing arms around. Responding to painful stimuli. O/E agitated – RR 28/min, PR 150/min regular good volume, BP143/93'. 'ECG SR 150/min, N axis, no acute ST changes.'

0515: BP 115/-

0540: BP 64/49

0550: 'Incontinent of haematuria O/N. Small melaena O/N, then 100mL @ 510, then 100mL @0530, then further melaena'. 'S/B Night Reg in view of melaena of 200mL as reported by nurse'

0557: Arrest call. Asystole throughout 36 mins of CPR."

Responses to Provisional Opinion

Ms A

I acknowledge Ms A's heartfelt response to my provisional opinion. She expresses concern about the care provided to her sister and the circumstances surrounding her death. I acknowledge the pain and anguish suffered by Ms G's family.

The second Public Hospital

The second Public Hospital advised me, in response to my provisional opinion, that its policy on "Transfusion of Blood Products" has recently been revised in consultation with the New Zealand Blood Service:

> "The new policy *blood component and blood product administration and the management of transfused patients (adults)* is an extension document that is currently awaiting sign off by the Quality Improvement Group, within the organisation. Once this is complete [the second Public Hospital] IV Nurse Consultant plans an extensive education program throughout the organisation and following on from that, an audit will be conducted by the organisation to ensure compliance with the new mandatory policy."

The second Public Hospital also advised:

> "The [Hospital has] undertaken a commitment to providing further education and training to [its] nursing staff. Two nurses have already attended the Blood Cell Transplant course at […] Hospital this year and four nurses are currently enrolled on a distance learning course at The New South Wales College of Nursing, Haematology and Haemopoietic Transplantation Nursing course. [The second Public Hospital] hope that through this further education and training, the nurses are able to reflect and challenge their practice, consolidate/acquire knowledge and implement new policies to ensure future safe practice in this area."

Dr C

In response to my provisional opinion, Dr C stated: "In a hospital setting it is always a serious matter when the instructions of a Consultant are not followed, and such behaviour can not be excused or condoned … The administration of cryoprecipitate was an error but not serious in the sense of having an adverse consequence for the patient." Dr C accepted that it was a "serious error in terms of clinical practice [with] serious implications for hospital systems".

In summary, Dr C submitted:

> "I acknowledge that an error was made. It was a communication error and resulted in my instructions being misrecorded. As such, it was unintentional and unforeseeable. As

the consultant charged with the oversight of Ms G's care, I must accept responsibility for that. I do not consider, however, that it therefore follows that I failed to meet the standard of a '*responsible consultant haematologist*', however that is defined."

Code of Health and Disability Services Consumers' Rights

The following Rights in the Code of Health and Disability Services Consumers' Rights are applicable to this complaint:

RIGHT 4
Right to Services of an Appropriate Standard

1) Every consumer has the right to have services provided with reasonable care and skill.

…

3) Every consumer has the right to have services provided in a manner consistent with his or her needs.

RIGHT 6
Right to be Fully Informed

1) Every consumer has the right to the information that a reasonable consumer, in that consumer's circumstances, would expect to receive, including –

a) An explanation of his or her condition; …

Opinion: Breach – The second Public Hospital

Incorrectly labelled plasma
On 27 June 2001 plasma was given to Ms G with someone else's name and date of birth stamped upon it. The second Public Hospital advised me that this was the result of a clerical error and that the plasma was the correct product for Ms G, having been matched to a blood sample taken from her. However, the sight of what appeared to be an incorrect blood product being administered to Ms G was naturally distressing for her family and presumably even more distressing for Ms G herself. In addition, these incorrectly labelled products had the potential to cause confusion and delay in Ms G's treatment. In my opinion, by not having a system in place to ensure that blood products were correctly

labelled, the second Public Hospital did not provide services with reasonable care and skill and breached Right 4(1) of the Code of Health and Disability Services Consumers' Rights.

I note that the second Public Hospital has advised that a new labelling system has been implemented which should prevent this problem from recurring.

Manner in which Ms G was informed of her diagnosis
Ms A complained that Ms G was woken at 2.00am on 27 June 2001 and handed an information sheet about her illness, which she found very distressing.

I accept that clinicians should not decide to withhold information in the belief that to do so may cause anxiety and distress. However, waking a patient at 2.00am to provide her with information that could wait until morning is inappropriate. The second Public Hospital advised me that when Ms G was provided with an information sheet about her illness in the early hours of 27 June, she was already awake. Nursing notes record that she was awake and given Panadol at 2.00am after complaining of a headache.

Ms G was entitled to an explanation of her condition. However, she was also entitled to receive this information in a manner consistent with her needs. I am satisfied that Ms G was already awake when she was provided an information sheet in the early hours of 27 June 2001. However, I do not accept that it was appropriate for nursing staff to give Ms G a complex information sheet about her illness at 2.00am, even if she was awake. In these circumstances, the second Public Hospital breached Right 4(3) of the Code by the manner in which information was provided to Ms G.

Toileting
Ms A complained that on 4 July 2001 Ms G was not given any assistance while toileting, despite being weak and shivering. Ms E, Customer Services Manager at the second Public Hospital, advised me that while nurses cannot be with patients 24 hours a day, if Ms G had sought assistance it should have been provided. I agree. Verbal requests for toileting assistance should be met as soon as reasonably practicable.

In my opinion, by failing to assist Ms G when she had requested help and clearly required it, the second Public Hospital failed to ensure that Ms G was provided with services in a manner consistent with her needs and breached Right 4(3) of the Code. The fact that there is no documented evidence that a request was made for assistance with toileting or that it was denied does not, in my view, mean that assistance was not sought or required. I am satisfied that Ms A's recollection of the lack of assistance in response to her sister's request for help to go to the toilet is likely to be accurate.

Opinion: Breach – Dr C

Use of cryoprecipitate
I accept that although Dr C ordered 10 units of CSP to be administered to Ms G on 2 July 2001, 10 units of cryoprecipitate were dispensed instead.

In his recollection of events, Dr C is clear that he instructed staff to administer CSP. Ms G's notes do not record this instruction, instead noting that cryoprecipitate be given intravenously. Dr C advised me that this was a communication error and resulted in his instructions being misrecorded. As a result, cryoprecipitate was administered to Ms G in error.

This was a serious error. In his response to my provisional opinion, Dr C acknowledges that the matter is a serious error in terms of clinical practice, with serious implications for hospital systems. I agree. Even if Ms G suffered no adverse consequence from the administration of cryoprecipitate, she was denied appropriate treatment because the correct therapeutic agent, CSP, was not administered.

The error may be partly attributable to a lack of consistency in the language used to describe blood products. Dr Baker pointed out that at various points CSP alone is referred to in the notes as "plasma cryoprecipitate depleted", "cryosupernatant", "cryosupernatant depleted", "PCS", "cryoprecipitate depleted plasma" and "cryo depleted plasma". While these terms and those used to describe other blood products such as cryoprecipitate make sense to a specialist haematologist, it is easy to see how they could cause confusion for an inexperienced house surgeon or nurse.

In his response to my provisional opinion, Dr C acknowledged that his instructions were not followed. He submitted: "[I]t is not feasible or practical to follow up every oral instruction to junior staff. If such a duty were imposed, it would place an impossible supervisory and monitoring burden on all hospital specialists under current staff resource realities." I agree with these comments; however, if instructions are given that are complex or easy to misinterpret they should be checked. A check of the notes would have picked up this error. In my opinion, in this instance, Dr C had a greater responsibility because of the rarity of Ms G's condition, the rarity of the use of the therapeutic agent, and the confusing nature of the terminology of blood products. Dr C acknowledged that "the terms used for the blood products at issue in this complaint are confusing because they both begin with the prefix 'cryo'".

Ultimately Dr C was the consultant responsible for Ms G's care on 2 July 2001, and for ensuring that his orders were interpreted correctly and carried out appropriately. Dr C failed to ensure that his orders were carried out appropriately and must accept responsibility for Ms G receiving the wrong blood product. In these circumstances, Dr C failed to meet the standard of reasonable care and skill expected of a responsible consultant, and breached Right 4(1) of the Code.

Opinion: No breach – The second Public Hospital

Employers are vicariously liable under section 72(2) of the Health and Disability Commissioner Act 1994 for ensuring that employees comply with the Code of Health and Disability Services Consumers' Rights. Under section 72(5) it is a defence for an employing

authority to prove that it took such steps as were reasonably practicable to prevent the employee from doing or omitting to do the thing that breached the Code.

Use of cryoprecipitate
Dr C was an employee of the second Public Hospital. However, in the absence of reasonable cause for concern, it is not the responsibility of a Public Hospital to ensure that senior clinicians communicate orders correctly and check that their orders are carried out appropriately. Accordingly the second Public Hospital is excused from vicarious liability for Dr C's breach of Right 4(1) of the Code.

Opinion: No breach – Dr B, Dr C and Dr D

Information about diagnosis and changes in Ms G's condition
Ms A complained that Ms G did not receive adequate information about her diagnosis and the significance of changes in her condition. On 27 June 2001 Ms G was provided with a comprehensive information sheet on TTP. However, she received little information prior to this and little about the changes that occurred in the following days.

It is clear that there were difficulties in keeping Ms G fully informed about her condition. However, my expert advisor noted that this was understandable given:

- the rarity and complexity of Ms G's illness
- the rapid changes in her condition, particularly in the last few days of her life
- the need for frequent, and sometimes unexpected, changes in her management
- apparent conflicts in the opinions of different staff involved in Ms G's care
- the fact that overall responsibility for Ms G's care changed during her admission owing to Dr C's departure overseas
- the need to balance communicating the gravity of Ms G's condition, with a desire to avoid causing excessive anxiety and distress.

Under Right 6(1)(a) of the Code, every consumer is entitled to an explanation of his or her condition. In this case I believe that the information provided to Ms G was as clear and appropriate as possible in the circumstances of her complex and rapidly changing condition. Therefore, in my opinion, Dr B, Dr C and Dr D did not breach Right 6(1)(a) of the Code with respect to the information provided to Ms G.

Plasma labelling
As noted above, the plasma provided to Ms G on 27 June 2001 was the correct blood product, but had been incorrectly labelled owing to a clerical error. Dr B and Dr C were responsible for Ms G's care this day. I accept my expert advice that they ensured that the correct blood product was administered. Therefore, in relation to this incident I am satisfied

that Dr B and Dr C provided services with reasonable care and skill and did not breach Right 4(1) of the Code.

Pain relief, diagnosis and treatment on 6 and 7 July
Ms A complained that Ms G's deteriorating condition was not diagnosed and treated early enough and that in the hours before her death on 7 July 2001, she was not provided with adequate pain relief. The clinicians responsible for Ms G's care on 6 and 7 July 2001 were Dr B and Dr D.

Dr Baker advised me that in light of the infection experienced by Ms G on 1 July 2001, it was appropriate for potentially toxic secondary treatments such as intravenous immunoglobulin to be withheld until the plasma exchange had been reintroduced. Ms G's condition began to deteriorate rapidly on 6 July 2001 and at this point the secondary treatments were instituted. Despite these measures, Ms G's condition continued to deteriorate. Epilim was administered and an urgent CT scan sought. There appears to have been some debate between staff about whether to transfer Ms G to the Intensive Care Unit. Dr Baker advised me that the treatment provided and the decision not to transfer Ms G were appropriate, as little more could have been done for her in ICU as opposed to the Haematology Ward. Dr Baker also advised that it would not have been clear to staff at this point that Ms G was dying.

During the night of 6/7 July 2001, Ms G was clearly distressed and in pain. Records indicate that the only pain relief administered to her was paracetamol. Dr Baker advised me that stronger pain relief agents would have hindered the assessment of Ms G's neurological status.

I accept my expert advice. In my opinion Ms G's deterioration on 6 and 7 July 2001 was rapid and could not have been diagnosed earlier. The treatment provided was appropriate and administered within reasonable timeframes. I also accept that the pain relief given to Ms G on the night of 6/7 July 2001 was appropriate given that any stronger pain relief could have compromised the assessment and treatment of her condition. In my opinion Dr B and Dr D provided services to Ms G with reasonable care and skill and did not breach Right 4(1) of the Code in relation to her care on 6/7 July 2001.

Other comments

Dr Baker advised me that he was concerned that Ms G's condition was not diagnosed at the first Public Hospital between 23 and 26 June 2001 despite features of TTP being clearly described in her clinical record. He regarded this delay as only a minor departure from good practice given the rarity of the condition and the fact that Ms G was not in a specialist haematology ward.

In the first Public Hospital's response to my provisional opinion, the physician advised that she discussed Ms G's case with Dr D, the on-call haematologist for the weekend. In my opinion her action in following Dr D's advice was appropriate.

Actions taken

Dr C has advised me that, as a result of the events that led to this complaint, it is now a standard requirement that a detailed written protocol for TTP therapy is inserted in a patient's notes. The second Public Hospital has revised its policy on "Transfusion of Blood Products". A new policy on "Blood Component and Blood Product Administration and the Management of Transfused Patients (Adults)" is under development, and will be included in a proposed education programme and subject to audit. I commend the second Public Hospital on its efforts.

Recommendations

I recommend that the second Public Hospital:

- Apologise in writing to Ms G's family for breaching the Code. This apology is to be sent to the Commissioner and will be forwarded to Ms A.

- Conduct an audit of the use of blood products to ensure that the blood products requested are correct and properly labelled, in accordance with the amended procedures.

I recommend that Dr C:

- Apologise in writing to Ms G's family for breaching the Code. This apology is to be sent to the Commissioner and will be forwarded to Ms A.

- Review his practice in light of this report.

Other Actions

- A copy of this report will be sent to the Medical Council of New Zealand and to the Chief Medical Advisor of the first Public Hospital.

- A copy of this report, with all details identifying the parties removed, will be sent to the Haematology Society of Australia and New Zealand, and to the Deputy Director-General, Clinical Services, of the Ministry of Health (for forwarding to the Chief Medical Advisor of each District Health Board), and placed on the Health and Disability Commissioner's website, www.hdc.org.nz, for educational purposes.